An Introduction to
CLINICAL EMERGENCY MEDICINE

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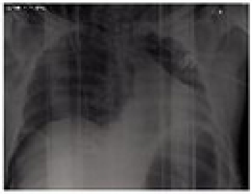

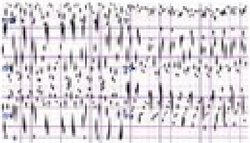

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S. V. MAHADEVAN
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An Introduction to

Clinical Emergency Medicine

Second edition

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Foreword

Although Emergency Medicine is a comparatively young specialty, it already boasts a good number of textbooks, many of which are quite good. There is a real place for the book you are holding, however, not merely because its editors are outstanding educators, or because it's particularly well written, or because it pays careful attention to details (although all of these are true). *An Introduction to Clinical Emergency Medicine, 2nd edition*, is a valuable tool for the right reader because it is addressed to a specific audience, and because of its extremely appropriate complaint-based approach. Before we think more about these two important characteristics, however, we need to reflect a little about the specialty of EM itself.

Some medical specialties are the product of a particular and circumscribed *body of knowledge*. Endocrinology, for example, came into being when new and complex information about human hormones began to be known, leading to a more and more complex understanding of metabolic processes and diseases; some patient problems required a degree of sophistication beyond the scope of generalist practitioners. The same process undoubtedly occurred for most or all of the medical sub-specialties, diagnostic radiology, neurology … and many others. Most surgical specialties, on the other hand (as well as some others, such as interventional radiology), focused less on special knowledge than on special *skills*.

EM is somewhat unique, not merely because it combines both particular knowledge and skills (many other specialties do this as well), but because the set of skills involved is for the most part not procedural, but rather *cognitive*. EM is quintessentially a diagnostic specialty, with *undifferentiated* disease presentation at its core, and the skills required of an EM specialist involve the ability to make crucial (sometimes even "life and death") decisions in the face of a number of rather extraordinary stresses. An emergency physician not only has to establish priorities rapidly in any given patient, she has to do the same *among* a large group of patients. She doesn't have the luxury of undertaking an orderly process comprised of history, then exam, then review of records, then labs or other work-up – as we were all taught in medical school – but often has to *act* entirely out of order, based on brief interactions and rapid assessment, without time to gather much of the information that could be helpful. And she's got to do this with a patient she's never met before, who is likely in pain, or anxious, or confused, or intoxicated, and who furthermore has never met this doctor before either, and so has no reason to trust her competence. Finally, these crucial decisions have to be made, and acted upon, quickly … knowing that other (potentially unstable) patients are waiting!

Learning to be an expert in Emergency Medicine is no easy trick, and – as with any specialty – it is best accomplished through a combination of *training* and *experience*. Residency training takes years, and achieving "mastery" of EM (to the extent that is ever truly possibly) requires as well the ongoing experience that comes from caring for many patients; if my own learning trajectory is any indication, the end of residency is merely the beginning of one's growth, and one continues to get better at this job for *many* years.

An Introduction to Clinical Emergency Medicine is designed primarily for learners at or near the start of a career in EM, and is tailored to such learners in a developmentally appropriate way – because it stresses *how to think as an emergency physician*. Recognizing that the vast majority of our patients present with undifferentiated complaints, this book is organized around an approach to symptoms (rather than diseases). The actual EM approach to diagnostic decision-making is far more complicated than the trendy "worst first" (rule out life threats) approach often cited; while we surely must keep this important consideration in mind, we also need to address a combination of disease *likelihood*, the *potential to intervene* in a way that matters, and an estimate of those circumstances in which *delays* in intervention *would limit effectiveness*. EM also emphasizes (in a way that is different from most other specialties, if not completely unique) the importance of treating acute symptoms (relieving suffering), in addition to the above concerns about identifying and addressing possible threats to life and limb.

While no book can replace the incremental learning obtained during a residency (and afterward), a good book can certainly help. Most books attempt to do so by trying to transmit knowledge; *An Introduction to Clinical Emergency Medicine* also tries to transmit cognitive skills, by focusing on the EM approach to evaluation. Like its first edition, this book is organized around specific complaints (symptoms), and stresses a standardized approach. This both makes for excellent readability, and keeps the focus on residents and senior students who are rapidly developing EM skills. This 2nd edition adds a critically important new element – the "red flag" approach that is the hallmark of how many expert EPs think about patients. For any and every patient presentation ("dizziness," headache, low back pain, shortness of breath, etc.), there are a host of possible etiologies that range from trivial to life-threatening, and from likely to remote. As noted earlier, an organized approach in EM concentrates on identifying (or in many cases, *excluding*) those that not only have potentially important consequences, but are also reasonably probable for the given presentation, *and* are amenable to treatment that can actually limit such adverse consequences, *and* require such treatment acutely if that benefit is to be achieved. Every EP should be able to call to mind the range of diagnoses that meet such criteria, for any given presentation. But that is not enough – because knowing why it is important to diagnose a sentinel subarachnoid bleed is not of much use unless one *also* knows under what circumstances it must be seriously considered and investigated (as in a headache that starts suddenly and is maximal at onset), and just importantly when it *shouldn't* be worked up (as in the average unilateral headache of gradual onset and progressive severity). An EP who orders an MRI for most patients with back pain will cause far more harm than good, but one who omits the MRI because he failed to ask about symptoms of cauda equina syndrome, or didn't look at the needle tracks underneath a patient's sleeve, is of course equally dangerous. An expert EP needs to consider PE in a patient who is suddenly short of breath in the setting of active cancer, but the EP who routinely orders a CT angiogram in patients with dyspnea is *not* an expert.

For every patient presentation, there are characteristics from the history and physical examination whose presence raises the likelihood of "do not miss" etiologies, and whose absence makes them much less likely. The expert EP will learn to organize his thinking not merely around such etiologies, but also around the findings that raise or lower the stakes. Most medical schools teach students to do a "complete" exam and take a "thorough" history. EM residencies, on the other hand, teach a "focused" work-up … but they also need to teach *why* one should ask a given question, because the answer (one way or the other) can and should decide your next step. Red flag questions are the most important ones we ask and this book can be an excellent tool to help learners understand when and why to ask them … and what to do with the answers.

Happy reading … and happy learning.

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Foreword to the 1st edition

Emergency Medicine represents the unique combination of rapid data gathering, simultaneous prioritization, and constant multi-tasking in a time-constrained fish bowl –with all decisions subject to second-guessing by others. It is a patient complaint-oriented specialty in which stabilization based on anticipation supersedes lengthy differentials and diagnostic precision.

In light of these unique aspects and attributes of clinical practice, one would expect the textbook-based literature supporting this specialty to be uniquely written and reflective of its singular approach. This has rarely been the case, a fact that has puzzled me for almost thirty years. It is true that sequential prose does not accurately represent the parallel processing necessary to practice effective and efficient Emergency Medicine. Still, it would seem the ideas of priority diagnoses, stabilization, initial assessment, prioritized differential diagnosis, and the rest that follows could be delineated and emphasized within the limitations of the printed word. I am pleased and delighted to find and convey to the reader that this text succeeds in translating this untraditional Emergency Medicine approach into a textbook format.

This text, edited by two academicians, S.V. Mahadevan, MD and Gus M. Garmel, MD from one of the nation's premier academic institutions and leading health care organizations, fulfills what I have longed believed is the correct and necessary pathway to understanding the approach and thought processes that drive clinical decision-making in Emergency Medicine. The focus of the text is appropriately "presenting complaint-oriented," with a thorough coverage of the chief complaints responsible for the majority of emergency department visits. Each chapter is structured in a consistent manner that allows the experienced and uninitiated alike to clearly track the thought process needed to bring one to a successful prioritized conclusion of care, even when a specific diagnosis has not been made.

The range of authorship is excellent, reflecting the talents and capabilities of an entire new generation of emergency physicians trained in the specialty. These authors clearly understand Emergency Medicine's unique principles.

It is a rare gift to witness and participate in the passing of our unique specialties' visions onto the capable hands of those you've had the opportunity to train and know. Because of this textbook's organization and content, I am pleased to finally "rest in peace," at least academically. Drs. Garmel and Mahadevan demonstrate their clear understanding and literary virtuosity in conveying the truth about our specialty to others.

It is my pleasure to congratulate them on a successful venture, to warn them that having started on this path serial additions and subsequent editions will rule their life for as long as they, the publisher, and the sales last, and to express a personal sense of satisfaction and pride in their accomplishment. To the reader, I say enjoy yourself. Take much away from this text and welcome the truth as we currently know it, presented in a manner that accurately reflects the way we practice.

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Acknowledgments

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Drs. Mahadevan and Garmel are especially grateful to their contributors, national and international authorities in emergency medicine, who donated their expertise to this project for the greater good of patients and clinicians. Finally, special mention goes to Jerome Hoffman, MS, MD, who contributed the insightful foreword to this edition, and Glenn Hamilton, MD, MSM, who shared his views in our first edition – thank you both for your invaluable contributions to this enduring project, and for recognizing its importance.

Preface

Building on the strengths of its award-winning predecessor, the second edition of *An Introduction to Clinical Emergency Medicine* is a must-have resource for individuals training and practicing in this challenging field. This unique text addresses a wide range of clinical topics essential to the practice of emergency medicine. Guided by the patient's presenting complaint, this text emphasizes a methodical approach to patient evaluation, management and problem solving in the Emergency Department. Unlike other textbooks that elaborate on known diagnoses, this extraordinary book approaches clinical problems as clinicians approach patients – without full knowledge of the final diagnosis. This text effectively reveals how to address patients with undifferentiated conditions, ask the right questions, perform a directed physical examination, develop a logical differential diagnosis, and accurately order and interpret laboratory and radiologic tests. Current management and disposition strategies are presented, as well as a summary of pearls, pitfalls and myths for each topic.

Fully revised and updated – including current advanced life support guidelines – the second edition introduces important new chapters on sepsis, bleeding, burns, patient safety, alcohol-related and dental emergencies. The clinically-focused appendix includes new sections on clinical decision rules and focused emergency ultrasound, and improved sections on common emergency procedures and interpretation of emergency laboratory studies. Stunning full-color chapters include high quality images (photographs, ECGs and radiologic studies), detailed illustrations and practical tables. Each chapter in the second edition now contains a critical section on 'red flag' warning signs and symptoms, incorporating the heuristic approach used by successful emergency clinicians.

Written and edited by experienced educators, researchers and clinicians, *An Introduction to Clinical Emergency Medicine, 2nd edition* is certain to remain core reading for medical students and residents, and serve as an important resource for practicing emergency physicians, teaching faculty, and other healthcare providers.

Dedication

S.V. Mahadevan, MD, FACEP, FAAEM

To my parents, Sarojini and Mahadeva S. Venkatesan: For your incredible sense of duty and continuous sacrifices for the sake of your children and grandchildren.

To my mentors: For teaching me not to follow blindly but to question, investigate and discover. Your encouragement and guidance has shaped my career.

To my fellows, residents and students (at home and abroad): For continually inspiring me with your genuine desire to learn, innovative ideas, and unbridled enthusiasm. It is an honor and privilege to teach, advise, and befriend each one of you.

To Rema, Aditya and Lavanya: For encouraging me to seek out new challenges and fulfill my dreams. You fill me with strength, hope and happiness.

Gus M. Garmel, MD, FACEP, FAAEM

To my parents, siblings, extended family and friends: I am truly blessed by your continued support.

To The Permanente Medical Group, Kaiser Santa Clara Medical Center, Stanford University Division of EM, my talented colleagues in and outside of EM, our amazing nurses, and my patients: Thank you for offering me such wonderful opportunities and for enriching my life.

To the Stanford/Kaiser EM Residency Program, its current residents and alumni: I hope that I have served you well over the past 20 years as an educator, administrator, role model and mentor.

To students and housestaff everywhere: As the future of health care, I encourage you to approach patient care responsibilities and treat each patient with honor and privilege.

And to Laura, my partner and best friend: Through you, I've learned how to appreciate love more than I believed possible.

Section 1

Principles of Emergency Medicine

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1 Approach to the emergency patient

Gus M. Garmel, MD

The emergency department (ED) is an extremely challenging environment for patients, families, and medical personnel. Many challenges result from the principles of our practice: available and prepared at any time for any patient with any complaint. Patients who come to the ED are most often unfamiliar with us, yet we must immediately help them feel confident about our abilities. Patients generally present to the ED during a time of great concern. Their needs may be as straightforward as a note excusing them from work or a prescription refill in the middle of the night, or as complex as an acute illness or injury, an exacerbation of a chronic condition, or a cry for help if depressed or suicidal. In their own way, patients almost always seek reassurance about something – is their child's fever dangerous, their headache cancer, or their abdominal pain appendicitis? Providing reassurance to patients, parents and families whenever possible is a critical function of emergency physicians (EPs).

Qualities successful EPs exhibit include intelligence, sensitivity, humility, insight, proficiency in making decisions with and acting on limited information, and the ability to multi-task. Working well with individuals of different backgrounds and ethnicities while at all times strongly advocating for patients are essential qualities. EPs must also be skilled at leadership, negotiation and conflict resolution. They must be exceptional communicators. In addition to these traits, EPs must be experts in both medical and trauma resuscitation of adults and children.

The majority of patients use the ED infrequently. Many are experiencing this setting for the first time. Because patients lack familiarity with this environment, they may have expectations that go unmet. Their fear, stress, waiting time, lack of privacy and discomfort that brought them to the ED can negatively impact their experience. These are only some of the issues that patients contend with in the ED.

EPs confront numerous challenges when taking care of patients presenting to the ED. Perhaps the greatest challenge is the extensive disease spectrum that EPs must be familiar with. Rather than having to know only the first few minutes (or hours) of an illness, EPs must be familiar with all stages of all illnesses, often presenting in atypical fashion. As boarding times increase and observation units become more common, patients remain under an EP's care for longer periods of time. In addition, time pressures inherent to providing emergency care, the lack of existing relationships with patients, unfamiliarity with their medical history, and the inability to review patients' medical records challenge EPs daily. EPs must rapidly and simultaneously evaluate, diagnose and treat multiple patients with multiple conditions, often with limited information, and not confuse subtle nuances between patients. They must be prepared to act and react to prevent morbidity and, when possible, mortality. EPs must maintain a healthy skepticism towards patient's answers to common questions. Considering worst-case scenarios is fundamental to emergency medicine (EM) practice. Most importantly, EPs must be comfortable providing detailed, often devastating information using clear, understandable language to patients and family members with different educational or cultural backgrounds. All this must be done under time constraints, while demonstrating empathy and compassion.

It is indeed a privilege to care for patients during their time of greatest need or when they lack other options. Approaching patients sensitively, recognizing their apprehension, pain, concerns, and perhaps shame is critical to our mission. This is true no matter how trivial a patient's problem may seem. Often, patients consult EPs seeking approval to leave an abusive spouse, for an opinion regarding a physician's recommendation for surgery, or to confirm that they are making the right decision about a parent, child, or loved one. Serving in this capacity without judgment is not only appropriate, but also essential.

It is imperative that EPs approach each patient with an open mind, committed to identify and address not only the presenting problem, but also any coexisting problems. For example, a patient with a history and presenting complaint consistent with esophageal reflux may in fact have an acute coronary syndrome (ACS). A patient presenting with insomnia may have an underlying concern about his or her safety, security, or mental wellness. The ability of an EP to evaluate each patient using history-taking and physical examination abilities, as well as selecting appropriate laboratory or imaging studies (when necessary), is only a portion of our skill set. An experienced EP's "sixth sense" is something that has become recognized and respected by non-EM colleagues.

Unfortunately, the ED is not always conducive to privacy. Despite the Health Insurance Portability and Accountability Act (HIPAA) of 1996 and Protected Health Information (PHI) for patients, attempts to maintain patient confidentiality in the ED present a continuous challenge. Discussions about patient care issues between health care providers, staff, patients and family members often take place behind nothing more than a curtain. Shared spaces, hallways, lack of private rooms or beds, and the demands of time-pressured discussions – often in open spaces, over the phone, or with consultants – stretch efforts at maintaining patient confidentiality. The leadership role that EPs have in the ED affords them the opportunity to demonstrate respect for patient confidentiality and to remind others of the importance of upholding this principle.

Within the last decade, there has been tremendous and appropriate attention placed on medical error and patient safety in hospitals. Human error may occur at any time, but is more likely during high patient volumes or when multiple complicated patients of high acuity present simultaneously. These situations are common in EDs around the world. Human error has been demonstrated to occur more frequently when provider fatigue is greatest (e.g., at the end of a challenging shift or after being awake all night). Systems errors are even more likely to occur during these circumstances. The airline industry has served as a model for reducing errors and improving patient safety in medical practice, especially in the ED. Airline pilots, however, are not required to fly more than one plane at the same time, while simultaneously taking off, landing, and changing course. The EM community should embrace the federal government's attention to medical systems and its role in medical error, as patient safety must always be a top priority. Hospital quality committees review errors of omission and commission, medication errors, errors in patient registration, and errors of judgment. Given the pace of the ED environment, it is remarkable that more errors do not occur. The rapid need for patient turnover, room changes, and test result reporting does not occur with such immediacy in most other areas of the hospital. Hospital administrators and regulators with limited insight about the uniqueness of EM practice should focus attention to, and provide support for, this essential aspect of patient care.

EPs must recognize that patients signed over to them at the end of a shift pose increased risk. These patients typically have pending laboratory or radiography results, are being observed for continued improvement or worsening in their condition, or are waiting for consultants. The EP who initially evaluated these patients should determine the treatment and disposition plans to the greatest extent possible, based on anticipated outcomes. However, some signed-over patients may not have well-established dispositions and may benefit from a new EP's perspective. In such cases, it is better to inform the receiving EP that a clear understanding about what is going on with that patient does not exist than leave things vague. As long as patients present to EDs at any time, patients signed over at shift's end will continue to challenge our ability to provide safe care within our practice. Many hospitals now have regulations in place regarding this aspect of emergency care.

Scope of the problem

A landmark article by Schneider, et al. in the EM literature defines our specialty as one "…with the principle mission of evaluating, managing, treating and preventing unexpected illness and injury." As emergency medical care is an essential component of a comprehensive health care delivery system, it must be available 24 hours a day. EPs provide rapid assessment and treatment of any patient with a medical emergency. In addition, they are responsible for the initial assessment and care of any medical condition that a patient *believes* requires urgent attention. Patients may believe they require urgent attention when in fact they do not. It remains our mission to provide quality medical care and reassurance to patients even under this circumstance. EPs also provide medical support for individuals who lack access to other care opportunities. As the number of uninsured and underinsured persons in the United States increases, and growing numbers of health clinics close, many of these individuals will use the ED for their primary as well as emergency care. This has placed a tremendous burden on the safety net provided by the specialty of EM. It is unclear exactly how governmental health care reform will impact EDs, patient volumes, and overall physician and patient satisfaction.

According to the Centers for Disease Control and Prevention (CDC), which publishes the National Hospital Ambulatory Medical Care Survey (NHAMCS), there were 119.2 million ED visits in 2006; 18.4 million of these patients arrived by ambulance. This is an increase of over 11 million visits from 2000. Patients were admitted to the hospital in 12.8% of ED visits. The ED was the portal of admission for slightly over 50% of all non-obstetric admissions in the United States in 2006, an increase from 36% in 1996. In California, patients visiting EDs were sicker than ever before, with an increase in critical emergency care visits by 59% between 1990 and 1999. In 2000, there were slightly more than 4,000 EDs, yet this number continues to decrease as hospitals and trauma centers close. A 2008 workforce study by Ginde, et al. reported that despite nearly 40,000 clinically active EPs, this was not adequate to treat the growing number of people who visit EDs each year. Despite an increased number of certified residency training programs producing board-prepared EPs, and the increase in EPs from less than 32,000 in 1999, there remains a critical shortage of capable EPs, especially in the rural and central United States. The number of nurse practitioners and physician assistants trained to work in emergency care settings has increased in response to this shortage as well as administrative and financial pressures, and many hospitals staff urgent care and fast-track areas with these practitioners. With decreased funding available for non-ED clinics, and increasing numbers of uninsured patients using the ED as their primary (or only) source of health care, the worsening of ED overcrowding is inevitable.

Hamilton described the clinical practice of EM as one that "…encompasses the initial evaluation, treatment, and disposition of any person at any time for any symptom, event, or disorder deemed by the person – or someone acting on his or her behalf – to require expeditious medical, surgical, or psychiatric attention." This philosophy creates tremendous challenges, as well as opportunities, unique to the specialty of EM. EDs must be fully staffed and always prepared while never entirely certain of patient needs at any given moment. Despite statistics on the number of patients presenting at different times on different days in different months, no model can predict the exact number of medical staff needed to care for even one emergency patient. Clearly, staffing an ED to be fully operational is an expensive proposition given this scenario.

Clinical scope of the problem

Table 1.1 provides the 10 most common reasons that patients visit an ED, according to a recent 2006 national survey. Of all ED visits, over 35% were for an injury. Lacerations of an upper extremity were number 11; lacerations in the aggregate therefore did not make this list because of the manner in which they were categorized and recorded. These data show remarkable consistency in numbers and rank from survey to survey.

Table 1.1 Top 10 reasons for an ED visit

1. Abdominal pain (8,057,000)
2. Chest pain (6,392,000)
3. Fever (4,485,000)
4. Headache (3,354,000)
5. Back symptoms (3,304,000)
6. Shortness of breath (3,307,000)
7. Cough (2,956,000)
8. Vomiting (2,635,000)
9. Pain, site not referable to a specific body system (2,512,000)
10. Throat symptoms (2,278,000)

From the 2006 National Hospital Ambulatory Medical Care Survey, Centers for Disease Control and Prevention.

In fact, patients come to the ED as a result of only a few general categories of problems or complaints. These may be grouped as follows, listed in decreasing frequency.

Pain

Pain is the most likely reason for patients to seek medical care at an ED. It can be traumatic or atraumatic in nature. Chest, abdominal, head, extremity, low back, ear, throat, and eye pain are only a few examples.

Difficulty with…

This can be difficulty with breathing, vision, urination, swallowing, concentration, speaking, balance, coordination, ambulation, or sensation. Difficulty controlling seizure activity would also fall into this broad category.

Fever

Fever is common in children and of great concern to parents. It can be a presenting complaint in adults as well. Conditions causing fever include viral or bacterial infections, such as upper respiratory infection (URI), gastroenteritis, otitis media, urinary tract infection (UTI), cellulitis, pneumonia, and bronchitis. Surgical conditions (such as appendicitis, cholecystitis, atelectasis, and postoperative wound infections), obstetric-gynecologic problems (such as pelvic or cervical infections, mastitis, postpartum infections), deep venous thrombosis (DVT), drugs and drug interactions, cancer, tick-borne infections, malaria or other parasitic infections, vasculitis, and arthritis are other conditions causing fever.

Bleeding

Bleeding may be painful or painless and may or may not have associated symptoms. Examples include lacerations, vaginal bleeding (with or without pregnancy), gastrointestinal (GI) bleeding, epistaxis, and hematologic illnesses such as anemia, von Willebrand's disease, or hemophilia (often resulting in spontaneous bleeding).

Social concerns

Social issues for which patients come to the ED include an inability to care for oneself, a change in behavior (either organic or functional), drug- and/or alcohol-related problems, homelessness, hunger, or concerns of family members that something might be wrong.

In EM, it is essential that care is *coordinated*. This means that EPs should seek assistance in providing patient care, relying on more than just the patient to assess the situation. Family members often provide additional information about illness progression that patients fail to recognize or neglect to share. Prehospital care providers often have useful information about the patient's living situation and whether or not it is appropriate. Psychosocial aspects of each patient must be considered when interpreting presenting complaints and determining patient dispositions, including the appropriate use of consultation. Involving a consultant who focuses solely on his or her area of expertise may result in a less optimal outcome, as he or she may overlook a combination of etiologies causing the problem. When the care of a particular patient is beyond the scope of EM practice, the EP must make certain that the "proper" consultants and the appropriate teams are involved. Social services, discharge planners, patient care coordinators, and, if necessary, behavioral health or chemical dependency specialists may need to be included. EPs must identify whom to turn to in order to ensure and maximize beneficence and patient benefit. EPs often coordinate patient care behind the scenes, which takes time and effort, yet they rarely receive recognition for this.

Anatomic essentials

Anatomic essentials for any patient presenting to the ED are covered in detail throughout the text. Airway, Breathing, Circulation, Disability, and Exposure (ABCDE) are crucial to the initial evaluation and management of patients with emergent or urgent conditions. This may be true for conditions that do not seem emergent at the time, such as the *airway* of a talking patient recently exposed to intense heat (fire, smoke, or steam). The airway is essential not only for gas exchange, but also for protection against aspiration. It may be used for the administration of certain medications. With conditions causing increased intracranial pressure (ICP), airway management with modest hyperventilation results in cerebral vasoconstriction, one aspect of therapy. *Breathing* depends not only on the lungs, but also on the thoracic cavity, respiratory musculature, and central nervous system (CNS). *Circulation* may be compromised as a result of hemorrhage, dehydration, vascular catastrophe, cardiovascular collapse, or vasoconstriction or vasodilatation in response to shock. Evaluating *disability* includes a focused neurologic exam, including an assessment of the level of consciousness (LOC), mental status, and evaluation of motor, sensory, reflexes, cranial nerves, and cerebellar function. A thorough understanding of the neurovascular supply to extremities, especially following traumatic lacerations or injuries, helps identify limb threats or potential morbidity. Knowledge of dermatomes is also helpful when assessing neurologic symptoms. The Alertness, Verbal response, Pain response, Unresponsive (AVPU) scale and the Glasgow Coma Scale (GCS) are two tools that can be recorded to describe the general neurologic status of a patient, as well as follow neurologic status over time. The National Institutes of Health Stroke Scale (NIHSS) is used for patients with cerebral vascular accidents (CVA). Several scores have been validated to predict stroke risk in patients with transient ischemic attacks (TIA); the ABCD2 score (Age, Blood pressure [BP], Clinical features, Duration, Diabetes) is preferred. *Exposure* is essential so injuries are not missed, as well as to consider possible environmental elements contributing to the presentation (e.g., heat, cold, water, toxins).

History

The patient's history has always been considered one of the most important elements in determining a final diagnosis. It is accepted that the history (and physical examination) can determine the diagnosis in up to 85% of patients. A patient's history should focus on the current problem(s), allowing room to identify additional information and determine its relevance. When patients present in extremis, the traditional approach to obtaining the patient's history must be abandoned. In this situation, history and physical examination information must be obtained concurrently. EPs are forced to rely on clinical assessment and impression, and utilize important diagnostic studies during their decision making. Studies that assist in establishing a final diagnosis, such as an electrocardiogram (ECG), glucose, urine dipstick, and other point-of-care (bedside) tests, can be obtained while gathering historical data. Despite this, establishing a final diagnosis is not always possible during the course of the patient's evaluation in the ED. Fortunately, having a final diagnosis is not always necessary, as an appropriate disposition with follow-up evaluation and tests during hospitalization or as an outpatient may be of much greater importance.

When approaching any emergency patient, providers should offer a brief introduction using the appropriate prefix (doctor or medical student) and relevant background information, such as their current level and specialty of training. A gentle yet professional touch, such as a handshake or touch of the wrist, is generally favorably received. Before questioning a patient about his or her present illness or medical history, sit down at the patient's bedside if the situation allows. This not only eliminates towering over a patient, but demonstrates that you are interested in what he or she has to say, and plan to be present and listen for a while (even if this time is short). Patients recall that the amount of time their physician spent with them was greater if their physician sat down during the interaction. After sitting down, *listen* to what the patient has to say. Physicians interrupt their patients early and often, with EPs being some of the biggest offenders. Look patients in the eye so they know you are present, listening and care about their concerns. If you take notes during the interview, do so following a short period of good eye contact. If these notes are done on a computer, remember not to "hide" behind the computer screen. Demonstrate respect for a patient's well-being and privacy by offering a pillow or blanket, adjusting their bed, assisting with covering their body, or providing water (if appropriate). These kind gestures are easy to do yet greatly appreciated, and can be done in a few seconds at the start of each patient interaction.

When possible, use open-ended questions to elicit historical information about a patient's condition. This allows patients to describe their concerns using their own terms. Certainly, some questions require yes or no answers ("Do you have diabetes?"). There will be times when directed questions are required, such as to a patient in extremis, or when a patient does not answer questions promptly or concisely. However, most patients will get to the point of their visit in a relatively short time.

The P-Q-R-S-T mnemonic assists with gathering important historical elements of a presenting complaint from a patient. Using pain as an example, questions relating to the history of a painful condition include those shown in Table 1.2.

Table 1.2 P-Q-R-S-T mnemonic for history of a painful condition

P	is for *provocative/palliative*, as in "What makes this pain worse or better?"
Q	is for *quality* of pain, as in "Describe your pain?" or, "Is your pain sharp or dull?"
R	is for *region/radiation*, as in "What region of your body does this pain occur?" and "Does it radiate, or move, to any other location(s)?"
S	is for *severity*, which may be communicated using a numeric scale from 0–10, a happy–sad faces scale, or the terms mild, moderate, or severe.
T	is for *timing/temporal* relationships associated with the pain. Questions include "When did the pain start?"; "How long did the pain last?"; and "What were you doing when the pain started (eating, exercising, watching television, going to bed)?"

Additional important historical information may be obtained using the mnemonic A-M-P-L-T-O-E (Table 1.3).

Table 1.3 A-M-P-L-T-O-E mnemonic for additional history

A	is for *allergies* to medications, food, latex, seasonal allergens, or other things.
M	is for *medications*, including prescription and non-prescription. Surprisingly, many patients do not consider acetaminophen, ibuprofen, oral contraceptives, insulin, vitamins or herbal remedies to be medications, and do not offer this information.
P	is for *previous or past medical history*, which may provide a clue to the present condition. If this patient has had a similar illness before, he or she may have it again or is at greater risk for it to recur.
L	is for *last meal*, perhaps the least helpful of these questions. Last meal does, however, relate to airway protection in the event of procedural sedation or a surgical procedure.
T	is for *tetanus* status, which should be updated every 5–10 years, depending on the type of wound and its likelihood for being tetanus-prone.
O	is for *other associated symptoms/operations*. Associated symptoms may assist in reaching a diagnosis and may afford the opportunity to relieve discomfort. Some patients do not include previous surgeries in their medical history.
E	is for *events/EMS/environment*, which include the events leading up to the illness, the role of emergency medical services (EMS) during transport (interventions, response, complications), and any environmental influences on the presentation (heat, cold, water, fire, altitude, rave or other party).

Information regarding a patient's family and social history should also be reviewed. Family members with similar illnesses or conditions are important to identify. Examples include a strong family history of cardiac or thromboembolic disease, appendicitis, gallbladder disease, bleeding disorders, or cancer. Social history includes the patient's living situation; marital status; use or abuse of tobacco, alcohol, and/or drugs; occupation; and handedness (in the setting of neurologic disease or extremity trauma).

Several key questions might therefore include:

- How did the pain begin (sudden vs. gradual onset)?
- What were you doing when the pain began?
- How would you describe your pain?
- On a scale of 0–10, how severe is the pain?
- Where is your pain?
- Has it always been there?
- Does the pain radiate anywhere?
- Does anything make the pain better or worse?
- Have you had this pain before?
- Have any family members had pain similar to this?
- What do you think is the cause of your pain?

Associated symptoms are important, as many diseases have a specific collection of symptoms associated with them. The concept of *parsimony* is an important one, in which a diagnosis has a higher likelihood of being correct if one disease can be used to explain the entire constellation of associated symptoms. This provides a more likely explanation than the coincidence of more than one disease being responsible for a patient's illness. Additional caution is needed when evaluating patients at the extremes of age (newborn and elderly), as the likelihood of serious infection and comorbid or coexisting conditions is greatly increased. This is also true for immune-compromised patients and others without physiologic reserve (morbidly obese, postoperative, malnourished, diabetic, steroid-dependent, or often those with mental illness). Some key associated symptoms are listed in Table 1.4. Warning signs in the history are provided in Table 1.5.

Table 1.4 Key associated symptoms

Cardiopulmonary symptoms	Cough, dyspnea, orthopnea, palpitations, dizziness, syncope, and chest pain
Gastrointestinal symptoms	Abdominal pain, nausea, vomiting, anorexia, constipation, diarrhea, and bleeding
Genitourinary symptoms	Dysuria, frequency, urgency, hematuria, and pneumaturia
Obstetric/gynecologic symptoms	Pregnancy, menses, age of menarche, contraception, infertility, sexual history, sexually transmitted infections (STI), vaginal discharge or bleeding, dyspareunia, previous surgeries, recent procedures, and other pelvic infections
Neurologic symptoms	Weakness, difficulty speaking, concentrating, swallowing, or thinking, imbalance, sensory or motor changes, visual problems, and headache

Table 1.5 Ten warning signs in the history

1. Sudden onset of symptoms (especially first time)
2. Significant worsening of symptom(s) that had been stable
3. True loss or alteration of consciousness
4. Cardiopulmonary symptoms (dyspnea, chest pain or pressure)
5. Extremes of age (newborn, elderly)
6. Immune compromise (HIV-positive, AIDS, cancer, diabetes, or on immunosuppressant therapy such as chemotherapy or chronic steroids)
7. Poor historian, including language barriers
8. Repeated visit(s) to a clinic or ED, especially recent
9. Incomplete immunizations
10. Patient signed over at the end of a shift

Physical examination

The physical examination for emergency patients should be complete to identify unexpected conditions, with special focus on areas likely contributing to or responsible for disease. Unfortunately, many EPs are challenged for time and must act quickly, performing abbreviated physical examinations while relying on laboratory and radiology studies. In some circumstances, this may be necessary. However, it is best to do a detailed, problem-pertinent physical examination so that important findings are not missed. In addition, concentrating on associated organ systems that may have a role in the illness is recommended. These areas may provide clues to the etiology of the pain or illness. In fact, establishing a comprehensive differential diagnosis for each complaint and examining areas of the body that may contribute to it allow EPs to prioritize the likelihood of other diagnoses causing the symptoms.

As this chapter describes the approach to the emergency patient, it addresses only general appearance, vital signs, and general physical examination pearls. Other chapters provide details for specific conditions or constellation of symptoms.

General appearance

This may be the most important element of the physical examination for EPs, as it assists with determining who is sick and who is not. Experienced EPs can look at patients and have a reasonably accurate idea of who needs to be hospitalized. This is one reason why EPs are concerned about patients in the waiting room whom they have not yet visualized. General appearance is particularly important in the pediatric population, as social interaction, alertness, playfulness, physical activity (including strength of cry), respiratory effort and hydration status (e.g., amount of tears) are significant findings that can be identified within moments. The younger the patient is, the more difficult it is for EPs to determine wellness based on general appearance alone. The fact that a patient's general appearance is less helpful to EPs at the extremes of age makes caring for these patients more challenging.

Vital signs

Vital signs are important for all emergency patients. A complete set of vital signs should be obtained and repeated at least once during the emergency visit. Often, the vital signs are obtained in triage and not repeated until many hours later when patients are placed in examination rooms. Many EDs have policies that vital signs must be repeated at certain intervals on patients in the waiting room. Though this is a wise strategy, abnormal vital signs may not require action, and normal vital signs may accompany serious illness. EPs should at the very least review one complete set of appropriate vital signs on every patient and address each abnormal vital sign (or consider why it is abnormal). At times, rechecking the vital signs is extremely important, such as the heart rate in a patient with ACS or acute myocardial infarction (AMI), the respiratory and heart rates in patients with difficulty breathing, or the temperature of a child who experienced a febrile seizure. It is of far greater importance to recheck the temperature of a previously afebrile patient with a possible surgical condition or serious bacterial infection (SBI) than a febrile child's temperature following acetaminophen or ibuprofen if they are now well-appearing, playful, and at low risk for a febrile seizure. Orthostatic vital signs (heart rate and blood pressure in supine, sitting, and standing positions) are inherently time-consuming, unreliable, and nonspecific. However, if the situation suggests that these measurements would be in the patient's best interest, they may provide useful information. It is good practice to recheck a patient's vital signs prior to discharge. Table 1.6 provides a list of vital signs to consider in the ED.

Table 1.6 Sixteen vital signs to consider in the ED

1. General appearance (perhaps the most important and underutilized vital sign)
2. Temperature (rectal temperature should be considered in newborns or infants, the elderly who are hypothermic, tachypneic and mouth-breathing, or in patients with alterations of consciousness)
3. Heart rate (including strength, quality, and regularity)
4. Respiratory rate (often miscalculated due to multiplication error)
5. Blood pressure (consider orthostatic BP, although may be falsely negative; also consider BP measurements in each arm or upper and lower extremities in certain conditions)
6. Oxygen saturation (pulse oximetry)
7. Blood sugar (bedside glucose), which provides an immediate value for situations including an altered LOC, a diabetic with the likelihood of abnormally high or low glucose, or when glucose is the only blood test necessary
8. Pain score (from 0–10, or happy–sad faces scale), repeated frequently and after interventions as indicated
9. GCS (best eye opening, verbal and motor responses) from 3–15, or other methods that measure LOC or mental status, such as AVPU or mini-mental status examination
10. Visual acuity (for patients with visual or certain neurologic complaints)
11. $\mathrm{ETCO_2}$ (to identify ventilatory status, especially for all intubated patients and during procedural sedation)
12. Fetal heart tones (for pregnant patients)
13. Peak flow (for asthmatic patients or those with difficulty breathing)
14. Bedside pulse CO-oximetry (when carbon monoxide exposure is suspected)
15. IOP (for suspected glaucoma)
16. Compartment pressure (for patients with suspected compartment syndrome and vascular compromise)

AVPU: *a*lertness, *v*erbal response, *p*ain response, *u*nresponsive; BP: blood pressure; $\mathrm{ETCO_2}$: end-tidal carbon dioxide; GCS: Glasgow Coma Scale; LOC: level of consciousness; IOP: intraocular pressure.

Pearls specific to the physical examination

Be professional

A professional greeting and introduction should evoke warmth and kindness. Patients want to know that the

EP they "have" (they did not "choose") is considerate, sensitive, thoughtful, competent, and listens well; in other words, a true professional. Most patients aren't interested in a joke or a discussion of current events when they are in the ED, at least not immediately. EPs should wash their hands when entering each patient room, preferably so that patients can witness this. They should wear clean and appropriate physician clothing; be polite, well-mannered and well-groomed; and appear well-rested. A current hospital ID badge with name and photograph should be prominently displayed. A health care provider should never bring food or beverages into the examination room.

Go slowly

Try not to rush patients, or seem rushed to them, despite how busy you may be. Speak slowly and clearly, with increased volume for elderly patients should they need it. Warm and clean hands are essential for patient comfort. If you are using gloves, let patients know that this is your practice for all patients. A well-lighted, warm room (if possible) is also preferred. Having a chaperone of the same gender as the patient present is always a good idea, especially during examination of private areas, such as the genitalia, rectum and breasts. Let patients know that this is your standard practice and you are doing it for their benefit (even if you are doing this to protect yourself). Having translators or family members present (when appropriate) also makes patients more comfortable.

Be gentle

Do not proceed immediately to the area of pain, and do not palpate a tender area using more pressure than is absolutely necessary. If possible, attempt to distract patients while examining a painful area. This is especially true for pediatric patients.

Be sensitive

Make patients aware that your focus is on them during your examination, not on other patients with other problems. Furthermore, let patients briefly know what you find immediately following each phase of the examination. There is no reason to do your entire examination and then tell the patient that it was normal. Share with patients that their heart or lungs sound fine immediately after auscultation. If patients have abnormal findings, they may be aware of these from a previous physician's examination. If they were unaware of this finding, avoid accusing their physician of missing something. When appropriate, promptly tell them that it is not dangerous or worrisome if this is the case. There is no reason to increase their anxiety by telling them they have a heart murmur if it is inconsequential. Offering findings in this manner increases patients' confidence in your abilities, especially when you identify a heart murmur that they knew existed.

Be thorough

This is important so that critical findings or other clues to the patient's final diagnosis are not missed. For example, lacerations, contusions or bruises might imply intimate partner violence. If it is relevant to the presenting complaint, expose the patient's skin during the examination of the body region. Rashes may be present that identify life-threatening infectious diseases or may eliminate the need for further diagnostic studies (e.g., meningococcemia or herpes zoster). Always examine the joints above and below an injured area, as injuries may coexist due to transmitted forces. Remove all constricting jewelry and clothing distal to an injured area, as swelling due to dependent edema is likely to occur. Patients may not appreciate this gesture at the time, but it is valuable in terms of patient safety and preventing damage to an item that may require removal later. Make sure that any removed item is given to the patient or a family member.

Be thoughtful

Use language that patients and family members understand. It does not impress patients when physicians use technical jargon to look smart. If patients are not familiar with abbreviations or terms that you have used, they may not be comfortable asking for their meaning. For example, despite the common use of the abbreviation "MI" for myocardial infarction, many people do not know what this means. You may tell a patient that he had an MI, only to be asked later if he suffered a heart attack. In children, involve parents with the examination, such as looking in a parent's throat or ear first. Other skills to use when examining children include letting the child touch your stethoscope or otoscope before using it. Involve older children in the examination by asking which ear they prefer be examined first. Recognize that hospital gowns are not flattering; it is thoughtful to assist a patient by offering to tie his or her gown, especially if they are getting up from their gurney.

Be efficient

An entire physical examination does not need to be done on every patient. For example, funduscopy does not need to be performed on a patient presenting with an ankle injury. Furthermore, examining patients starting with the position they are in rather than the traditional head-to-toe method saves time. For example, if the patient is supine, consider examining their abdomen before their lungs.

Differential diagnosis

Following a thorough history and physical examination with careful review of the vital signs, a differential diagnosis should be established. This differential diagnosis should be as comprehensive as possible, as it suggests which diagnostic tests should be obtained, and in which order. This differential diagnosis also establishes which therapeutic approaches should be initiated, if they have not already begun.

Diagnostic testing

Diagnostic testing in the ED is performed to identify ("rule in") or exclude ("rule out") conditions responsible for the patient's symptoms. As such, it is imperative that EPs have a notion of pretest probability, including disease incidence and prevalence, and the sensitivity, specificity, positive and negative predictive values, and accuracy of the tests they are ordering. It is also helpful to be familiar with likelihood and odds ratios.

Laboratory studies

Because of the time pressures for patient dispositions, many tests now can be performed at the bedside to decrease the turnaround time for results. Classic examples of *point-of-care testing* are the bedside (fingerstick) glucose and urine dipstick or pregnancy (hCG) tests. Numerous implications of this rising technology's role in EM have been studied. Extensive research using new bedside tests for cardiac markers and other tests of cardiac function is ongoing. Treadmill tests on low-risk cardiac patients have been performed from (or in) the ED to risk-stratify patients regarding their need for hospitalization or further testing. The role of nuclear medicine testing has increased tremendously in diagnostic cardiac evaluation from the ED, perhaps in part due to its decreased role in the diagnostic evaluation of pulmonary embolism. Bedside ultrasonography is a test being utilized by EPs with increased frequency to assist with patient diagnosis, treatment, and disposition. As more EDs subscribe to these practices, and more EPs gain skills in these areas, these tests will assume an even greater role in the evaluation and treatment of emergency patients. Unfortunately, government regulations have removed some tests from the ED that were previously performed there. Having these tests done in a laboratory increases the time to receive results, if for no other reason than sample transport time. The implications of increased laboratory and radiology turnaround times are enormous given ED closures, lack of ED and hospital bed availability, and increased patient volumes in EDs across the United States.

Some tests are being ordered or performed by certified nurses during the triage process, where patients register for evaluation and wait for EPs. These tests include urine collection to screen for pregnancy, blood, or infection; ECGs to evaluate cardiac function; and radiographs. Often nurses use protocols to order blood tests from the triage area, and several high-volume EDs have EPs evaluating patients in the triage area to assist with patient throughput. Research has developed rules that health professionals may use to determine a patient's need for X-ray. If these clinical criteria are met, trained nurses in many institutions may order X-rays from the triage area in an effort to streamline care and reduce overall patient time in the ED. Examples of some rules found in the literature include the Ottawa ankle, knee, and foot rules; the Pittsburgh knee rule; the NEXUS rule for cervical spine radiographs; and several head computed tomography (CT) rules (see Appendix A). Depending on the situation, nurses generally use extremity rules in their practice, whereas physicians apply decision rules for C-spine and head CT.

Electrocardiography

It is a good idea to review old ECGs whenever possible and compare these with the new (current) ECG. This is of particular importance in patients with abnormal conduction, abnormal intervals, or abnormal ST and T wave segments. ECGs should be repeated in the ED if patients develop chest pain or if their chest pain resolves, whether spontaneously or following intervention. The importance of serial ECGs cannot be overemphasized in the setting of ACS or chest pain possibly of cardiac etiology. ECGs are invaluable in patients with acute ST-segment elevation MI (STEMI), as the decision to pursue thrombolysis or percutaneous coronary intervention (PCI) is influenced by the timing of the first diagnostic ECG. They also serve as useful adjuncts in the evaluation of several toxic ingestions or presenting symptoms, such as weakness, dizziness, abdominal pain, back pain, confusion, or alterations of mental status.

Radiologic studies

All physicians seem to rely on diagnostic imaging to a greater extent than they did years ago. This has many factors, including the greater role imaging plays in patient care, the increased availability of CT scanners, the manner in which physicians are currently trained, and the increased concern over litigation. Diagnostic imaging (especially CT) has become a standard that physicians must accept and that patients often demand. Failure to order radiologic studies to identify certain conditions may be indefensible, as these tests are sensitive, specific, and readily available 24 hours a day in nearly all EDs. The development of guidelines to help determine which patients require X-rays has provided physicians the ability to safely reduce the number of radiographs ordered. Physicians and patients should be cognizant of the implications of radiation exposure.

EPs use bedside ultrasonography as part of their physical examination skill set in many hospitals, often with the support of radiology. This situation arose out of the need for EPs to have ultrasound available for their patients on a 24-hour basis. Limited focused bedside sonography by EPs can identify hemoperitoneum following abdominal trauma, abdominal aortic aneurysm (AAA), gallbladder disease, cardiac tamponade, intrauterine (and possibly ectopic) pregnancy, DVT, foreign body or abscess, ocular problems, and pneumothoraces, to name a few. Ultrasound research by EPs is identifying additional pathology important for emergency care. EPs first used bedside ultrasonography for the focused assessment with sonography in trauma (FAST) exam. Tremendous success with this limited use encouraged EPs to incorporate ultrasound technology into other necessary areas of their clinical practice. It is important for both EPs and radiologists to work collaboratively in this area, keeping patient advocacy and safety and not financial matters the first priority at all times.

General treatment principles

When evaluating and treating patients in the ED, it is imperative to address life-threats first. A tremendous amount of information can be obtained from the patient's general appearance, vital signs, and history of presenting illness (HPI). This assessment takes less than 1 minute. Risk stratification into "sick" or "not sick," or "stable" or "unstable" is part of this process. Attention to the ABCs (airway, breathing, circulation) is critical, as is having the correct personnel, equipment, and monitoring available. Much of this process occurs simultaneously, often automatically, with more than one health care provider involved. While nurses and techs measure vital signs, connect patients to monitors, and start peripheral intravenous (IV) catheters for blood draw and circulatory access, physicians can intervene with airway management and assess breathing and circulation. In trauma patients, the mnemonic ABCDEFG is addressed in the primary and secondary surveys (Table 1.7).

Table 1.7 ABCDEFG mnemonic for trauma patients

A	Airway
B	Breathing
C	Circulation
D	Disability (neurologic)
E	Exposure
F	Foley (following inspection of the perineum and rectal examination, provided contraindications absent)
G	Gastric decompression (provided contraindications absent)

Cervical spine immobilization and protection is part of the primary survey. "F" also reminds us of the importance of family and friends. They may provide information about the circumstances leading up to the present condition, and should be kept updated as much as possible. When caring for pediatric patients, current literature demonstrates that family members' presence during resuscitation efforts or invasive procedures is extremely important, provided their presence does not interfere with medical care delivery.

At times, histories and physical examinations must be abbreviated and more focused than one might prefer. This is often a necessary part of EM practice. Treatment may need to be initiated based on limited information, previous episodes, physician experience, or physician speculation. In true emergencies, assessment and treatment occur simultaneously. It may be necessary to determine a patient's resuscitation status in an instant. Attempts should be made as quickly as possible to learn this information from the patient, prehospital care providers, family members, nursing home or skilled facilities. Advance directives or durable powers of attorney may provide this information. Having a system in place with electronic medical records or a designated individual (social services, ED tech, or nurse) to make calls may save precious minutes. When in doubt, always do what is medically indicated for the patient, rather than making assumptions that may be incorrect. Remember to do no harm, and always relieve pain, suffering and anxiety.

Adequate pain control is an important element of EM practice. If a patient has a painful condition, it is good practice to address issues of pain control as early as possible. This is true not only for patients presenting with abdominal pain, but in patients with traumatic injuries who would benefit from adequate analgesia. Waiting to administer pain medication to a patient with a clinical fracture until after the X-ray is reviewed is inappropriate. Reassess patients after each intervention, whether following intubation for airway control or the administration of analgesia. Continued reassessment of all patients is critical, particularly the sickest or those at greatest risk of decompensating.

All patients should be treated sensitively, with attention paid to their fears and anxieties. Patients don't wish to be in the ED, where privacy concerns, noises, and discomfort predominate. They would much rather be at home without pain, or in a familiar physician's office. In this sense, EPs and EDs start out with strikes against them. Additionally, long waits, uncertainty, and any unpleasant interactions are rarely interpreted favorably by patients. Respectful treatment, without discrimination or condescension, should be integral to our approach towards all patients.

The American College of Emergency Physicians (ACEP) and other organizations have developed a number of clinical policies by consensus in an attempt to improve patient care and reduce medical error. Although many EPs feel that these policies might be used against them in litigation, or are an attempt to standardize patient care, these policies were established using research and opinion, and are excellent resources. This is especially true for policies addressing complex conditions or those with unclear or rapidly changing diagnostic and treatment approaches. These policies are generally available at no charge. Many similar treatment guidelines are available on-line to assist providers with an evidence-based medicine (EBM) approach to patient care.

Special patients

Elderly

Individuals over 85 years of age are the fastest growing segment of the population. With advances in medical care and the increasing importance placed on disease prevention, diet and exercise, this portion of the population will continue to grow at a tremendous rate. The majority of medical care expenses are spent on the geriatric population during their last few years of life. Geriatric patients are at risk for falls, functional decline, and changes in cognition, as well as cardiac, pulmonary and vascular emergencies. They have reduced physiologic reserve and often are too ill, weak, or complicated to use medical offices for even routine care. As such, many elderly individuals depend on EDs for their overall health care, if they get care at all. When geriatric patients present to the ED, they are far more likely to be admitted to the hospital than younger patients. They are also far more likely to require and benefit from social services if discharged. The best solution is to integrate social services into the care of all geriatric patients. EPs should consider why social services should *not* be asked to see an elderly patient in the ED, as home safety checks, access to meals, transportation to medical appointments, social isolation, depression, financial security, and feelings of being a burden to family members can be addressed. Furthermore, elder neglect or abuse is far more prevalent than reported. From a social perspective, geriatric patients prefer being referred to as "young" rather than "old" (as in 75 years young), and prefer being referred to as "older" rather than "old."

Many medical conditions in older patients do not present as they might in a younger or healthier patient. A UTI in an elderly patient may present with confusion, as might ACS or pneumonia. Many geriatric patients are not able to mount a febrile response to sepsis or infections. In fact, geriatric patients are often hypothermic when septic. As a result, rectal temperatures should be frequently measured in this population. Geriatric patients commonly use over-the-counter medications; on average, elderly patients take five prescription medications daily. Polypharmacy is a frequent concern, and therefore increases the likelihood of drug–drug interactions. Primary providers are often unaware of all medications their elderly patients take, as physician colleagues, consultants, and urgent care providers may prescribe additional medications without sharing this information. Prehospital personnel should be encouraged to bring all medication bottles with patients to the ED so they can be reviewed. This may help identify potential adverse drug interactions, as well as prescriptions of the same medication (or class) with different names. Many drugs interact with warfarin, a common prescription in the geriatric population. Special ID bracelets should be provided to and worn by elderly patients, with select medical conditions, addresses, contacts, medications, and allergies. It is common to see do-not-resuscitate orders included on these bracelets.

Eyesight and hearing often fail in the geriatric population. It is therefore important to check these and consider outpatient referrals to optometry or audiometry. Difficulties with eyesight may result in the inability to read food labels or medication instructions, especially insulin doses. Difficulty with vision in low light makes it nearly impossible for elderly patients to reliably comment on their stools turning darker (hematochezia or melena). Decreased flexibility of the neck and spine makes it challenging for elderly patients to look in the toilet for changes in their stool. Driving abilities may be impaired by visual difficulties or by arthritis (which makes it difficult to change lanes), muscle power (required for defensive maneuvers), fine motor control, coordination, or response time (to avoid collisions). Driving is vital to their independence, and many elderly are unwilling to relinquish this activity.

Falls are more common in the elderly, not only because of visual difficulties, but also because of their diminished ability to avoid objects, climb stairs, or maintain balance and posture. As financial issues are of great concern, medications may not be taken regularly or may be cut in half to decrease the cost. The same goes for food – soups are inexpensive and easy to cook, although many have high sodium content. A dietician or nutritionist can discuss healthy eating habits with elderly patients. Plans for assisted living or skilled facilities should be addressed with geriatric patients before the need is imminent, as should advance directives and powers of attorney. Even a discussion of wills and plans for death should be addressed, although this is best done at a scheduled time in the primary care provider's office. Postal carriers, apartment managers, or neighbors are particularly important to the safety of the elderly population who live alone, as they can check to see that mail is picked up daily, make sure that the individual has eaten or gotten up that morning, or provide brief social contact. These resources can be investigated by social workers.

Pediatric

Pediatric patients often make up a high percentage of patient visits to an ED, especially at night when pediatric clinics are closed and parents are home from work. Many EDs have separate patient care and waiting areas for pediatric patients so they are not as frightened during their visit. Some EDs have special pediatric rooms with colors and decorations to improve the overall experience. Coloring books, stickers and stuffed animals may be helpful as well. It is inadvisable to have a belligerent patient sharing a room with a child (or any patient, for that matter). EDs should have a resuscitation area and equipment especially for children, with color-coded equipment storage matching the colors on the Broselow resuscitation tape. For computer-based medication order systems, pediatric weight-based dosing may help reduce medication error.

Pediatric patients are generally evaluated with parents, which may help the evaluation or make it more difficult. It is important to observe the manner in which children interact with their parents. Physical, emotional, and sexual abuse or neglect should be considered in *all* pediatric visits, especially cases of traumatic injury, genitourinary complaints, or failure to thrive. At times, it may be necessary to have a discussion with a verbal pediatric patient without a parent present. If this situation is necessary, it is advisable to have a second health care professional, preferably of the same gender as the child, in the room with you. Every attempt should be made to minimize a child's time away from his or her parent or guardian unless this separation is warranted. Parents are often concerned about their child's fever, but their true concern may be meningitis or some other serious infection. With as much certainty as possible, these concerns should be addressed. Pediatric patients with ventriculoperitoneal (VP) shunts, leukemia, cancer, cardiac or lung disease, transplants, seizure disorders, or other specialized conditions are generally closely followed by their pediatricians or pediatric specialists. These individuals should be included in or informed of care decisions as early and as much as possible. As younger pediatric patients are at risk for SBI and have less reserve than older children or adults, next day follow-up of patients and cultures (if obtained) should be encouraged according to hospital practices, as patients in this age group can worsen rapidly or unexpectedly.

Drug-seekers

The practice of EM attracts a unique set of patients who use and abuse the ED. Patients who seek drugs, whether they are drug-addicted, drug-dependent, or in constant pain, are common patients seen after clinic hours or when primary physicians are unavailable. Some of these patients simply have decreased abilities to tolerate pain. Many hospitals and EDs have policies about providing narcotic medication to drug-seeking patients, or patients who have abused the system. It is far easier for administrators to write policies for such patients than for EPs to apply them in clinical practice. Whatever the outcome, it is always best to be sensitive to a patient's condition and to treat patients with respect, whether or not drugs are provided. There have been several situations in which denying narcotics to a patient demanding them resulted in injury to or death of health care providers. Referrals to pain clinics, psychiatry, narcotics anonymous, and social services are always appropriate but rarely helpful.

Difficult patients

Patients with Axis II disorders, malingerers, manipulators, litigious patients, and patients with behavioral problems often use the ED for their health care. These individuals may not have insurance, may not have access to clinics, or may enjoy the attention given to them in the ED. These patients are particularly challenging to the medical staff. Federal law prohibits EDs from turning away patients without first performing a medical screening examination (MSE) to evaluate for an emergency medical condition (EMC). At times, security personnel or the police are required to control these challenging patients. In our role as the physicians for health care's safety net, EPs must interact with these unique and challenging patients on a regular basis given the ED's open-door policy. An EP's goals are to be respectful and professional, set strict limits, refer aggressively, and recognize other factors that may influence their behavior. Conditions such as reflex sympathetic dystrophy (RSD), fibromyalgia, postherpetic neuralgia, claudication, osteomyelitis, abscess, or psychosocial conditions such as abuse or depression may not have been considered during prior visits.

Frequent flyers

Patients labeled as "frequent flyers" may or may not have addiction to drugs and alcohol or psychiatric illnesses, although they often do. However, isolation, homelessness, hunger, boredom, mental illness, or searching for attention and care may be reasons for repeat visits. Despite overutilizing the ED, these individuals should be treated respectfully. Many medical staff fear that nice treatment will encourage repeat visits, but providing a meal or a warm place to sit for a short time may be necessary regardless of the number of visits. Abuse of the prehospital care system is even more upsetting to many emergency medical personnel, as the number of available ambulances and prehospital providers decreases when service is provided to these individuals. However, it is always possible that frequent flyers have or will have real illness. It is therefore necessary to carefully focus the evaluation and minimize testing, although studies are often performed despite their high likelihood of being negative. Input from support staff and nursing personnel who have frequently interacted with a particular individual may provide clues of a subtle difference in presentation or behavior. The use of derisive or condescending language to individuals who abuse the medical system is never acceptable. Respectfully addressing their abuse of the system and its impact on others is certainly warranted. When possible, ED or hospital administrators should be notified of these abuses using mechanisms that are in place.

Police custody

Sadly, patients in police custody who need or desire medical attention for evaluation and treatment have no place to go other than the ED (occasionally, some urgent care centers have contractual agreements for this). Often, police bring patients to the ED for medical clearance. This requires an EP to determine whether or not the patient's actions can be explained by a medical or psychiatric condition. Patients often come to the ED in police custody with injuries following an altercation, often with a police officer or officers. This establishes a difficult context for EPs because officers may have injured certain patients in response to their aggressive behavior. This is especially true given the increased use of TASER® technology. If the EP feels safe, he or she should interview patients outside of police presence. It is always difficult to feel comfortable evaluating patients handcuffed to gurneys, with or without police present. However, a thorough yet cautious evaluation for injuries, including contusions, bruises, marks, scratches, abrasions and bites, must be performed and documented. Patients may be placed into police custody from the ED if they are violent, abusive, stealing supplies, or exhibiting inappropriate behavior. Police must be notified about all violent injuries, and may place patients in custody or take them from the ED to jail. Police often deliver intoxicated patients to an ED so they can sober before going to jail. Patients who are intoxicated may have additional reasons for combative behavior or altered mental status, including traumatic brain injury, hypoglycemia, hypothermia or other medical conditions, thus mandating a thorough evaluation. Intoxicated patients may be released to the care of the EP and medical staff if they have cooperated with the police and are not under arrest. When this occurs, careful observation until daylight hours, a meal if possible, and careful plans for disposition, follow-up, and referral should be discussed with a non-intoxicated family member or friend. Clearly, a close working relationship between fire, police and emergency personnel is crucial to our safety and success.

Violent patients

Unfortunately, patients may become violent while in the ED. They may act aggressively towards staff or other patients. This may be due to fear, problems with anger management, psychiatric issues, alcohol and drugs, or gang-related activities. Any patient who physically (and in many cases, verbally) assaults medical personnel, another patient or family member, or staff must be restrained and possibly arrested. Hospital security must be notified immediately, as well as the police. Such behavior must never be tolerated. Care must be taken to protect visitors from patients who might cause harm. In addition, caution must be exercised to prevent visitors from harming patients, as might occur in situations of abuse or gang violence.

Many patients and visitors bring weapons to the ED, having them on their person. Inadvertent discharge of a firearm or accidental injury from a knife or other sharp object necessitates extreme diligence of all health care team members when moving or touching patients. Many EDs have metal detectors and security personnel at their entrance, although weapons can still find their way into the ED. It is recommended that medical personnel position themselves between the door and the patient at all times so a patient can't block their exit. Staff should never place themselves in the way of potential harm, despite how heroic this might seem. Studies have demonstrated that helpful actions include a calm appearance, non-threatening approach, soothing voice, show of both hands (rather than holding them behind your back), and eye contact that is neither intense nor direct (somewhat askew is preferred). When needed, a show of force with multiple large individuals may de-escalate a potentially violent situation. The potential for workplace violence in the ED must be recognized at all times. Chemical and physical restraints are important components of protecting staff and patients, but are not as easy to use as described in the literature. Furthermore, these may not prevent injury from occurring and may result in injury during their application. Therefore, a well-rehearsed approach to the violent patient is critical to help reduce the likelihood of potential harm.

Disposition

Consultation

Dealing with consultants is an art that is often difficult. Consultants respect straightforward, focused, and well-planned presentations with a direct question or goal clearly stated. However, they are unlikely to appreciate being told what to do, such as "this patient needs to go to the operating room" (even when this is clearly needed). On the other hand, depending on the culture of your institution, this direction may be helpful and appreciated for trauma or other critically ill patients. Every consultation is unplanned work for a consultant. Reimbursement issues may negatively impact consultants to a far greater extent than most EPs recognize. Despite such issues, EPs must serve as their patient's advocate at all times. EPs should never do something that makes them uncomfortable, even if a consultant recommends it. This is especially true if a consultant does not formally evaluate the patient. Disagreements about the best plan of action for patients are common. These may be due to financial, time, or hospital pressures. In general, consultants do not wish to hospitalize patients who, in their opinion, do not need admission. Because EPs do not wish to send patients home who, in their opinion, should not be discharged, conflict may be inherent to this interaction. EPs must always keep the patient's best interests in mind. Consider alternate options such as holding patients in the ED until the next consultant comes on duty, finding a different service to admit the patient, enlisting the assistance of social services, admitting the patient to an observation unit (either in the ED or the hospital), or recognizing that it may be safe to send that particular patient home despite your initial concerns. If absolutely needed, EPs can always contact the chief of service, administrator on call, or chief of staff for truly unacceptable situations. When possible, notifying a patient's primary physician or specialist with information about his or her visit, evaluation, laboratory and radiology results, and treatment plan is uniformly appreciated. This is also in the patient's best interest. Not only does this serve as an opportunity for continued care, it also assists in transferring care for that patient. Follow-up notification by EPs to patient's physicians earns additional respect for our specialty and is a fantastic way to let other physicians know that we care about their (our) patients. If interested, request follow-up from these physicians to learn about longitudinal patient care outcomes.

Serial evaluation

Repeat evaluation of patients is an important aspect of emergency care, as a patient's condition may change over time. Many presentations warrant repeat evaluation, including head or traumatic injuries, seizures, hypoglycemic episodes, abdominal pain, shortness of breath, and chest pain. Time may allow a diagnosis to become more apparent or declare itself, or may lead to the resolution of symptoms. It is critical that patients who are impaired (drug or alcohol intoxication, altered mental status, or confused) or are restrained (chemical, physical, or both) have frequent and repeated evaluations by physicians and nurses. Serial evaluation is necessary following interventions, such as the administration of nitroglycerin (NTG), analgesics, bronchodilators, or anxiolytics. This is important not only to determine the patient's response to that intervention, as many interventions are diagnostic as well as therapeutic, but also to determine whether additional or different interventions are needed. Documentation of any response to therapy is important, particularly because it records the patient's ED course. Repeat evaluations of patients after important laboratory or X-ray results become available, and/or before they are discharged is recommended, although the extent of this reevaluation differs with each clinical scenario.

Admission/discharge

The decision to admit or discharge a patient from the ED is perhaps the most challenging aspect of EM practice. Multiple factors must be considered in this decision, including psychosocial, biological, medicolegal, and, unfortunately, financial. When feasible, a patient's wishes should be included in this decision. With the advent of more aggressive outpatient strategies, such as low-molecular-weight heparin for DVT therapy and longer-acting antibiotics with greater potency for many infections, and research suggesting similar outcomes in selected patients, many patients who were previously hospitalized may now be safely treated as outpatients with close follow-up. This greatly expands the role of EPs.

Since disposition strategies for certain conditions vary between hospitals and admitting physicians, it is a good idea for EPs to familiarize themselves with hospital or community practices. In smaller hospitals, EPs may be responsible for writing admission orders for patients. Although EM organizations discourage this practice, it still occurs. Admission orders written by EPs should clearly transfer the care of the patient to the admitting physician immediately upon the patient's arrival to the floor. The nurses should be instructed to notify the admitting physician upon the patient's arrival, if the patient has any special needs, or for any change in vital signs, including pain. Unstable or particularly complex patients should remain in the ED until the admitting physician has the opportunity to evaluate them. In some hospitals, EPs on duty are responsible to respond to in-hospital medical emergencies. Hospital or ED policies should set guidelines to define the circumstances under which the EP does and does not respond to acute medical care situations within the hospital. Similarly, hospital policies should address acceptable time standards for admitting physicians to evaluate their patients so they do not remain in the ED for extended periods.

For patients being discharged, clear and legible discharge instructions should encourage patients to return if their symptoms worsen, change, or don't improve. All discharge instructions should include four categories of instructions: (1) what to do, (2) what not to do, (3) when (and where) to follow-up, and (4) reasons to return to the ED. ***What to do*** includes instructions such as rest, ice, compression, and elevation for an ankle injury. ***What not to do*** instructions might include don't smoke, don't drive, don't use alcohol, or don't stop your antibiotics until completed or instructed by your physician. ***When (and where) to follow-up*** for re-evaluation, and with whom, is beneficial information for discharged patients. The time frame for follow-up should directly relate to the certainty of the diagnosis and the likelihood that the illness or injury will degenerate to a critical condition. Close follow-up is important for all patients with high-risk medical conditions and comorbidity. The ideal situation is to schedule a follow-up appointment for the patient at the time of his or her discharge. Give the patient this follow-up physician's name, the date and time of the appointment, and the address with directions to the clinic. Perhaps the most important discharge instruction is the list of ***reasons to return to the ED***. These might include but are not limited to any increase in pain, new or different pain, worsening of symptoms, inability to take medications or fluids, allergic reactions to any medications, fever, vomiting, bleeding, or any other concerns or fears. Preprinted discharge instruction sheets are helpful if they are written in a language and at a level that patients can understand. These allow EPs to be more efficient. However, patients deserve personalized instructions as well, as each patient is an individual, not a disease or set of symptoms. It is best when someone reviews these instructions with a patient prior to discharge.

Assisting patients with filling their prescriptions at discharge is important, although this does not necessarily ensure compliance. If this is not possible, discharging patients with a 1- or 2-day supply of medication is reasonable. Testing a patient's gait prior to discharge helps determine their balance, coordination, and likelihood of success at home. If a patient walked in to the ED, or "should be able to walk," then this patient should be able to walk at discharge. Patients should be discharged to a safe environment, preferably in the company of a responsible adult who also understands the discharge instructions. If they have been in the ED for an extended period, providing a meal is appropriate, as they may be too ill or tired to prepare one for themselves upon returning home. Staff should assist patients to their vehicle, by wheelchair if necessary. Patients should not drive if they might be distracted, were given medication that may interfere with driving, or presented with a lapse of consciousness that may recur without warning. In this last situation, a report must be filed with the appropriate authorities, and the patient needs to be informed of this. They should not drive until an appropriate physician and the proper authorities approve this at a follow-up appointment. Transportation home or to a shelter may be necessary, often paid for by the ED or hospital. A clean or warm set of clothes or shoes may be needed. If the hospital or ED budget does not provide for this, donations from the medical staff or other sources should be solicited.

Pearls, pitfalls and myths

- Always address life-threats first, including patient and staff safety.
- An exact diagnosis is not always possible in EM, and not always necessary. An appropriate disposition, such as admission to a monitored bed, intensive care unit (ICU), operating room, skilled nursing facility (SNF), or discharge home with close follow-up and reasons to return is acceptable.
- Not all is what it seems in EM; expect the unexpected, or you won't identify it. Consider alternative diagnoses and the possibility of lab error or false-negative (or false-positive) test results if things don't make sense. Repeat tests if the original test result doesn't "fit" with what you expected. Be wary about the wrong test results being placed in the wrong patient's chart, or a laboratory specimen or radiograph being mislabeled, improperly marked, or incorrectly collected.
- Attempt to get the appropriate service or consultant involved when necessary to improve patient outcome. This is often required before all laboratory or X-ray results have returned. Make every effort to inform a patient's primary care provider about the circumstances leading to the ED visit, the care provided, laboratory and X-ray results, and your recommended follow-up plan.
- People with psychiatric illness may have medical illnesses too. Consider ingestions, cardiac, metabolic, infectious, and CNS derangements as the cause of a presenting complaint. This also holds true for intoxicated patients (drugs or alcohol), as concomitant brain injury or metabolic derangements as an explanation for alterations of mental status or behavior are "present until proven otherwise."
- Many elderly patients have uncommon presentations for common conditions, such as ACS or sepsis. Furthermore, polypharmacy and drug–drug interactions should be considered, along with elder abuse, neglect, depression, and suicidal gestures or attempts. Consider the safety of an elderly patient being discharged, and always remain his or her advocate. Social services or safety checks on all elderly patients presenting to the ED should be encouraged.
- Never rush a patient out of the ED with a condition that may recur, such as asthma, seizures, chest pain, breathing difficulty, or alteration of consciousness (following head trauma or intoxicants).
- Be sensitive, sit with patients, make good eye contact, and listen well for obvious as well as hidden issues. Patients may wait to gain your trust before sharing these concerns.
- Review nursing and EMS notes on all patients. Look for clues that the patient may not offer or tell you. Enlist the assistance of others to help you with patient care, including nursing, family, EMS, social services, consultants, or a patient's primary care physician. Poison centers and on-line resources may be extremely valuable as well.
- Use caution in patients with language or cultural barriers. Translators and family members may not provide complete or accurate information, details which you might have been able to elicit if these barriers did not exist. This is especially true for patients who are deaf or have speech impediments.
- Think about abuse or neglect in *every* case. If you aren't thinking about it, you will not uncover it.
- Document appropriate findings in the medical record clearly, including repeat examinations, laboratory results and radiograph interpretations, discussions with consultants or primary care providers, and discharge instructions. Documenting a consultant's name, service, time you spoke, and brief summary of the conversation is prudent.
- Consider dangerous outcomes or the worst-case scenario in every patient. Minimize the likelihood of these outcomes with appropriately focused histories, physical examinations, laboratory and radiograph ordering and interpretation, and disposition. Never do something you are uncomfortable doing, even if a consultant or colleague recommends it.
- Enjoy the privilege of providing emergency care to all patients and serving in the role of patient care advocate.

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2 Airway management

S.V. Mahadevan, MD and Shannon Sovndal, MD

Scope of the problem

Airway management is arguably the single most important skill taught to and possessed by emergency physicians. Timely effective airway management can mean the difference between life and death, and takes precedence over all other clinical considerations with the sole exception of immediate defibrillation of the patient in ventricular fibrillation. It represents the "A" of the mnemonic ABC (airway, breathing, circulation), and forms the foundation for the resuscitation of critically ill and injured patients. Airway management encompasses the assessment, establishment and protection of the airway in combination with effective oxygenation and ventilation.

This chapter reviews airway anatomy and assessment, approaches for noninvasive airway management, and indications and techniques for definitive airway management. The approach to the challenging patient with a difficult or failed airway will also be explored, as well as specialized devices, techniques and medications employed in these formidable clinical situations.

Anatomic essentials

A clear understanding of airway anatomy is essential for airway evaluation and management. The term *airway* represents many structures and well-defined spaces. Internally, the airway originates at the nasal and oral cavities (Figure 2.1). The *nasal cavity* extends from the nostrils to the posterior nares or choanae. Resistance to airflow through the nose is almost twice that of the mouth, explaining why patients mouth-breathe when they require high flow rates (e.g., with exercise). The *nasopharynx* extends from the end of the nasal cavity to the level of the soft palate. The tonsillar lymphoid structures are the principal impediments to airflow through the nasopharynx.

The *oral cavity* is bounded by the teeth anteriorly, hard and soft palate above, and tongue below. The *oropharynx*, which communicates with the oral cavity and nasopharynx, extends from the soft palate to the tip of the epiglottis. The tongue is the principal source of obstruction in the oropharynx. This obstruction results in part from decreased muscle tone of the genioglossus muscle, which contracts to move the tongue forward during inspiration and dilate the pharynx.

The oropharynx continues as the *laryngopharynx (hypopharynx)*, which extends from the epiglottis to the upper border of the cricoid cartilage (at the level of the C6 vertebral body). The *larynx*, which lies between the laryngopharynx and trachea, serves as an organ of phonation and a valve to protect the lower airway from aspiration. The larynx is made up of muscles, ligaments and cartilages, including the thyroid, cricoid, arytenoids, corniculates and epiglottis.

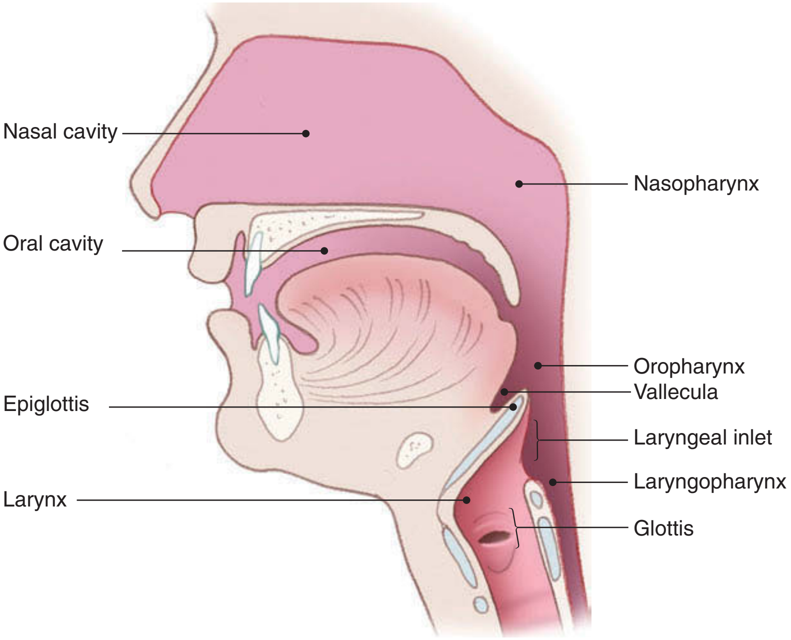

Figure 2.1
Lateral view of airway anatomy. © Chris Gralapp.

The flexible *epiglottis,* which originates from the hyoid bone and base of the tongue, covers the glottis during swallowing and provides protection from aspiration. During laryngoscopy, the epiglottis is as an important landmark for airway identification and laryngoscopic positioning (Figure 2.2). The *vallecula* is the space at the base of the tongue formed posteriorly by the epiglottis and anteriorly by the anterior pharyngeal wall. The *laryngeal inlet* is the opening to the larynx bounded by the epiglottis, aryepiglottic folds, and arytenoid cartilages. The *glottis* is the vocal apparatus including the true and false vocal cords and the glottic opening. The triangular fissure between these vocal cords is the *glottic opening,* the narrowest segment of the larynx in adults.

Externally, clinicians should be familiar with key anatomic landmarks, integral to the assessment and management of the airway (Figure 2.3). The *mentum* is the anterior aspect of the mandible, forming the tip of the chin. The *hyoid bone* forms the base of the floor of the mouth. The *thyroid cartilage* forms the laryngeal prominence ("Adam's apple") and thyroid notch. The *cricoid cartilage,* lying inferior to the thyroid cartilage, forms a complete ring that provides structural support to the lower airway. The *cricothyroid membrane* lies between the thyroid and cricoid cartilage and serves as an important site for surgical airway management.

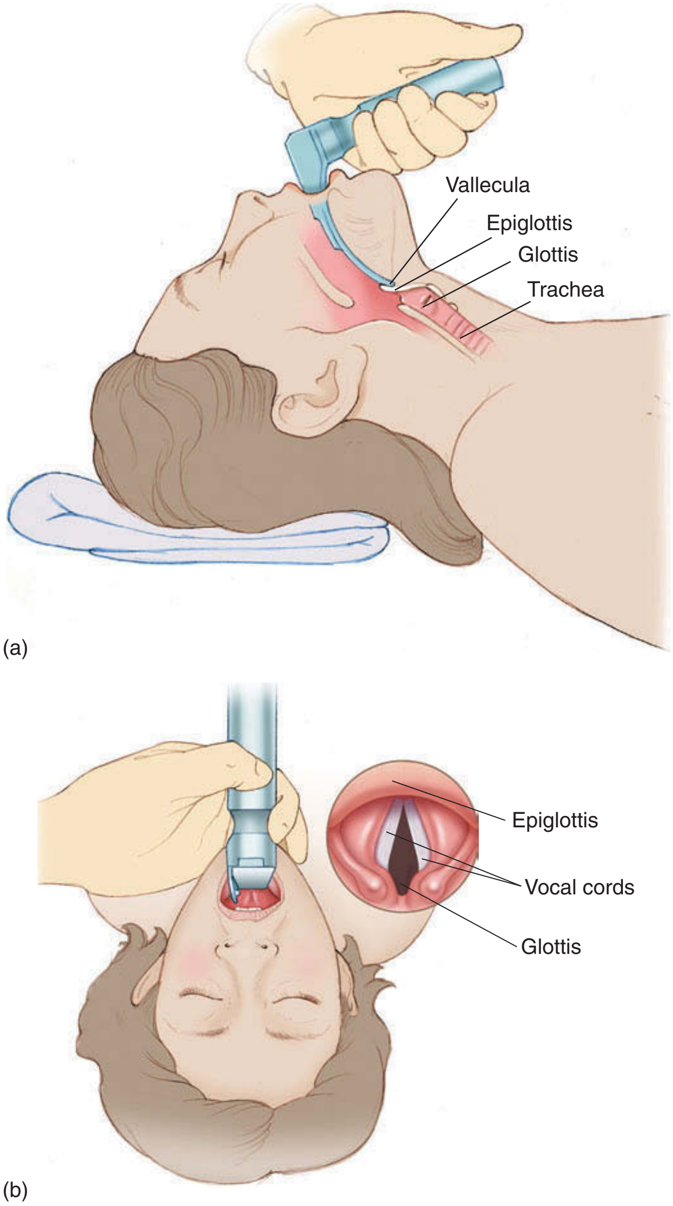

Figure 2.2
(a) Position of laryngoscope blade when using a curved blade.
(b) Operator's view of anatomy. © Chris Gralapp.

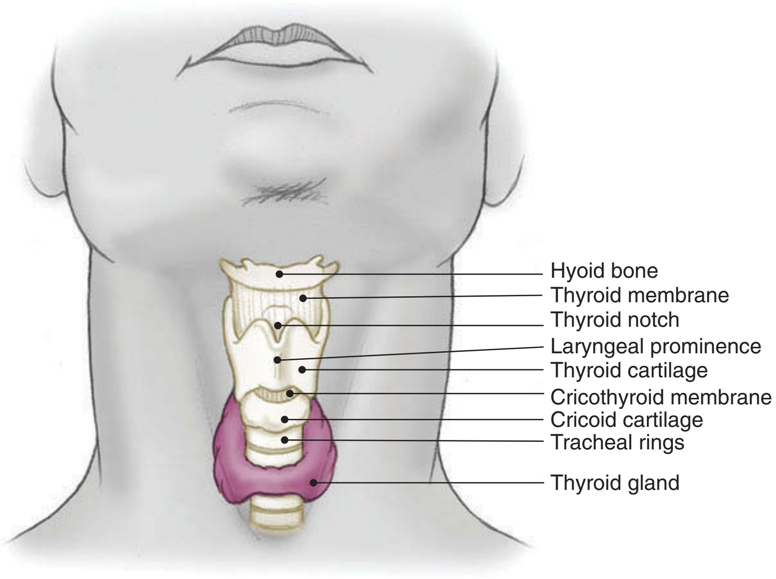

Figure 2.3.
External airway anatomy. © Chris Gralapp.

Initial airway assessment

The initial assessment of airway patency and respiratory function focuses on determining:

1. Whether the airway is open and protected;
2. Whether breathing is present and adequate.

This is carefully achieved through inspection, auscultation and palpation.

The patient should be observed for objective signs of airway compromise. Agitation may represent hypoxia, obtundation suggests hypercarbia, and cyanosis indicates hypoxemia.

The patient's respiratory rate and pattern are important. Bradypnea or tachypnea may be a sign of impending respiratory compromise. Respiratory muscle fatigue may result in the recruitment of accessory muscles of respiration, clinically manifested as suprasternal, supraclavicular or intercostal retractions. Look for a symmetrical rise and fall of the chest. A traumatic injury to the chest may result in paradoxical or discordant chest wall movement.

The presence or absence and quality of speech may be used to identify airway abnormalities. A normal voice suggests that the airway is adequate for the moment. *Stridor*, a high-pitched inspiratory sound, may be associated with partial airway obstruction at the level of the larynx (inspiratory stridor) or the trachea (expiratory stridor). Snoring usually indicates partial airway obstruction at the pharyngeal level, whereas hoarseness suggests a laryngeal process. *Aphonia* in the conscious patient is an extremely worrisome sign; being too short of breath to speak is a sign of impending respiratory collapse.

The central face and mandible should be inspected and palpated for structural integrity; injuries to these structures may lead to airway distortion or loss. The anterior neck should be carefully inspected for penetrating wounds, asymmetry or swelling that may herald impending airway compromise. The palpation of subcutaneous air suggests a direct airway injury and is cause for concern.

Feel for air movement at the mouth and nose. Open the mouth and inspect the upper airway, taking care not to extend or rotate the neck. Look for and remove any vomitus, blood or other foreign material. Identify swelling of the tongue or uvula, sites of bleeding, or other visible abnormalities of the oropharynx. The patient's ability to spontaneously swallow and handle secretions is an important indicator of intact protective airway mechanisms. In the unconscious patient, the absence of a gag reflex has traditionally been associated with loss of protective airway reflexes.

Auscultation should demonstrate clear and equal breath sounds. Diminished breath sounds suggest pneumothorax, hemothorax or pleural effusion. Wheezing and dyspnea imply lower airway obstruction.

In pediatric patients, visual signs of possible airway and respiratory compromise include tachypnea, cyanosis, drooling, nasal flaring and intercostal retractions. A child with severe upper airway obstruction may get in to the "sniffing position" to straighten the airway and reduce occlusion. A child with severe lower airway obstruction may assume the "tripod" posture – sitting up and leaning forward on outstretched arms – to augment accessory muscle function.

Noninvasive airway management

Opening the airway

The first priority in airway control is ensuring airway patency for adequate oxygenation and ventilation. The conscious patient uses the musculature of the upper airway to maintain patency and protective reflexes to protect against aspiration of foreign substances, gastric contents or secretions. In the severely ill, compromised or unconscious patient, these protective airway mechanisms may be impaired or lost.

The most common cause of upper airway obstruction in the unconscious patient is posterior displacement of the tongue and epiglottis at the level of the pharynx and larynx. This occlusion results directly from loss of submandibular muscle tone, which provides direct support to the tongue and indirect support to the epiglottis.

Two simple manual maneuvers can alleviate this occlusion and reestablish airway patency and airflow. The *head tilt with chin lift* (Figure 2.4) is an effective technique for opening the airway, but should be avoided in any patient with a potentially unstable cervical spine. The *jaw thrust without head tilt* (Figure 2.5), however, can be performed while maintaining cervical spine alignment. Although these techniques work well, they require the continuous involvement of a single provider to maintain airway patency.

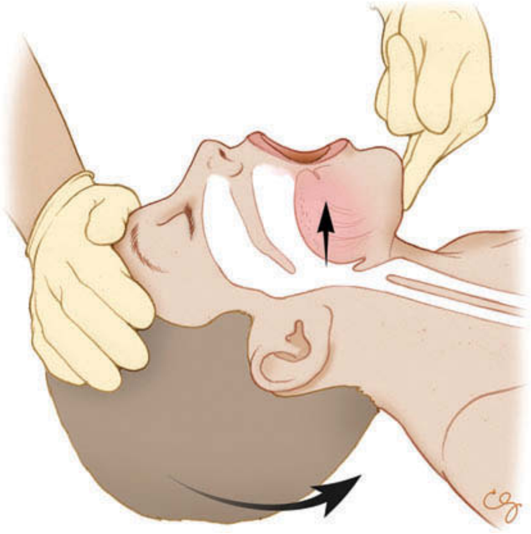

Figure 2.4
Head tilt with chin lift. © Chris Gralapp.

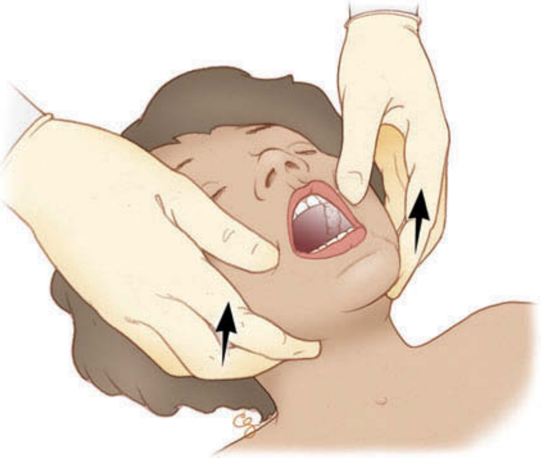

Figure 2.5
Jaw thrust without head tilt. © Chris Gralapp.

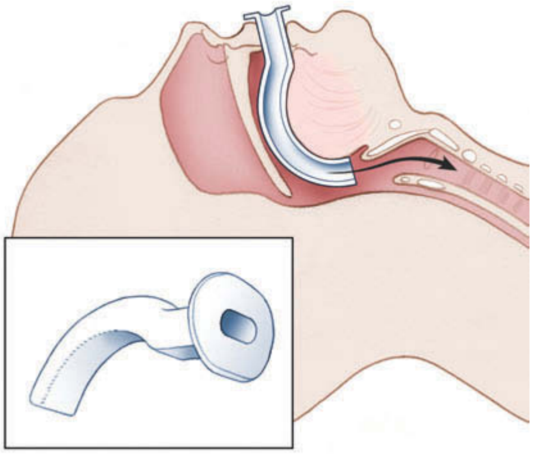

Figure 2.6
Oropharyngeal airway. © Chris Gralapp.

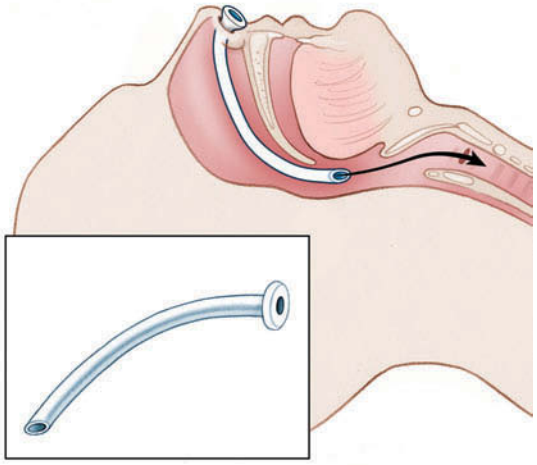

Figure 2.7
Nasopharyngeal airway. © Chris Gralapp.

Airway adjuncts can maintain airway patency while freeing the health care provider to perform other duties. The *oropharyngeal airway* (OPA) is an S-shaped device designed to hold the tongue off the posterior pharyngeal wall while providing an air channel and suction conduit through the mouth (Figure 2.6). It is most effective in patients who are spontaneously breathing but lack a gag or cough reflex. The use of an OPA is contraindicated in a patient with a gag or cough reflex as it may stimulate vomiting or laryngospasm. The OPA comes in various sizes to accommodate children through large adults. The proper OPA size is estimated by placing the OPA's flange at the corner of the mouth; the distal tip of the device should reach the angle of the mandible.

The *nasopharyngeal airway* (NPA) is an uncuffed trumpet-like tube made of soft rubber or plastic that provides a conduit for airflow between the nares and pharynx (Figure 2.7). It is commonly used in intoxicated or semiconscious patients who do not tolerate an OPA. It is also effective when trauma, trismus ("clenched teeth") or another obstacle (e.g., wiring of the teeth) preclude the placement of an OPA. Proper NPA length is determined by measuring the distance from the tip of the nose to the tragus of the ear. Though OPAs and NPAs help establish artificial airways, they do not provide definitive airway protection from aspiration.

Supplemental oxygen

Oxygen (O_2) should be administered to all seriously ill or injured patients with cardiac disease, respiratory distress, shock or trauma, even if their measured arterial O_2 tension is normal. A variety of O_2 delivery techniques may be employed depending on the desired O_2 concentration and clinical circumstance (Table 2.1). Administration should begin at a high level and then be titrated downward. Though O_2 should never be withheld from a hypoxic patient with respiratory distress, care should be exercised when treating patients with chronic hypercarbia, such as patients with chronic obstructive pulmonary disease (COPD). Unmonitored treatment of these patients with high O_2 concentrations can result in respiratory depression from loss of their hypoxic ventilatory drive.

Ventilation

Adequate ventilation implies the following:

1. Sufficient O_2 delivery to the alveoli, and
2. Sufficient carbon dioxide (CO_2) removal from the lungs.

Despite an open airway and supplemental O_2, a patient with inadequate ventilation cannot conduct meaningful gas exchange.

The sequence of interventions for the inadequately ventilating patient includes opening the airway, placement of an OPA, and bag-mask ventilation (BMV). The self-inflating ventilation bag with facemask provides an emergent means of ventilation. It is equipped with several valves that allow for coordinated flow of air into and out of the patient. This includes a non-rebreathing valve that allows exhaled CO_2 to escape into the atmosphere without being entrained back into the lungs. When attached to a high-flow O_2 source (10–15 L/min), the bag-mask can supply an O_2 concentration of nearly 100%. The adapter for the facemask is interchangeable with an endotracheal tube (ETT), so the same bag can be used postintubation.

BMV is arguably the most important emergency medical skill. Competence with BMV is a prerequisite for using paralytic agents to intubate a patient. Substantial proficiency is required to use one hand to maintain an adequate mask seal, position the patient's head, and assure airway patency, while using the other hand to ventilate. Although mastery of solo BMV technique is imperative, recruitment of another individual allows one person to perform a jaw thrust and ensure a good mask seal with both hands while the second individual squeezes the bag.

The effectiveness of BMV is determined by observing the chest rise and fall, feeling for resistance in the bag, and monitoring the patient's O_2 saturation and end-tidal CO_2.

Indications for definitive airway management

A definitive airway implies "patency and protection." This requires an ETT in the trachea, secured in place, with the cuff inflated, and attached to an O_2-rich ventilation device. The inability or failure to secure a definitive airway in a timely manner can have disastrous consequences for the patient.

Although the ultimate decision to intubate a patient is often complicated and may depend on a variety of clinical factors, there are four fundamental reasons that patients require definitive airway management:

1. Inability to maintain or protect the airway
2. Failure of ventilation or oxygenation
3. Potential for deterioration based on the patient's clinical presentation
4. Patient safety and protection

Inability to maintain or protect the airway

An open airway is required for adequate oxygenation and ventilation. Patients who are unable to swallow

Table 2.1 Oxygen delivery techniques

O_2 delivery technique	Flow rate (L/min)	Concentration delivered (%)	Other
Nasal cannula	1–6	24–44	Inspired O_2 concentration depends on flow rate and patient's tidal volume
Simple face mask	6–10	35–60	May promote CO_2 retention at lower flow rates
Venturi mask	2–12	24–60	Accurately controls proportion of inspired O_2 Use in patients with chronic hypercarbia (i.e., COPD)
Face mask with O_2 reservoir	12–15	65–75	Provides high inspired O_2 concentration
Bag-mask	15	90–97	Provides the highest inspired O_2 concentration
Blow-by	6–10	Varies	For infant or young child who will not tolerate face mask or cannula

COPD: chronic obstructive pulmonary disease; CO_2: carbon dioxide; O_2: oxygen.

spontaneously and handle their secretions, or lack a gag reflex, are at risk for aspiration. Although repositioning maneuvers (chin lift, jaw thrust) or airway adjuncts (OPA, NPA) may serve as temporizing measures, they do not provide protection from aspiration of gastric contents, which carries a significant associated morbidity and mortality. Therefore, patients who are unable to maintain or protect their own airway need intubation. The exception to this rule is the patient with a rapidly reversible condition, such as narcotic overdose or dysrhythmia.

Failure to ventilate or oxygenate

The patient who is inadequately ventilating despite maximal clinical therapy or remains severely hypoxemic despite supplemental O_2 may need intubation. The decision to intubate these patients is based on a combination of clinical findings including general appearance, work of breathing, perfusion status, O_2 saturation and clinical course. When deciding to intubate, arterial blood gases (ABG) are rarely necessary, can be misleading and may delay definitive therapy. Intubation allows for the delivery of higher concentrations of O_2 as well as positive-pressure ventilation, which tends to improve most circumstances of hypoxia and ventilatory failure.

Potential for deterioration based on the patient's clinical presentation

Anticipating airway compromise before it occurs is one of the most challenging aspects of emergency airway management. Certain conditions mandate the need for definitive airway management even in the absence of specific airway, ventilatory or oxygenation failure. This decision to intubate is based on anticipated anatomic or physiologic airway deterioration or ventilatory compromise. For example, the decision to intubate an awake, talking patient with a suspected thermal injury to the airway may be difficult but necessary to avoid future airway occlusion and compromise. Delaying definitive airway management in this patient could allow the interval development of significant airway edema, making endotracheal intubation extremely difficult if not impossible. Other patients in whom early airway management should be considered include those with significant facial fractures, penetrating neck trauma, tracheal or laryngeal injuries, severe head injury, multiple trauma, sustained seizure activity or certain overdoses (e.g., tricyclic antidepressant).

Patient safety and protection

Agitated, combative or confused patients may harm themselves in certain clinical situations, making them candidates for prophylactic intubation. For an agitated multiple trauma patient with an unstable cervical spine injury, sedation and intubation may be the only safe way to adequately immobilize and protect the patient during the initial assessment, diagnosis and treatment.

Definitive airway management

Rapid sequence intubation

Rapid sequence intubation (RSI) is a series of defined steps intended to allow for rapid oral intubation of a patient without interposed BMV. Because most patients requiring emergent intubation have not fasted and may have full stomachs, BMV may inadvertently lead to gastric distention followed by regurgitation and aspiration. To minimize the risk of aspiration, the patient is first pre-oxygenated with 100% supplemental O_2 to allow for a period of apnea without assisted ventilation. This is followed by the sequential administration of an induction agent and a rapidly acting neuromuscular blocking agent (NMBA) to induce a state of unconsciousness and paralysis, respectively. The patient may then be intubated without the need for BMV.

The steps making up RSI can be thought of as nine "Ps" (Table 2.2).

Table 2.2 The nine Ps of rapid sequence intubation

Time	Action
0–10 min	Possibility of success
0–10 min	Preparation
0–5 min	Preoxygenation
0–3 min	Pretreatment
Time zero	Paralysis (with induction)
0 + 20–30 sec	Positioning and protection
0 + 45 sec	Placement
0 + 45 sec	Proof
0 + 1 min	Postintubation management

Possibility of success

The patient should be carefully evaluated for a potentially difficult airway, and assessed for ease of BMV should the intubation prove difficult or impossible.

When evaluating a patient for ease of intubation and bag-mask ventilation, it is important to use a consistent approach. Two logical, easily remembered mnemonics for anticipating the difficult laryngoscopy and the difficult BMV are the *LEMON* law and *MOANS*, respectively.

LEMON Law: Anticipating the difficult laryngoscopy

Look externally

A brief and targeted exam of the jaw, mouth, neck and internal airway may help identify features that predict a difficult airway. Initial inspection should identify anatomic features such as morbid obesity, abnormal facial shape, facial or neck trauma, large or abnormal teeth, protruding tongue or the presence of facial hair that may pose a challenge to intubation, ventilation or both. An abnormal facial shape, extreme cachexia, a "toothless" mouth with sunken cheeks, trauma to the lower face or facial hair may prevent an adequate seal for effective BMV. Large buckteeth or central incisors, a receding mandible or a short bull-neck may provide anatomic barriers to oral intubation. Obesity generally makes intubation and ventilation more challenging. Some of these features may also be remembered by the mnemonic *BONES* (Beard, Obese, No teeth, Elderly, Sleep apnea/snoring.)

Evaluate the 3-3-2 rule

The 3-3-2 rule describes the ideal dimensions of the airway that facilitate direct visualization of the larynx. It is easily remembered as three (of the patient's) fingers in the mouth, three fingers under the chin and two fingers at the top of the neck. The ability to accommodate three fingers in the mouth indicates an adequate mouth opening. Three fingers from the tip of the chin (mentum) to the floor of the mouth (hyoid bone) indicate the patient's mandible is large enough to accommodate a normally sized tongue. A small mandible and large tongue may obstruct access to the larynx during intubation. Finally, two fingers' breadth from the floor of the mouth (hyoid bone) to the thyroid cartilage indicates an adequate neck length and laryngeal position. A high or anteriorly placed larynx may be very difficult to visualize during laryngoscopy.

Mallampati

The Mallampati classification is a scale (I–IV) used to predict the ability of a patient's mouth to accommodate both the laryngoscope and ETT. To determine a patient's classification, have the patient to extend their neck, open their mouth as widely as possible and stick out their tongue without phonating. The degree to which the base of the tongue, faucial pillars, uvula and posterior pharynx are visible determines the Mallampati class (Figure 2.8). Class I and II predict greater oral access for the laryngoscope and superior laryngeal exposure, thereby portending a greater likelihood of successful intubation. In the case of Class III and IV scores, the tongue is large in relation to the oral cavity, signifying limited oral access, a limited view and higher intubation failure rates.

Obstruction of the airway

Upper airway obstruction can make intubation and ventilation difficult if not impossible. When time allows, patients should be screened for the presence of upper airway infections (epiglottitis, peritonsillar abscess, prevertebral abscess), laryngeal masses or tumors, or any other upper airway conditions that may complicate laryngoscopy and BMV. Foreign bodies, extrinsic airway compression and direct airway trauma (including the possibility of airway disruption) should be considered strong evidence of an obstruction that could hinder or preclude intubation and ventilation.

Neck mobility

Proper mobility and alignment of the head and neck can facilitate laryngoscopy and intubation. Certain conditions such as cervical spine immobilization and degenerative arthritis may limit mobility and complicate intubation.

MOANS: Anticipating difficult bag-mask ventilation

Mask seal

Conditions that make mask seal difficult (e.g., bushy beard, facial disruption, and crusted blood on the face).

Obstruction or obesity

Obstruction of the airway (e.g., angioedema, upper airway infections, tumors, hematomas, foreign bodies). Obesity and term pregnancy (redundant tissue, chest and abdominal wall weight, resistance to diaphragmatic excursion by the abdominal contents).

Age

Older patients (age >55) have a loss of muscle and tissue tone in the upper airway.

No teeth

An edentulous patient's face collapses inward, so leave the dentures in for BMV but remove them for intubation.

Stiff lungs

High airway resistance or decreased pulmonary compliance (e.g., asthma, COPD, pulmonary edema, acute respiratory distress syndrome (ARDS), restrictive lung disease, and pneumonia).

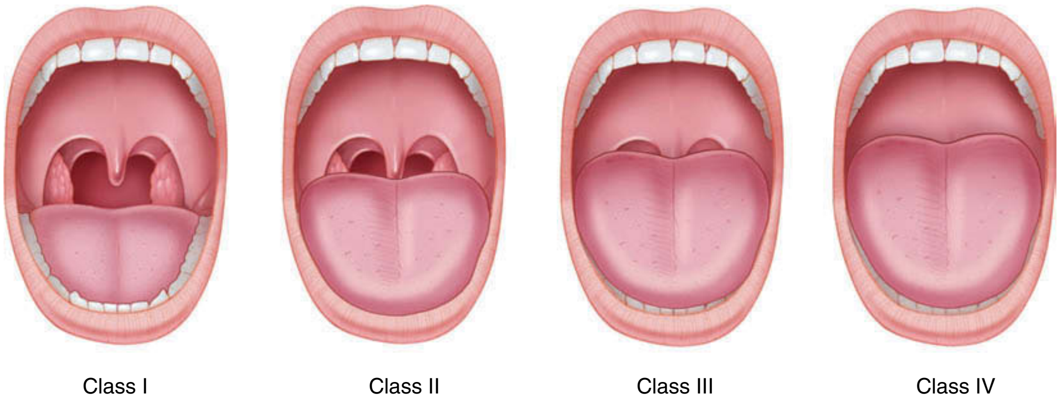

Figure 2.8
Mallampati classification. The classification of tongue size relative to the size of the oral cavity as described by Mallampati and colleagues. Class I: faucial pillars, soft palate, and uvula visualized. Class II: faucial pillars and soft palate visualized, but the uvula is masked by the base of the tongue. Class III: only the base of the uvula can be visualized. Class IV: none of the three structures can be visualized. © Chris Gralapp.

Preparation

Prior to initiating RSI, careful preparation is essential to achieving success. This point cannot be emphasized enough. The *SOAP ME* mnemonic is used to summarize the necessary preparatory steps.

SOAP ME

Suction

Suction should be tested and available at the bedside.

Oxygen

A high-flow O_2 mask and BMV device should be ready for use.

Airway equipment

At least two functioning laryngoscope handles and the appropriately sized and shaped laryngoscope blades should be available. The anticipated blades of choice should be clicked into position to ensure that the light functions properly. An ETT should be chosen based on the patient's anatomy, and one smaller size should be prepared as well. The typical adult male will accept a 7.5- or 8.0-size ETT, the typical adult female a 7.0- or 7.5-size ETT. In children, the ETT size may be estimated by the formula ETT size = 4 + (age in years/4). The ETT cuff should be inflated to test for an air leak. A stylet should be inserted within the ETT to shape it into a configuration that will facilitate insertion into the airway. This configuration varies between physicians, although most prefer a gentle curve at the distal portion to a near 45-degree angle. Care must be taken to ensure that the tip of the stylet does not protrude from the end of the ETT or through the small distal side port (Murphy's eye). Preparation of the ETT with the stylet inserted is recommended, as it is easier to remove a stylet (if not needed) than to add one during RSI.

Pharmacy

The patient should have at least one IV line, and patency should be ensured. The specific RSI medications, proper dosing and sequence of administration should be determined, and the agents drawn up and labeled.

Monitoring equipment

Cardiac blood pressure and pulse oximetry monitoring are mandatory for all patients. If available, an end-tidal CO_2 ($ETCO_2$) monitor should be prepared as well.

Respiratory therapy should be at the bedside, as they play a crucial role in assisting with airway management, including securing the ETT and postintubation care. When dealing with a complicated airway, anesthesiology or ear, nose and throat (ENT) specialists should be called in to assist with airway management.

Preoxygenation

During RSI, the process of direct laryngoscopy and ETT placement precludes simultaneous O_2 delivery to the paralyzed apneic patient. This could lead to detrimental arterial O_2 desaturation (<90%). Preoxygenation establishes a reservoir of oxygen within the patient's lungs and body tissues that allows for a period of prolonged apnea without O_2 desaturation. Administration of 100% O_2 to the patient for 5 minutes prior to paralysis effectively leads to "nitrogen washout," replacing room air (80% nitrogen, 20% O_2) within the lung with nearly 100% O_2. Except in critically ill and elderly patients, administration of 100% O_2 for longer than 5 minutes provides little additional benefit.

The time to desaturation following preoxygenation is determined by the duration of preoxygenation as well as the patient's age and body habitus. Children, late-term pregnant women and obese adults tend to desaturate more rapidly than adults. Following preoxygenation, healthy adults will typically maintain their oxygen saturation >90% for 8 minutes. Healthy children will typically begin desaturating after just 4 minutes.

A nonrebreather O_2 mask delivers O_2 concentrations in the range of 70–75%. A ventilation bag and mask placed over the patient's mouth and nose (without actively bagging) delivers close to 100% O_2 to the patient. In circumstances in which time is limited, a patient can be quickly preoxygenated by taking eight vital-capacity (the largest possible) breaths in rapid succession from a 100% O_2 source.

Pretreatment

During RSI, the act of intubation and the use of succinylcholine (SCh) can lead to a number of adverse effects, including increased intracranial pressure (ICP), increased intraocular pressure, increased intragastric pressure, bronchospasm in patients with reactive airway disease, increased sympathetic discharge and bradycardia (especially in children).

Selected pretreatment medications may be given to mitigate these adverse effects. These medications, their indications, mechanisms of action and doses are summarized in Table 2.3.

Paralysis (with induction)

The next step in RSI is the rapid IV administration of an induction agent followed immediately by an NMBA to induce complete unconsciousness and motor paralysis. These medications are not titrated; rather, they are given IV push to rapidly induce deep sedation and muscular relaxation.

Induction agents

All patients with few exceptions (e.g., benzodiazepine overdose) should receive an induction agent prior to neuromuscular blockade. Induction agents induce complete loss of consciousness prior to NMBA-induced paralysis. Paralysis without sedation can lead to detrimental physiologic and undesirable psychologic sequelae. When combined with NMBAs, induction agents enhance muscle relaxation, thereby creating improved intubating conditions.

Table 2.3 Pretreatment medications

Drug	Indication	Mechanism	Adult dose (IV)	Pediatric dose (IV)	Notes
Lidocaine	↑ ICP, RAD	↓ intracranial response to intubation, mitigates bronchospasm in patients with RAD	1.5 mg/kg	1.5 mg/kg	Don't give in high-grade heart block (Mobitz II or 3rd degree)
Opioid (fentanyl)	↑ ICP, ischemic heart disease, aortic dissection	Blunts sympathetic response to laryngoscopy	3–6 mcg/kg	1–3 mcg/kg	Use with caution in young children
Atropine	Children <1 years Adults receiving a second dose of SCh	Mitigates bradycardic response to SCh	2 mg	0.02 mg/kg (minimum dose 0.1 mg)	Only give prior to first dose of SCh

ICP: intracranial pressure; RAD: reactive airway disease; SCh: succinylcholine.

There is no single induction agent of choice for RSI in the ED. The choice of an induction agent is based on the patient's clinical circumstance and the agent's attributes. The most commonly used induction agents are discussed below and summarized in Table 2.4.

Etomidate

Etomidate is a non-barbiturate sedative-hypnotic agent that provides no analgesia. For most ED patients, it is the induction agent of choice for RSI. It has a rapid onset, brief duration of action and causes minimal respiratory and myocardial depression. Although etomidate is remarkably hemodynamically stable, the dose should be reduced by 50% in hemodynamically unstable patients. Etomidate reduces cerebral blood flow and cerebral metabolic O_2 demand without adversely affecting cerebral perfusion pressure. Due to its cerebroprotective effects and hemodynamic stability, etomidate is an excellent induction agent for patients with elevated ICP. Side effects of etomidate include vomiting, pain at the injection site, myoclonic movements and hiccups. Adverse effects from cortisol suppression have not been reported with single-dose injection used in RSI.

Ketamine

Ketamine is a dissociative anesthetic derived from phencyclidine (PCP) that induces a cataleptic state rather than true unconsciousness. Ketamine provides analgesia, amnesia and anesthesia, while preserving protective reflexes (i.e., airway). Ketamine causes a rise in heart rate, blood pressure, myocardial consumption and bronchodilation by stimulating the endogenous release of catecholamines. For these reasons, it is the induction agent of choice for hypotensive (e.g., sepsis), hypovolemic (e.g., hemorrhage) and bronchospastic patients requiring intubation. Due to the sympathetic stimulation, care should be taken in patients with ischemic heart disease. Because ketamine increases ICP, cerebral blood flow, and cerebral metabolic rate, it is generally avoided in patients with potentially increased ICP. Ketamine is known to enhance laryngeal reflexes, increase airway secretions and precipitate laryngospasm. Therefore, atropine 0.02 mg/kg IV may be given in conjunction with ketamine to promote a drying effect. Ketamine may produce an unpleasant emergence phenomenon, including hallucinations or frightening dreams in the first 3 hours after awakening. Such reactions are more common in adults than children and can be reduced through the concomitant administration of a benzodiazepine such as lorazepam (0.05 mg/kg) after intubation.

Thiopental and methohexital

The barbiturates thiopental and methohexital are short-acting sedative-hypnotic agents that provide no

Table 2.4 Induction agents

Induction agent	Induction dose (IV)	Onset of action	Duration of action	Benefits	Precautions
Barbiturates					
Thiopental	3–6 mg/kg (adult) 1–3 mg/kg (elderly)	<30 sec	5–10 min	↓ ICP	↓ BP Laryngospasm
Methohexital	1–3 mg/kg	<30 sec	5–10 min	↓ ICP Short duration	↓ BP Laryngospasm Seizures
Benzodiazepines					
Midazolam	0.2–0.3 mg/kg	30–60 sec	15–30 min	Reversible Amnestic Anticonvulsant	Apnea No analgesia Variable dosing
Etomidate	0.3 mg/kg	15–45 sec	3–12 min	↓ ICP Rarely ↓ BP	Myoclonic jerks Vomiting No analgesia
Ketamine	1–2 mg/kg	45–60 sec	10–20 min	↑ BP Bronchodilator Dissociative amnesia	↑ Secretions ↑ ICP Emergence phenomenon

BP: blood pressure; ICP: intracranial pressure.

analgesia. These agents have a short onset of action and induce rapid depression of central nervous system (CNS) activity. They also reduce ICP (by reducing cerebral blood flow) and provide cerebroprotective effects (by reducing cerebral metabolic O_2 consumption) while still maintaining cerebral perfusion pressure. As a result, both barbiturates are excellent induction agents for hemodynamically stable patients with increased ICP (e.g., intracranial hemorrhage) or status epilepticus. Their propensity to induce significant hypotension from myocardial depression and venodilation is the reason to avoid these agents in hypotensive patients. Other side effects of thiopental include central respiratory depression, histamine release (avoid use in asthmatic patients), and tissue injury and necrosis with extravasation. Methohexital is shorter-acting and more potent than thiopental, and not surprisingly associated with more profound hypotension and respiratory depression.

Midazolam

Benzodiazepines cause amnesia, anxiolysis, central muscle relaxation, sedation and hypnosis. They also have strong anticonvulsant effects. As induction agents, their strengths are the ability to promote sedation and amnesia, and their primary weakness is their gender- and age-dependent dosing variability. Of the benzodiazepines, midazolam is best suited as an induction agent because of its rapid onset of action. Like other benzodiazepines, midazolam is a myocardial depressant and reduces systemic vascular resistance, and it should be used with caution in hemodynamically unstable and elderly patients. Although midazolam may be used as a primary induction or adjunctive agent during RSI, it is more commonly utilized for sedation in combination with an analgesic agent in patients who are intubated. When used as an induction agent, midazolam is frequently underdosed.

Neuromuscular blockade

NMBAs (neuromuscular blocking agents) are used to paralyze the patient, facilitating rapid endotracheal intubation. They do not provide analgesia, sedation or amnesia. There are two classes of NMBAs: depolarizing and non-depolarizing.

The ideal NMBA would have a rapid onset, short duration of action and few adverse side effects. Succinylcholine (SCh), a *depolarizing* NMBA, comes closest to meeting these traits and is the paralytic agent of choice for most intubations in the ED. SCh binds tightly to acetylcholine receptors at the neuromuscular junction, causing depolarization of the motor endplate and muscle contraction. Clinically, this initially manifests as muscle fasciculations followed by paralysis. IV administration of SCh results in muscle fasciculations within 10–15 seconds followed by complete paralysis after 45–60 seconds. Because of its short duration of action, patients may begin spontaneously breathing within 6–10 minutes.

The dose of SCh is 1.5 mg/kg rapid IV push in adults. In children <10 years of age, the recommended dose is 2 mg/kg rapid IV push. There is little harm in giving too much SCh; however, giving too little SCh can lead to inadequate paralysis and difficulty intubating.

The main drawbacks to SCh are its side effects, including muscle fasciculations, bradycardia, hyperkalemia, prolonged neuromuscular blockade, trismus (masseter muscle spasm) and malignant hyperthermia. The bradycardia that follows the administration of SCh most commonly occurs in children <1 year of age and can be mitigated by pretreatment with atropine (0.02 mg/kg). Under usual circumstances, SCh induces a small but clinically insignificant rise in serum potassium of 0.5 mEq/L. However, in large burns, crush injuries, denervation or neuromuscular disorders, and intra-abdominal sepsis, the administration of SCh may lead to an exaggerated rise in potassium levels of 5–10 mEq/L and result in hyperkalemic dysrhythmias or cardiac arrest. Fortunately, the hyperkalemia risk is not immediate in these patients but occurs typically 5 days after the event, depending on the injury or underlying process.

Non-depolarizing NMBAs compete with acetylcholine for receptors at the neuromuscular junction, thereby causing paralysis. Although these agents are commonly used for postintubation paralysis, they may also be used as the primary RSI paralytic agent in specific patient populations or in patients who have a contraindication to SCh. These agents have fewer side effects than SCh but are generally less effective for intubation because of their delayed time to paralysis, prolonged duration of action, or both.

Of the non-depolarizing NMBAs, rocuronium has the most favorable profile for RSI. Like SCh, rocuronium has a short onset of action (45–60 seconds at a dose of 1 mg/kg IV) but an extended duration of action like other non-depolarizing NMBAs. Research is underway to evaluate reversal agents for non-depolarizing NMBAs and one such drug, sugammadex, has shown promise.

Specific attributes of the depolarizing and non-depolarizing NMBAs are listed in the Table 2.5.

Table 2.5 Neuromuscular blocking agents

Neuromuscular blocking agent	Intubating dose (IV)	Onset	Duration
Depolarizing agent			
Succinylcholine	1.5 mg/kg (adult) 2 mg/kg (child)	45–60 sec	6–12 min
Non-depolarizing agent			
Rocuronium	1 mg/kg	50–70 sec	30–60 min
Vecuronium	0.15 mg/kg	90–120 sec	60–75 min
Pancuronium	0.1 mg/kg	100–150 sec	120–150 min

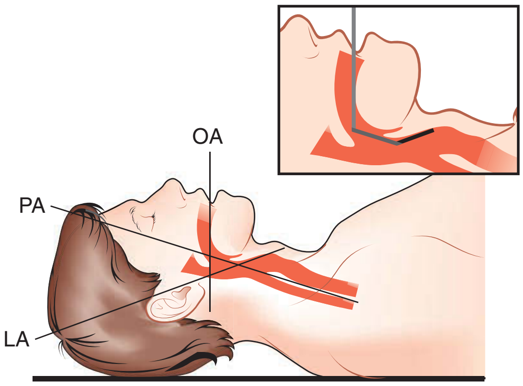

Figure 2.9
Head on bed, neutral position. PA: pharyngeal axis; OA: oral axis; LA: laryngeal axis. Reproduced with permission from Walls RM, Murphy RF, *Manual of Emergency Airway Management*, 3rd ed, and *Companion Manual to the Airway Course* (www.theairwaysite.com). Lippincott Williams & Wilkins, Philadelphia, 2008.

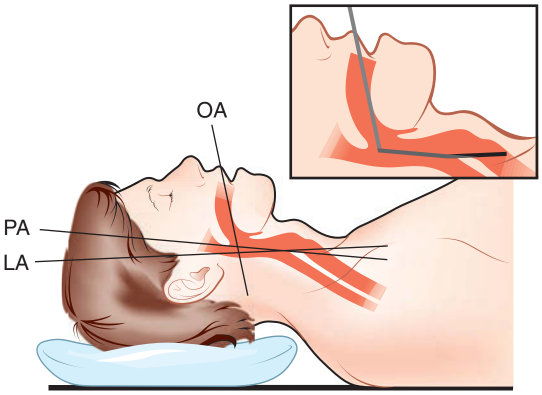

Figure 2.10
Head elevated on pad, neutral position. PA: pharyngeal axis; OA: oral axis; LA: laryngeal axis. Reproduced with permission from Walls RM, Murphy RF, *Manual of Emergency Airway Management*, 3rd ed, and *Companion Manual to the Airway Course* (www.theairwaysite.com). Lippincott Williams & Wilkins, Philadelphia, 2008.

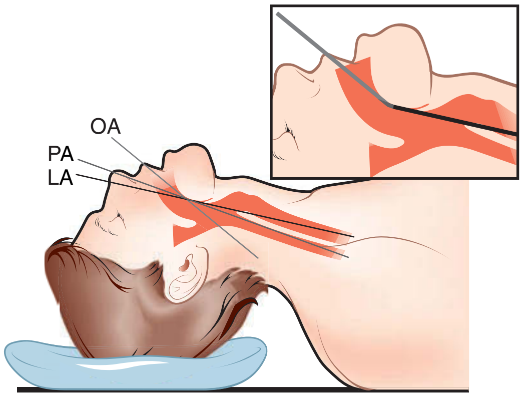

Figure 2.11
Head elevated on pad, head extended on neck. PA: pharyngeal axis; OA: oral axis; LA: laryngeal axis. Reproduced with permission from Walls RM, Murphy RF, *Manual of Emergency Airway Management*, 3rd ed, and *Companion Manual to the Airway Course* (www.theairwaysite.com). Lippincott Williams & Wilkins, Philadelphia, 2008.

Positioning

Based on the patient's age, anatomy and other conditions (cervical arthritis, cervical spine precautions), the patient should be carefully positioned in the manner that increases the odds of successful intubation.

The airway has three separate axes: the oral, pharyngeal and laryngeal. In the neutral position, these axes are misaligned (Figure 2.9). However, with proper positioning prior to laryngoscopy, the clinician can obtain a clear line of site to the glottis.

Placing a small pillow under the patient's occiput flexes the lower cervical spine relative to the torso, aligning the pharyngeal and laryngeal axes (Figure 2.10). Positioning the patient in the "sniffing" position with extension of the head on the neck aligns all the three axes (Figure 2.11).

Patients with possible cervical spine injury should be maintained in the neutral position.

Protection

Following administration of induction and paralytic agents, the patient will predictably lose consciousness and become apneic – usually 45 seconds after the paralytic agent. Just as the patient loses consciousness, an assistant should apply firm backward pressure to the patient's cricoid cartilage (Sellick's maneuver). Although its efficacy has been questioned, Sellick's maneuver theoretically compresses the esophagus and prevents passive regurgitation of the gastric contents (Figure 2.12).

Sellick's maneuver is typically maintained until the ETT has been placed, its position verified, and the cuff inflated. If the application of cricoid pressure impairs visualization of the vocal cords, it should be released or modified. If Sellick's maneuver is applied too early, the patient may find it uncomfortable or vomit. This maneuver should be discontinued if the patient is actively vomiting because of the risk of esophageal rupture.

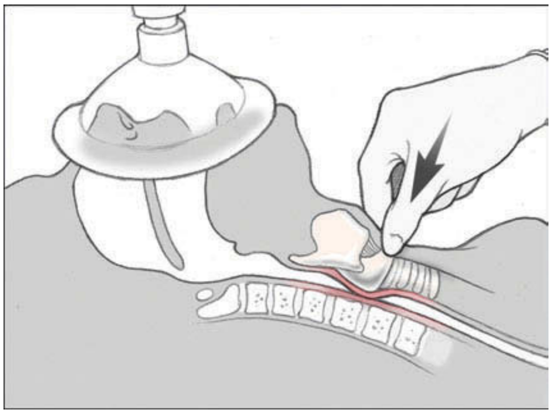

Figure 2.12
Cricoid pressure (Sellick's maneuver). © Chris Gralapp.

Placement

After the administration of SCh, the patient will predictably have muscle fasciculations followed by paralysis and apnea. If the patient has been adequately preoxygenated, arterial O_2 saturations will remain normal despite apnea. Gently grasping the patient's mandible and checking for flaccidity can confirm complete muscular paralysis. It is important to wait until the patient is completely paralyzed before proceeding with intubation.

With the laryngoscope in the left hand, the mouth is opened with the right hand. The laryngoscope is gently inserted into the right side of the patient's mouth, and the tongue is displaced to the left. The curved (Macintosh) blade is slid into the vallecula; the straight (Miller) blade is positioned below the epiglottis. The laryngoscope handle is advanced along the axis of the blade at an angle of 45° to the patient's body. Care should be taken not to use the teeth as a fulcrum for the laryngoscope.

If the glottic aperture is not readily visible, the intubator or an assistant may perform the BURP (Backward, Upward, Rightward Pressure) maneuver. Manual application of BURP and the resulting displacement of the thyroid cartilage backwards against the cervical vertebrae, upward or as superiorly as possible, and laterally to the right have been found to significantly improve the view of the glottis during laryngoscopy (Figure 2.13).

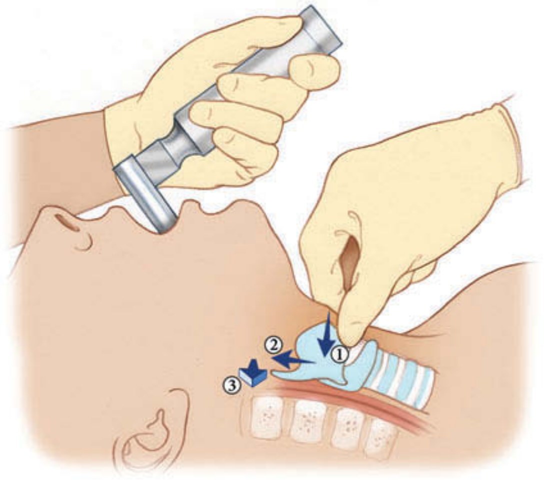

Figure 2.13
BURP maneuver: Backward, upward, rightward pressure. © Chris Gralapp.

With a clear view of the glottis, the right hand gently inserts the ETT until the cuff is about 2–3 cm past the vocal cords. In the typical adult male, the 23-cm marker of the ETT will be located at the corner of the mouth (21 cm in women). Once in place, the stylet should be removed and the cuff inflated until there is no audible air leak with BMV.

Adequate preoxygenation will allow the laryngoscopist several attempts at intubation (without intervening BMV) before arterial O_2 desaturation occurs. A dedicated team member should be focused on the patient's cardiac rhythm, blood pressure and O_2 saturation during laryngoscopy, and should alert the intubator to any abnormalities. After any unsuccessful attempt, always recheck the patient's position and make needed adjustments. Consider changing the size or type of laryngoscope blade. It is important to change "something" prior to a second look to ensure the same problem is not encountered.

Proof: confirmation of endotracheal tube placement

Proper placement of the ETT within the trachea needs to be confirmed after every intubation. Failure to recognize an esophageal intubation can be disastrous. Sellick's maneuver should not be released until confirmation of correct ETT placement.

Methods used to confirm correct ETT placement include clinical assessment, pulse oximetry, $ETCO_2$ detection and aspiration techniques. Chest radiography should be used to assess ETT position but cannot confirm ETT placement within the trachea. Because the esophagus lies directly behind the trachea, an ETT placed in the esophagus may appear to be within the trachea on an AP chest X-ray (CXR).

Clinical assessment

Classically, a combination of clinical observations has been used to confirm correct ETT placement. These include:

1. Direct visualization of the ETT passing through the vocal cords during intubation
2. Auscultation of clear and equal breath sounds over both lung fields
3. Absence of breath sounds when auscultating over the epigastrium
4. Observation of symmetrical chest rise during ventilation
5. Observation of condensation ("fogging") within the ETT during ventilation.

Although these clinical findings should be assessed in every intubated patient, they are prone to error and should not be the sole means for confirming ETT placement.

Pulse oximetry

Continuous noninvasive pulse oximetry should be standard for every patient being intubated. A drop in the measured O_2 saturation following intubation is worrisome for an esophageal intubation; if the patient was adequately preoxygenated, this drop may be delayed for several minutes, giving health care providers a false sense of security. In certain patients (i.e., hypotensive), O_2 saturation measurements may be unreliable or difficult to detect. Although pulse oximetry is important, it should not be the primary indicator of successful ETT placement.

End-tidal carbon dioxide ($ETCO_2$) detection

Immediately following intubation, an $ETCO_2$ detection device should be attached to the ETT and assessed through six manual ventilations. The presence of exhaled CO_2 after six manual ventilations is an effective and reliable method for confirming proper placement of the ETT within the airway. False-positive $ETCO_2$ detection may occur if the tube is placed just above the glottis. False-negative $ETCO_2$ detection may occur (despite ETT placement within the trachea) after cardiopulmonary arrest and profound circulatory collapse, as CO_2 production and delivery to the lungs abruptly declines.

Colorimetric $ETCO_2$ detectors are small disposable devices that connect between the bag and the ETT. When the device detects $ETCO_2$, its colorimetric indicator changes from purple to yellow; the absence of this color change indicates the tube is incorrectly placed in the esophagus.

Qualitative $ETCO_2$ detection devices use a light indicator with an audible alert, as opposed to a color change, to indicate the presence of exhaled CO_2. Many of these devices have alarms that sound if the detection of $ETCO_2$ ceases.

Quantitative $ETCO_2$ detectors perform capnography, the graphic display of CO_2 concentrations seen as a wave form on the monitor, or capnometry, the measurement and display of CO_2 concentrations.

Aspiration devices

The *bulb* and *syringe aspiration* devices have a secondary role for confirmation of proper ETT placement. These appliances work based on the principle that the trachea is a rigid air-filled structure, while the esophagus has collapsible walls. Once the device is attached to the ETT, attempts to draw air through an esophageal intubation will meet resistance, whereas air will flow freely through an ETT in the trachea. Although these aspiration techniques are easy to perform, they are not as reliable as $ETCO_2$ detection; however, they have utility as backup devices in patients with cardiopulmonary arrest.

Postintubation management

After correct placement of the ETT in the trachea has been verified, a few "housekeeping" issues must be addressed. The ETT needs to be secured. The patient's blood pressure and other vitals should be repeated frequently. Bradycardia following intubation should be assumed due to esophageal intubation and resulting hypoxia. Hypertension postintubation suggests inadequate sedation. Hypotension may be the result of a tension pneumothorax, decreased venous return, a cardiac cause, or the induction agent.

Configure the mechanical ventilator according to the patient's size and needs. A postintubation CXR will assess ETT position (depth of placement) and the condition of the patient's lungs. Proper tube depth is generally 2–3 cm above the carina. Insertion of the ETT into the right mainstem bronchus is a common complication (Figure 2.14).

Following intubation, long-term sedation and analgesia using a benzodiazepine and opioid agent will facilitate mechanical ventilation. IV diazepam (0.2 mg/kg), lorazepam (0.05–0.1 mg/kg) or midazolam (0.1–0.2 mg/kg) may be given initially for sedation and repeated for any signs of awareness. An opioid agent such as fentanyl (3–5 mcg/kg) or morphine sulfate (0.2 mg/kg) should be added for additional patient comfort. Pancuronium (0.1 mg/kg) or vecuronium (0.1 mg/kg) may be used if long-term paralysis is desired; a repeat dose (one-third the initial dose) may be given after 45–60 minutes if motor activity is detected.

Immediate "crash" intubation

Patients with respiratory arrest, agonal respirations or deep unresponsiveness require immediate intubation without the use of supplemental medications. The advantages of this approach are technical ease and immediacy.

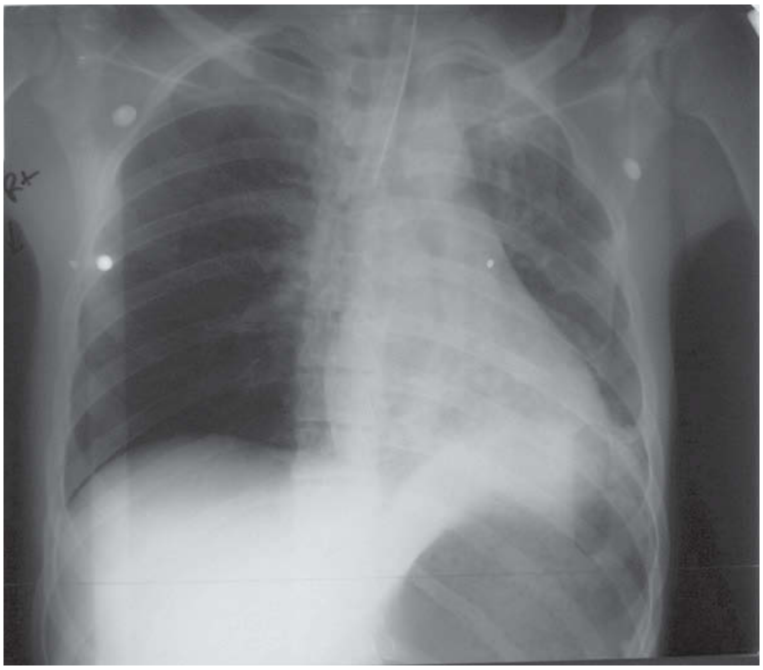

Figure 2.14
Right mainstem bronchus intubation. AP supine chest radiograph on a trauma board showing an endotracheal tube in the right mainstem bronchus, hyperinflation of the right lung and marked loss of volume in the left lung.

Disadvantages include the potential for increased ICP from the stress of intubation, as well as possible emesis and aspiration.

Awake oral intubation

Awake oral intubation is a technique utilizing liberal topical airway anesthesia (i.e., spray, nebulization or local anesthetic nerve block) and mild IV sedation prior to inspection or intubation of an "awake" patient's airway. The approach conveniently allows for the preservation of the patient's airway reflexes and spontaneous breathing while the laryngoscopist takes a gentle look at the glottis, vocal cords and internal airway anatomy. Whether to intubate the patient immediately or defer for a controlled RSI depends on the potential for progressively increased airway difficulty or compromise. One might elect to immediately intubate a patient with an airway burn or anaphylaxis with progressive swelling.

The classic scenario for employing this technique is the patient with distorted upper airway anatomy resulting from blunt or penetrating anterior neck trauma. Under these circumstances, intubation by RSI may be unsuccessful or impossible and subsequent BMV may allow air to enter the neck via the airway injury, complicating further management. Disadvantages of the awake oral intubation technique include oversedation, discomfort and stress, and potential for deleterious effects in patients with cardiac disorders or increased ICP.

Blind nasotracheal intubation

Although commonly employed previously, blind nasotracheal intubation (BNTI) has lost ground to other more effective airway approaches. When compared with RSI, BNTI consumes more time, fails more often, involves the passage of a smaller ETT, and results in a higher number of complications.

BNTI may be the preferred route of intubation in clinical circumstances in which RSI is not advisable and alternatives (e.g., fiberoptic) are not available. In the patient with anatomic features that may pose a challenge to RSI and BMV, awake BNTI can be performed while preserving the patient's spontaneous respirations.

BNTI is contraindicated in the apneic patient, since air movement is essential to tube placement. BNTI is also contraindicated in patients with the possibility of cribriform plate, basilar skull, or midface injuries out of concern that the tube may enter the cranial vault. Patients with bleeding disorders or coagulopathy may develop massive epistaxis from BNTI.

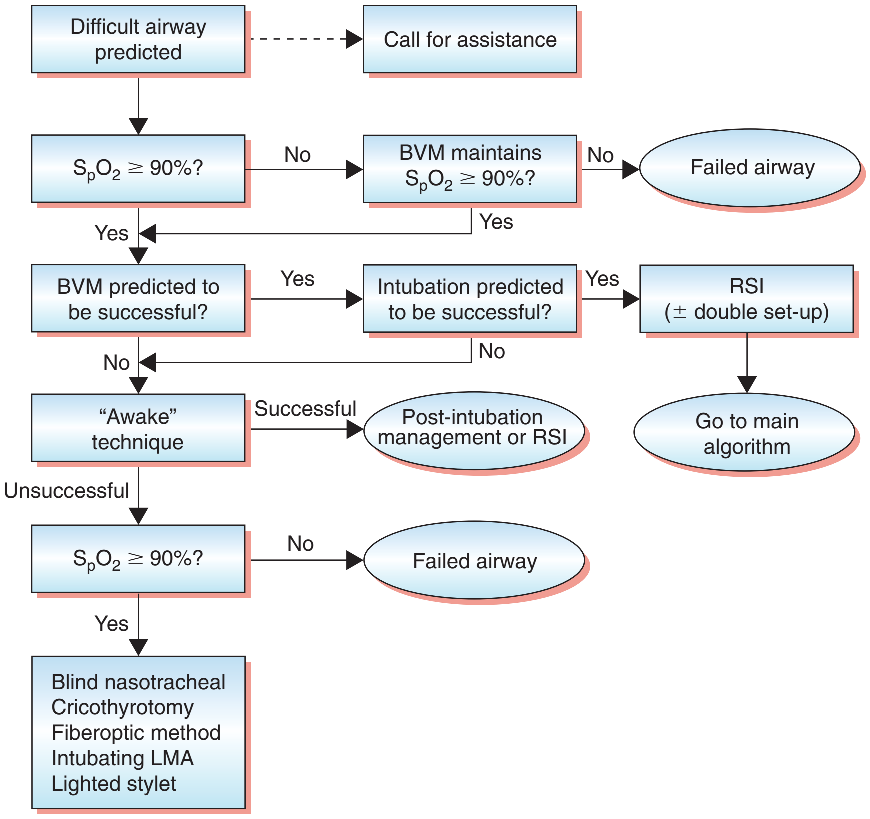
Difficult airway predicted
Call for assistance
$S_pO_2 \geq 90\%$?
No
BVM maintains $S_pO_2 \geq 90\%$?
No
Failed airway
Yes
Yes
BVM predicted to be successful?
Yes
Intubation predicted to be successful?
Yes
RSI (± double set-up)
No
No
"Awake" technique
Successful
Post-intubation management or RSI
Go to main algorithm
Unsuccessful
$S_pO_2 \geq 90\%$?
No
Failed airway
Yes
Blind nasotracheal
Cricothyrotomy
Fiberoptic method
Intubating LMA
Lighted stylet

Figure 2.15
Algorithm for the difficult airway. LMA: laryngeal mask airway; BVM: bag-valve-mask; RSI: rapid sequence intubation; SpO2: saturated pressure of oxygen measured with pulse oximetry. Reproduced with permission from Walls RM, Murphy RF, *Manual of Emergency Airway Management*, 3rd ed, and *Companion Manual to the Airway Course* (www.theairwaysite.com). Lippincott Williams & Wilkins, Philadelphia, 2008.

The difficult airway

It has been estimated that between 1–3% of patients present with a "difficult airway," defined as difficulty securing the airway under direct laryngoscopic vision. Before administering NMBAs, the emergency physician should always assess the likelihood of a difficult intubation and the probability for success. Every intubation should be assumed difficult, especially in the pediatric population, and a back-up plan should be formulated prior to proceeding. Success or failure is often directly related to the airway manager's ability to anticipate problems, prepare for the worst-case scenario and address failure (Figure 2.15).

The failed airway

The failed airway may be clinically defined in two manners:

1. The *cannot intubate, can oxygenate* scenario occurs when a skilled airway manager fails to intubate on three attempts but can successfully BMV the patient.
2. The *cannot intubate, cannot oxygenate* scenario arises when the failure to intubate, regardless of the number of attempts, occurs in the face of O_2 saturations that cannot be maintained above 90% using BMV.

The management of the failed airway is dictated by whether or not the patient can be oxygenated (Figure 2.16).

Devices and techniques for the difficult or failed intubation

This section briefly describes some of the devices and techniques that may be employed in the event of a difficult or failed intubation.

Extraglottic devices

Laryngeal mask airway

The laryngeal mask airway (LMA) is a modified ETT with an inflatable, oval collar ("laryngeal mask") at its base (Figure 8.2). The LMA is blindly inserted into

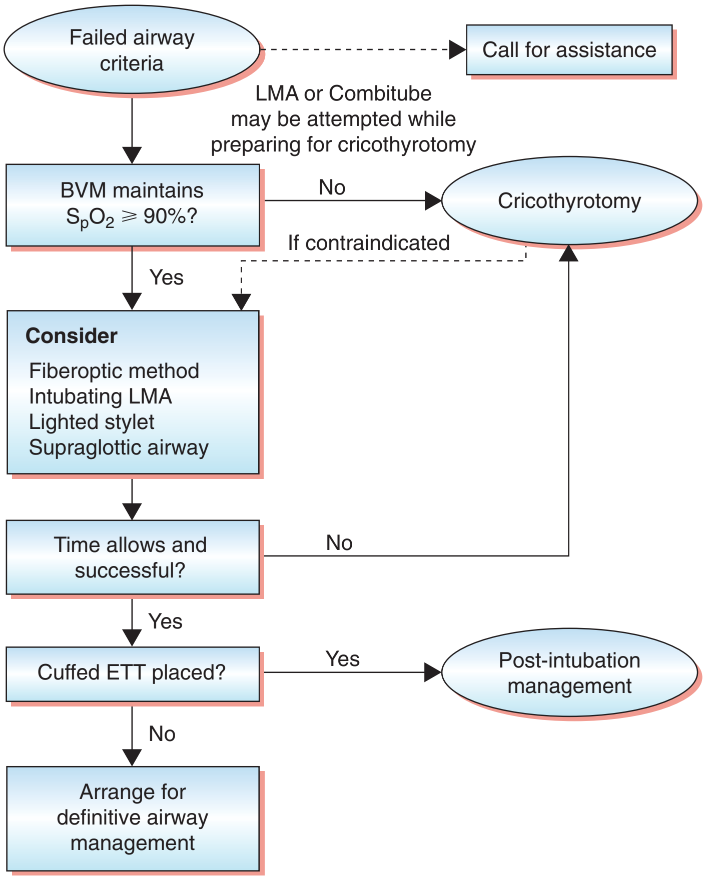
Failed airway criteria
Call for assistance
LMA or Combitube may be attempted while preparing for cricothyrotomy
BVM maintains $S_pO_2 \geqslant 90\%$?
No
Cricothyrotomy
If contraindicated
Yes
Consider
Fiberoptic method
Intubating LMA
Lighted stylet
Supraglottic airway
Time allows and successful?
No
Yes
Cuffed ETT placed?
Yes
Post-intubation management
No
Arrange for definitive airway management

Figure 2.16
Algorithm for the failed airway. ETT: endotracheal tube; BVM: bag-valve mask; SpO2: saturated pressure of oxygen measured with pulse oximetry. LMA: laryngeal mask airway. Reproduced with permission from Walls RM, Murphy RF, *Manual of Emergency Airway Management*, 3rd ed, and *Companion Manual to the Airway Course* (www.theairwaysite.com). Lippincott Williams & Wilkins, Philadelphia, 2008.

the pharynx until the collar covers the glottic opening. Inflation of the collar provides a seal that allows tracheal ventilation with minimal gastric insufflation. Although these devices are relatively easy to use, they do not prevent aspiration. However, the LMA can serve as a rescue ventilation device, equivalent or superior to bag-mask ventilation, until a definitive airway can be established.

The intubating LMA (ILMA) facilitates both rescue ventilation and blind endotracheal intubation. Following successful ILMA placement, an ETT can be advanced through the device and into the trachea with success rates comparable to those of direct laryngoscopy.

Combitube™

The Combitube™ is a dual-lumen, dual-cuffed esophageal/tracheal airway (Figure 8.1). One lumen functions as an esophageal airway while the other performs as a tracheal airway. After blind insertion of the Combitube, the device typically enters the esophagus; however, the presence of dual lumens allows ventilation even following inadvertent tracheal entry.

King LT®

The King LT® is a single-lumen, dual-cuffed airway with ventilation outlets between the pharyngeal and esophageal cuffs. The King LT is inserted blindly, and a single pilot balloon inflates both cuffs simultaneously. Though similar to the Combitube, the King LT is shorter, easier to insert and easier to inflate.

Video laryngoscopy

Video laryngoscopes, consisting of a micro videocamera and a modified laryngoscope, transmit images of the glottis to an external monitor, allowing intubation to be performed while looking at the video screen. Compared with traditional laryngoscopy, video laryngoscopes, such as the GlideScope®, provide superior glottic views and greater airway anatomic detail without requiring alignment of the axes. As a result, the GlideScope is associated with high intubation success rates and less cervical spine movement. Although rigorous trials to assess performance are limited, clinical experience, including that of the authors, indicates these devices are highly effective.

Fiberoptic intubation

Fiberoptic techniques for endotracheal intubation have emerged as invaluable tools for management of the difficult airway. Devices such as flexible fiberoptic intubating bronchoscopes and fiberoptic intubating stylets require considerable technical skill and repeated practice to maintain speed and success.

Lighted stylet intubation

The lighted stylet apparatus utilizes transillumination of the soft tissues of the neck to signify correct ETT placement within the trachea. Due to the anterior location of the trachea relative to the esophagus, a well-defined, circumscribed glow can readily be seen in the anterior neck when the ETT and light enter the glottic opening. If the tip of the tube is placed in the esophagus, the light glow is diffuse and poorly seen.

Retrograde intubation

Retrograde intubation involves needle puncture of the cricothyroid membrane followed by threading a guidewire retrograde through the vocal cords and out the nose or mouth. The wire is then used to guide the ETT through the glottis before it is removed.

Digital intubation

Digital intubation is a technique in which the index and long fingers of the nondominant hand are used to identify the epiglottis and then manually direct an ETT into the larynx. This technique requires a profoundly unresponsive patient.

Surgical airway

Surgical airway management, unlike conventional airway management, entails the creation of an opening in the trachea to provide oxygenation and ventilation. Proficiency with surgical airway techniques can mean the difference between life and death.

Cricothyrotomy

When a surgical airway is absolutely necessary, cricothyrotomy is the procedure of choice. Cricothyrotomy allows emergent placement of an ETT or cuffed tracheostomy tube through a surgical opening in the cricothyroid membrane (Figure 2.17). The primary indication for cricothyrotomy is the need for a definitive airway when orotracheal or nasotracheal intubation fails or is contraindicated, and bag-mask ventilation cannot maintain an adequate oxygen saturation. A classic example is the patient with severe facial trauma in whom conventional airway management is extremely complicated or unfeasible.

The primary contraindication to cricothyrotomy is young age. Due to anatomic considerations, the procedure should be avoided in children <10–12 years of age. Other contraindications to cricothyrotomy include preexisting tracheal or laryngeal pathology, anatomic obliteration of the landmarks (e.g., hematoma), coagulopathy and operator inexperience with the procedure. Complications of cricothyrotomy include incorrect airway placement, hemorrhage, tracheal or laryngeal injury, infection, pneumomediastinum, subglottic stenosis and voice change.

Transtracheal jet ventilation (TTJV)

An alternative surgical airway procedure is needle cricothyrotomy with percutaneous TTJV (Figure 2.18). In this

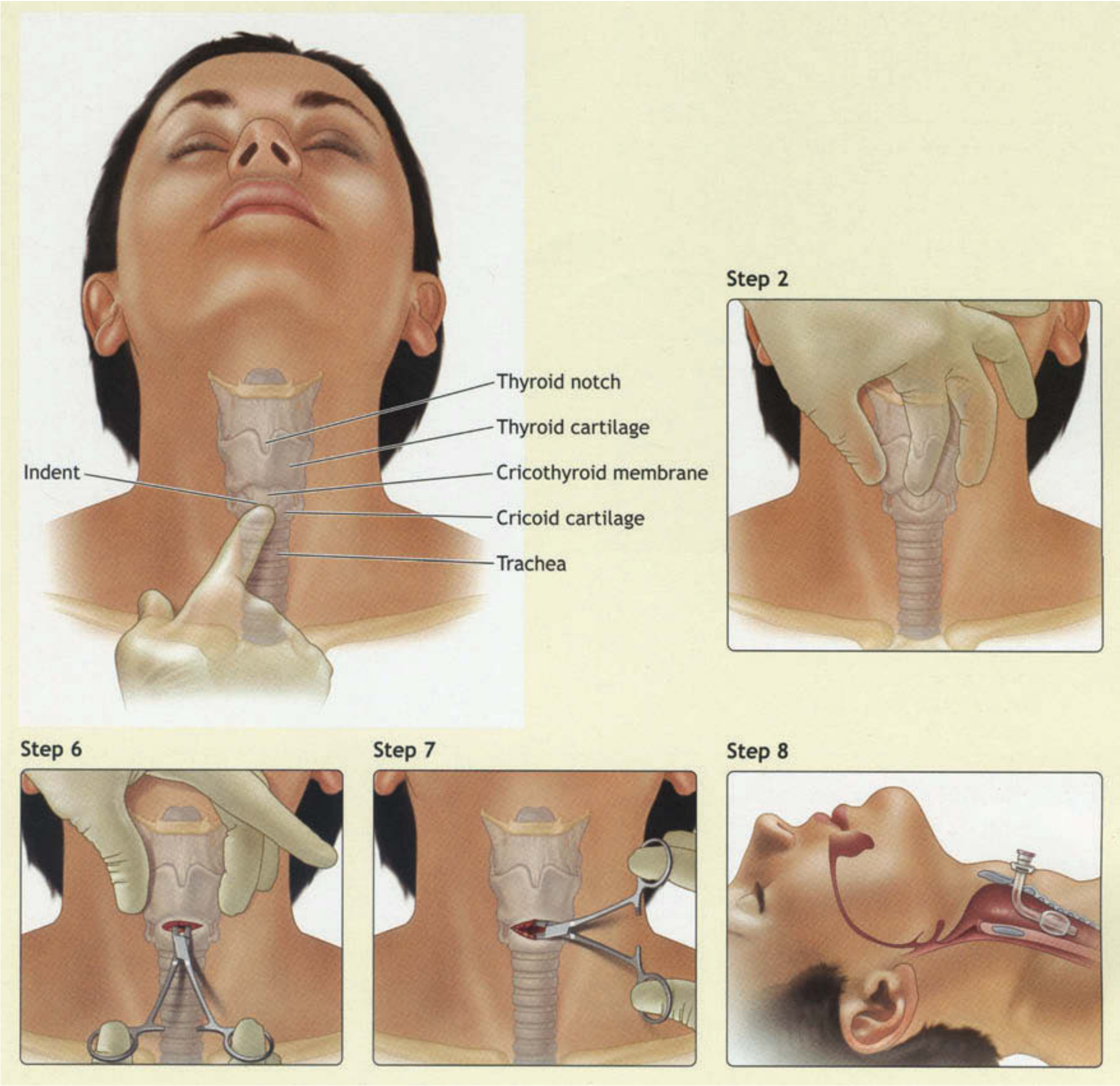

Figure 2.17
Surgical cricothyrotomy. Reproduced with permission from *ATLS: Advanced Trauma Life Support for Doctors Student Manual*, 8th edition, American College of Surgeons, 2008

technique, a transtracheal catheter is inserted through the cricothyroid membrane into the trachea and connected to a jet ventilation system which includes high-pressure tubing, an oxygen source at 50 psi, and an in-line one-way valve for intermittent administration of oxygen. One hundred percent oxygen is then delivered at 12–20 bursts per minute. The inspiratory phase should last 1 second, whereas the expiratory phase lasts 2–4 seconds.

Advantages of this technique include simplicity, safety and speed. There is typically less bleeding compared with cricothyrotomy, and age is not a contraindication, making it the preferred surgical airway in children <12 years.

During TTJV, the upper airway must be free of obstruction to allow for complete exhalation, or else the patient may develop barotrauma from air stacking. All patients receiving TTJV should have an oral and nasal airway placed. Unlike cricothyrotomy, TTJV does not provide complete airway protection; it should be considered a temporizing measure until a definitive airway can be established.

Special patients

Pediatric

Though the principles of airway management in adults and children are the same, a number of age-related differences must be accounted for when managing the pediatric airway. Specific anatomic differences between adults and children and their clinical significance in airway management are summarized in Table 2.6 and Figure 2.19.

Physiologically, pediatric patients have a higher rate of O_2 consumption and smaller functional residual capacity; therefore, they tend to desaturate more rapidly than adults. Compared with adults, children tend to have

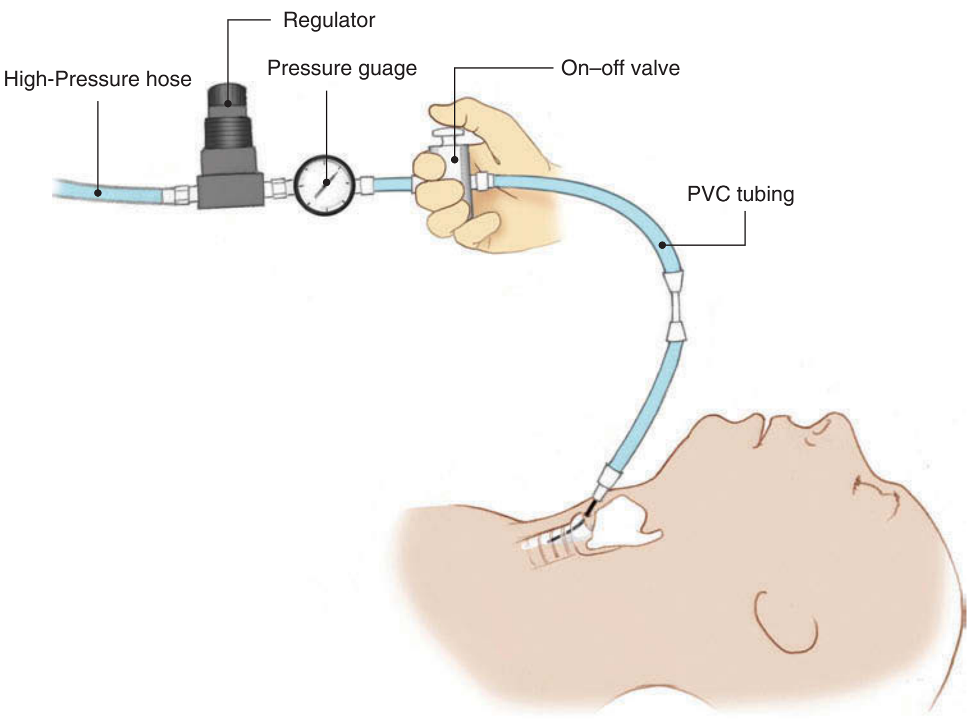

Figure 2.18
Transtracheal jet ventilation. PVC: polyvinyl chloride. © Chris Gralapp.

a shortened period of protection from hypoxia following preoxygenation, and infants and small children may require BMV during RSI to avoid hypoxia.

Airway equipment selection is based on the child's weight and length (Table 2.7). A child's ETT size can be estimated by the size of his/her external naris, the diameter of his/her little finger, or the formula ETT size = 4 + (age in years/4). The depth of ETT placement may be remembered as approximately three times ETT size or (age in years/2) + 12.

Drug dosing in children and the choice of medications for RSI are also based on a child's age and weight. Of note, the dose of SCh is higher in children (2 mg/kg) than in adults. Atropine (0.02 mg/kg) may be administered to

Table 2.6 Anatomical differences between adults and children

Anatomy	Clinical significance
Large intraoral tongue occupying relatively large portion of the oral cavity	Straight blade preferred over curved to push distensible anatomy out of the way to visualize the larynx
High tracheal opening: C-1 in infancy vs. C-3 to C-4 at age 7, C-4 to C-5 in the adult	High anterior airway position of the glottic opening compared with that in adults
Large occiput that may cause flexion of the airway, large tongue that easily collapses against the posterior pharynx	Sniffing position is preferred. The larger occiput actually elevates the head into the sniffing position in most infants and children. A towel may be required under shoulders to elevate torso relative to head in small infants
Cricoid ring is the narrowest portion of the trachea as compared with the vocal cords in the adult	Uncuffed tubes provide adequate seal because they fit snugly at the level of the cricoid ring Correct tube size essential because variable expansion cuffed tubes not used
Consistent anatomical variations with age with fewer abnormal variations related to body habitus, arthritis, chronic disease	Younger than 2 years, high anterior; 2 to 8 years, transition; older than 8 years, small adult
Large tonsils and adenoids may bleed; more acute angle between epiglottis and laryngeal opening results in nasotracheal intubation attempt failures.	Blind nasotracheal intubation not indicated in children; nasotracheal intubation failure
Small cricothyroid membrane	Needle cricothyrotomy difficult, trachea is the landmark, surgical cricothyrotomy impossible in infants and small children

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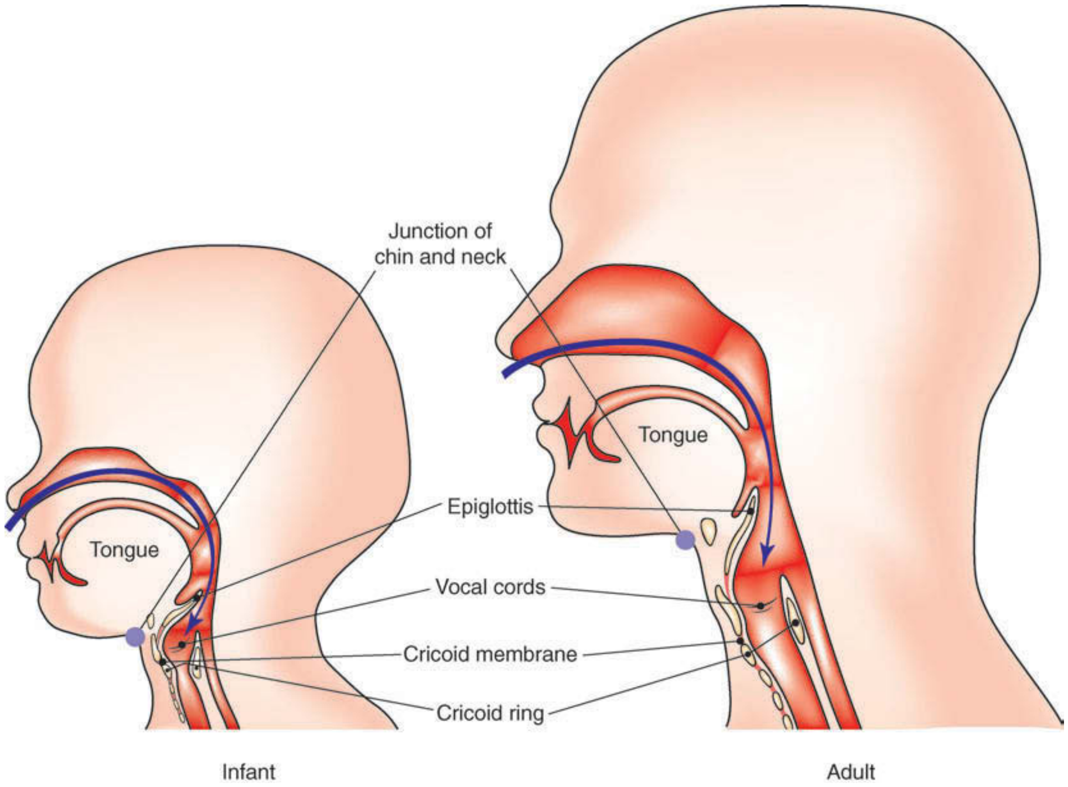

Figure 2.19
Anatomic airway differences between children and adults. The anatomic differences particular to children include: 1. Higher, more anterior position for the glottic opening. (Note the relationship of the vocal cords to the chin/neck junction). 2. Relatively larger tongue in the infant, which lies between the mouth and glottic opening. 3. Relatively larger and more floppy epiglottis in the child. 4. Cricoid ring is the narrowest portion of the pediatric airway; in adults, it is the vocal cords. 5. Position and size of the cricothyroid membrane in the infant. 6. Sharper, more difficult angle for blind nasotracheal intubation. 7. Larger relative size of the occiput in the infant. Reproduced with permission from Walls RM, Murphy RF, *Manual of Emergency Airway Management*, 3rd ed, and *Companion Manual to the Airway Course* (www.theairwaysite.com). Lippincott Williams & Wilkins, Philadelphia, 2008.

children <1 year of age receiving SCh to prevent bradycardia. Fentanyl should be used with caution in infants and small children, as it may lead to respiratory depression or hypotension.

Status asthmaticus

Managing the sick asthma patient in the ED can present a tremendous challenge. Fatigue from prolonged respiratory effort in the face of severe small airway resistance commonly results in respiratory failure. Approximately 1–3% of asthma exacerbations require intubation. In general, these patients are extremely difficult to preoxygenate due to a reduced functional residual capacity, and may be very difficult to BMV as a result of severe airway obstruction.

The single most important intervention in a patient with status asthmaticus and respiratory failure is early control of the airway. RSI is the method of choice (performed by the most experienced laryngoscopist), along with preparation for rescue cricothyrotomy. When compared with RSI, BNTI is more time-consuming, results in greater O_2 desaturation, and has a higher rate of complication or failure.

Prior to intubation, allow patients to sit upright and preoxygenate them to the greatest degree possible. Pretreatment with lidocaine (1.5 mg/kg) suppresses coughing, improves ETT tolerance and reduces bronchospasm. Ketamine (1.5 mg/kg) is the induction agent of choice in status asthmaticus as it stimulates the release of catecholamines and produces bronchodilation.

Upon loss of consciousness, the patient should be laid supine and intubated. The largest possible ETT should be used to allow for aggressive pulmonary toilet. The intubated asthmatic patient should then be paralyzed and sedated to facilitate oxygenation and ventilation. Be aware that a patient's clinical condition may worsen after intubation if they prove difficult to ventilate, develop a tension pneumothorax or develop hypotension.

Increased intracranial pressure

The presence or suspicion of increased ICP directly impacts the approach to RSI, as the techniques and medications used may further increase the patient's ICP. There is a reflex sympathetic response to laryngoscopy that results in a systemic release of catecholamines and

Table 2.7 Pediatric airway equipment selection

Length (cm) and weight (kg) based pediatric equipment chart

	Pink	**Red**	**Purple**	**Yellow**	**White**	**Blue**	**Orange**	**Green**
Weight (kg)	6–7	8–9	10–11	12–14	15–18	19–23	23–31	31–41
Length (cm)	60.75–67.75	67.75–75.25	75.25–85	85–98.25	98.25–110.75	110.75–122.5	122.5–137.5	137.5–155
ETT size (mm)	3.5	3.5	4	4.5	5	5.5	6 cuff	6.5 cuff
Lip-tip length (cm)	10.5	10.5	12	13.5	15	16.5	18	19.5
Laryngoscope	1 Straight	1 Straight	1 Straight	2 Straight	2 Straight	2 Straight or curved	2 Straight or curved	3 Straight or curved
Suction catheter	8F	8F	8F	8–10F	10F	10F	10F	12F
Stylet	6F	6F	6F	6F	6F	14F	14F	14F
Oral airway	50 mm	50 mm	60 mm	60 mm	60 mm	70 mm	80 mm	80 mm
Nasopharyngeal airway	14F	14F	18F	20F	22F	24 F	26F	30F
Bag-valve device	Infant	Infant	Child	Child	Child	Child	Child/adult	Adult
Oxygen mask	Newborn	Newborn	Pediatric	Pediatric	Pediatric	Pediatric	Adult	Adult
Vascular access	22–24/23–25	22–24/23–25	20–22/23–25	18–22/21–23	18–22/21–23	18–20/21–23	18–20/21–22	16–20/18–21
Catheter/butterfly	Intraosseous	Intraosseous	Intraosseous	Intraosseous	Intraosseous	Intraosseous		
Nasogastric tube	5–8 F	5–8F	8–10F	10F	10–12F	12–14F	14–18F	18F
Urinary catheter	5–8 F	5–8F	8–10F	10F	10–12F	10–12F	12F	12F
Chest tube	10–12F	10–12F	16–20F	20–24F	20–24F	24–32F	24–32F	32–40F
Blood pressure cuff	Newborn/ infant	Newborn/ infant	Infant/child	Child	Child	Child	Child/adult	Adult
LMA†	1.5	1.5	2	2	2	2–2.5	2.5	3

Directions for use: 1. Measure patient length with centimeter tape, or with a Broselow tape. 2. Using measured length in centimeters or Broselow tape measurement, access appropriate equipment column. 3. For endotracheal tubes, oral and nasopharyngeal airways, and laryngeal mask airways (LMAs), always select one size smaller and one size larger than the recommended size, in addition to the recommended size.

†Based on manufacturer's weight-based guidelines.

Mask size:	1	1.5	2	2.5	3
Patient size (kg):	up to 5	5–10	10–20	20–30	Over 30

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subsequently increased ICP. This response can be blunted through the administration of fentanyl (3 mcg/kg), given over 30–60 seconds during the pretreatment phase of RSI. Laryngoscopy or any laryngeal stimulation (e.g., suctioning) may also increase ICP by a direct reflex mechanism unrelated to this catecholamine surge. The administration of lidocaine (1.5 mg/kg) during the pretreatment phase effectively blunts this response.

The ideal induction agent should reduce ICP, maintain cerebral perfusion and provide some cerebral protective effect. Although sodium thiopental effectively reduces ICP and confers a cerebroprotective effect, it is a potent venodilator and negative inotrope that can induce hypotension, a factor associated with significant mortality in head-injured patients. Etomidate (0.3 mg/kg) has emerged as the induction agent of choice in patients with increased ICP. It reduces ICP and confers cerebroprotection in a manner similar to thiopental but provides remarkable hemodynamic stability.

Suspected cervical spine injury

Inadvertent neck movement in a patient with an unstable cervical spine injury can lead to permanent neurologic disability or death. As a result, many trauma patients are transported to the ED in a stiff cervical collar and immobilized to a backboard. Although immobilization provides protection of the cervical spine, it places the patient at risk for aspiration and ventilatory compromise.

If the patient requires airway management, precious time should not be wasted obtaining a single lateral radiograph of the cervical spine to exclude cervical spine injury. This approach delays definitive airway management and provides a false sense of security, as a single view is inadequate to exclude injury to the cervical spine. Numerous studies have shown that RSI with in-line immobilization (Figure 2.20) is a safe, effective approach for managing these patients. Paralyzing the patient reduces the risk of patient movement during intubation and provides the best conditions for laryngoscopy. Having a dedicated individual maintain immobilization of the head and neck in the neutral position throughout the procedure prevents neck hyperextension during laryngoscopy.

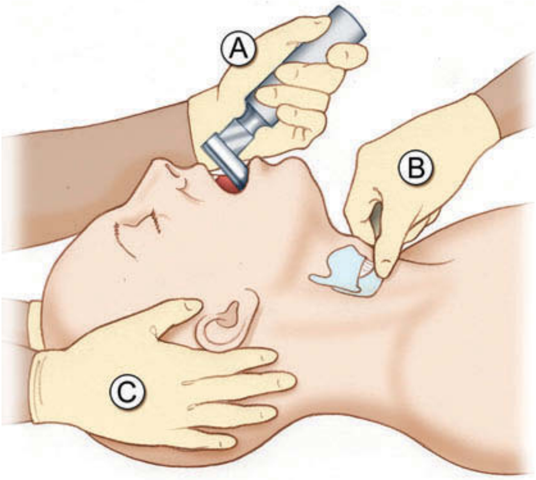

Figure 2.20
In-line immobilization of the cervical spine. © Chris Gralapp.

Pearls, pitfalls and myths

- Learn to recognize objective signs of impending airway compromise.
- Although bedside maneuvers or airway adjuncts can reestablish airway patency, they do not provide definitive airway protection.
- A definitive airway requires an ETT in the trachea, secured in place, with the cuff inflated and attached to an O_2-rich ventilation device.
- Competence with bag-mask ventilation is requisite for airway management.
- Understand the 9 Ps of rapid sequence intubation.
- Prior to initiating RSI, prepare for intubation using the mnemonic *SOAP ME*.
- Adequate preoxygenation may allow the laryngoscopist several intubation attempts prior to arterial O_2 desaturation.
- Select your pretreatment (LOA) medications to mitigate the adverse effects of SCh and the act of intubation.
- NBMAs do not provide analgesia, sedation, or amnesia, so always provide an induction agent prior to neuromuscular blockade.
- After any unsuccessful intubation attempt, change “something” prior to the next attempt.
- Proper ETT placement needs to be confirmed after every intubation; do not rely on only one approach as the sole means for confirming ETT placement.
- Following confirmation of ETT placement, secure the ETT, check vital signs, order a chest X-ray and provide long-term sedation and paralysis (if appropriate).
- Not every patient needs RSI. Learn the indications and techniques for awake oral intubation and BNTI.
- Every intubation should be assumed difficult, and a back-up plan should be formulated prior to proceeding. Success favors the prepared.
- Be familiar with the algorithms, devices and techniques used for the failed airway.
- Pediatric airway equipment selection and drug dosing is based on a child’s age, weight and length.
- Use RSI with in-line immobilization for airway management of any patient with potential cervical spine injury.

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3 Cardiopulmonary and cerebral resuscitation

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Scope of the problem

Emergency providers must be experts in the management of all acutely ill patients. Among the most challenging patient management scenarios are victims of cardiac arrest. Treating victims of cardiopulmonary arrest requires critical thinking and immediate action. Appropriate and timely interventions, both in the field and in the emergency department (ED), can determine the difference between meaningful recovery and death.

The greatest chance of survival after cardiac arrest occurs when there is (1) early recognition of warning signs, (2) timely activation of emergency medical services (EMS), (3) rapid initiation of basic cardiopulmonary resuscitation (CPR), (4) appropriate defibrillation, and (5) initiation of advanced cardiovascular life support (ACLS). Unfortunately, most resuscitative efforts in patients experiencing cardiopulmonary arrest are unsuccessful. Fewer than 6% of patients survive to hospital discharge, and approximately 450,000 die annually in the United States from sudden cardiac arrest.

Pathophysiology

During cardiac arrest, cessation of blood flow rapidly leads to cellular hypoxia and anaerobic metabolism. As toxic metabolites accumulate within the cell, a cascade of events potentiates tissue injury, including calcium release, generation of free radicals and activation of catabolic enzymes. Damage to vital organ systems occurs rapidly. The brain suffers irreversible injury within 5 minutes of arrest. Full neurologic recovery rarely occurs in patients experiencing greater than 10 minutes of untreated cardiac arrest.

Survival after cardiac arrest from ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) decreases approximately 7–10% for each minute a patient is not defibrillated. Immediate institution of basic CPR can reduce this mortality rate by half. Properly performed CPR generates up to 30% of baseline cardiac output. The blood circulated by properly performed chest compressions can deliver just enough oxygen and metabolic substrate to temporarily sustain the brain, heart and other vital organs, and prolongs the time in which the myocardium is responsive to defibrillation.

Principles of resuscitation

Preparation

Advanced preparation is a vital component of a well-run resuscitation. The first step in this process is familiarity with available resources, personnel and equipment that should be "at the ready" for patient arrival. Most EDs have specially designed "resuscitation" or "code" rooms that are larger than other patient care spaces. Resuscitation rooms should have the capacity to accommodate ancillary staff, nurses and other health care specialists assisting with the resuscitation. Essential equipment such as monitors, personal protective gear and procedure sets must be easily accessible. The resuscitation team, especially the team leader, should be familiar with the layout of the room and the location of emergency supplies.

ED resuscitation optimally starts prior to patient arrival. The resuscitation commences upon notification that a critically ill patient is en route. The call from the EMS team to the ED (the "ringdown") alerts providers to important patient and event details minutes prior to ambulance arrival. When possible, emergency providers should listen to the ringdown so they can advise EMS personnel regarding time-sensitive interventions. The presence or absence of vital signs and a description of the patient's general appearance and surroundings help the emergency physician determine if additional prehospital interventions are indicated, or if a "scoop-and-run" approach is advantageous. Listening to the ringdown also allows the EM provider to begin detailed planning prior to patient arrival. A general preparation checklist is shown in Table 3.1. Some patients may require additional items not listed below, and ringdown information may not always be accurate.

Table 3.1 ACLS resuscitation room essential equipment list

Airway equipment
Bag-mask device
Airway adjuncts including nasopharyngeal and oropharyngeal airways
Suction
Oxygen
Intubation equipment (several sizes of endotracheal tubes, laryngoscope handle with several sizes of Macintosh and Miller blades, stylet, 10-mL syringe, ETCO$_2$ detection device)
Monitoring/diagnostics
Cardiac, pulse oximetry, and quantitative capnography monitors
Defibrillator with transcutaneous pads
12-lead ECG machine
Ultrasound
Intravenous access
IV kit with various gauge IV needles
Central IV access kit
Arterial line kit
Intraosseous needles and drill (if available)
IV pressure bags

Assign team member roles

Assigning expected roles to each team member prior to patient arrival is a key element of effective resuscitations (Table 3.2). For example, designating a team member responsible for high-quality CPR during resuscitation allows that person to properly position him- or herself prior to patient arrival; assigning a care provider responsible for IV access prioritizes acquisition and priming of the necessary equipment. Assigning roles also serves as a crowd control measure, as the spectacle of resuscitation attracts many staff members to the room. Although additional staff may want to help, they may be detrimental by limiting space for others.

Whenever possible, team members should be addressed by name. If the physician leader does not know a team member's name, direct communication using identifying information is preferred. For example, "Jason has been performing chest compressions for several minutes. Can you, in the blue scrub top, please take over chest compressions after this cycle?"

Table 3.2 Roles of ACLS code team members

Resuscitation team leader
Basic airway management
Advanced airway management
CPR
IV access
Recorder
Medications
EMS handoff

The arrival of the patient with EMS providers can be a time of chaos. There is a tendency for hospital providers to want to rapidly initiate measures to assist the patient. Although certain actions (e.g., safe gurney transfer, monitor placement, continuation of chest compressions, and airway and spinal cord protection) take priority, it is helpful for the physician leader to listen to the paramedics' report as it often provides vital information. Field providers may have spoken to family members or witnesses who can recount historical information relevant to the patient's ongoing care. EMS providers may also have documents such as physician orders for life-sustaining treatment (POLST), other advanced resuscitation directives, or contact information for a health care proxy. Access to this information may guide the resuscitation. Finally, EMS providers can briefly and accurately describe field interventions. This entire process allows the emergency physician to transition into the role of resuscitation team leader.

Team resuscitation approach

Physicians have traditionally taken an authoritarian role in critical patient resuscitation. This outdated approach may lead to breakdowns in trust and communication within the care team, and ultimately may be detrimental to the patient's care. Although the emergency physician is typically the team leader in a critical care situation, a successful team leader recognizes the skill sets of each team member, sets the tone by maintaining a calm demeanor, and focuses on initiation of lifesaving interventions. He may accomplish these goals by providing encouragement to team members, giving direction, and keeping the room calm. The care team looks to the team leader to set the tone in critical situations. A physician who easily becomes unnerved, loses his temper, or "freezes" in a code will undermine the team's trust, leading to disorganization. The physician can improve his ability to function in this manner through frequent, periodic review of ACLS protocols; politely asking for quiet in the room at the beginning and as needed during the resuscitation (speaking rather than yelling); and sharing thoughts about the resuscitation's progress with the team.

Direct communication using simple terms is pivotal to effective leadership in the code situation. The stress of a critical situation can make it difficult to think and act, and gentle direction will allow code team members to accomplish their patient care goals simultaneously. Furthermore, real-time feedback is important not just for the immediate care of the patient, but for the education and growth of each team member. Reinforcing a medical student to "push hard, push fast" (to the beat of *Stayin' Alive*) while he is performing chest compressions is more effective than doing so later during a debriefing. It is also important that the lead physician be genuinely open to receiving feedback. Often, other team members may be in a position to observe or identify interventions that the physician might have overlooked. Prior to cessation of resuscitative efforts, input from the entire resuscitation team regarding additional or alternative approaches should be sought.

Initial evaluation and management

A systematic approach to resuscitation allows for thorough yet rapid assessment and care of the patient. The most common approach follows the concept of the *primary* and *secondary* surveys with adherence to basic life support (BLS) and ACLS protocols. In 2010, American Heart Association (AHA) guidelines adapted the mnemonic C-A-B (compressions, airway, breathing) to facilitate retention. Notably, the adaptation of the C-A-B sequence is a change from previous BLS guidelines recommending an A-B-C approach for adult and pediatric patients. This change is predicated on the observation that the initial round of 30 chest compressions will delay a single provider's assessment of the airway by only 18 seconds. In scenarios where limited providers are present, care should progress sequentially – each assessment should be followed by an appropriate intervention (i.e., correcting any immediate life-threats) prior to proceeding to the next step. When more than one provider is present, multiple interventions should be performed simultaneously while maintaining a systematic approach. The primary survey focuses on immediate life-threats. The secondary survey follows, seeking to identify and correct emergent but less immediate conditions.

Primary survey

In the primary survey, the focus is on BLS, including CPR and defibrillation:

Immediate actions

Circulation: Give chest compressions
Airway: Open the airway
Breathing: Provide positive-pressure ventilation

While C-A-B is in progress

Defibrillation: Identify and shock VF and pulseless VT

Note: Per 2010 AHA Guidelines, it is reasonable for health care providers to tailor this approach to address the most likely cause of arrest. For example, in newborns the ABC approach is still preferred.

Circulation

Chest compressions are the rhythmic application of pressure over the lower sternum, intended to create blood flow by increasing intra-thoracic pressure and directly compressing the heart. To correctly perform chest compressions in an adult patient, the heel of one hand is placed in the midline on the lower part of the sternum. The other hand is placed on top of the first hand and the fingers interlocked and kept off of the chest. The provider's shoulders are positioned directly over the hands, and the elbows locked. The sternum is depressed at least 2 inches and released, allowing time for full chest recoil (Figure 3.1). The tag line "push hard and push fast" has been coined to help correct the most common errors made by CPR providers: delivering compressions too gently and too slowly. Properly performed CPR can produce a systolic blood pressure of 60 mmHg and deliver a small but potentially life-sustaining amount of oxygen and energy substrate to cerebral and myocardial tissues.

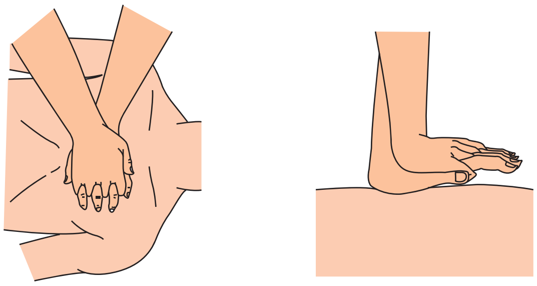


Figure 3.1
Proper hand position and compression technique for adult CPR.

Circulation assessment begins with checking for central pulses at either the carotid or femoral arteries. A provider should spend no more than 10 seconds determining the presence or absence of pulses to avoid delays in initiating CPR. In the absence of a definite pulse, CPR is initiated. Notably, 2010 AHA guidelines de-emphasize the importance of pulse checks even for health care providers because assessment of central circulation can be difficult for anyone in the critical care setting. Furthermore, the delivery of CPR in a non-cardiac arrest patient rarely leads to serious injury.

In the adult patient, compressions are initiated at a rate of at least 100 compressions per minute, with a depth of at least 2 inches. Pediatric and neonatal advanced support guidelines recommend different compression-to-ventilation ratios than adults, 15:2 and 3:1 respectively. Recommended compression depth is at least one-third of the anterior-posterior diameter of the chest, which is approximately 2 inches in children and 1.5 inches in infants. Once a definitive airway is established, compressions should be continuous without pausing for ventilations.

As delivery of effective chest compressions is paramount to successful resuscitation, the resuscitation team leader should pay careful attention to provider fatigue. The ability of a provider to deliver effective compressions diminishes after several CPR cycles. The provider performing chest compressions should alternate approximately every 2 minutes, ideally switching out quickly (i.e., <5 seconds) to minimize the interruption of CPR. The resuscitation team leader generally should not assume the role of giving compressions because the physical strain makes decision-making and oversight of the code difficult.

Although the AHA continues to recommend compressions coordinated with ventilations as the ideal BLS resuscitation strategy, increasing recognition has been given to cardio-cerebral resuscitation (CCR) in recent years. CCR refers to compression-only CPR for the lay rescuer or single care provider. Surveys of both lay rescuers and health care providers show that many providers are reluctant to give mouth-to-mouth rescue breaths to unknown cardiac arrest victims, and thus are reluctant to intervene at all. Numerous studies have demonstrated that outcomes are improved in patients receiving chest compressions without ventilations compared with those receiving no CPR. It is hypothesized that some passive ventilation occurs during chest recoil and occasional agonal gasps, and that this small minute ventilation may be sufficient given the limited circulation of blood in cardiac arrest patients. Therefore, current AHA recommendations are for laypersons to perform compression-only CPR if they are unable or unwilling to perform rescue breathing.

Airway

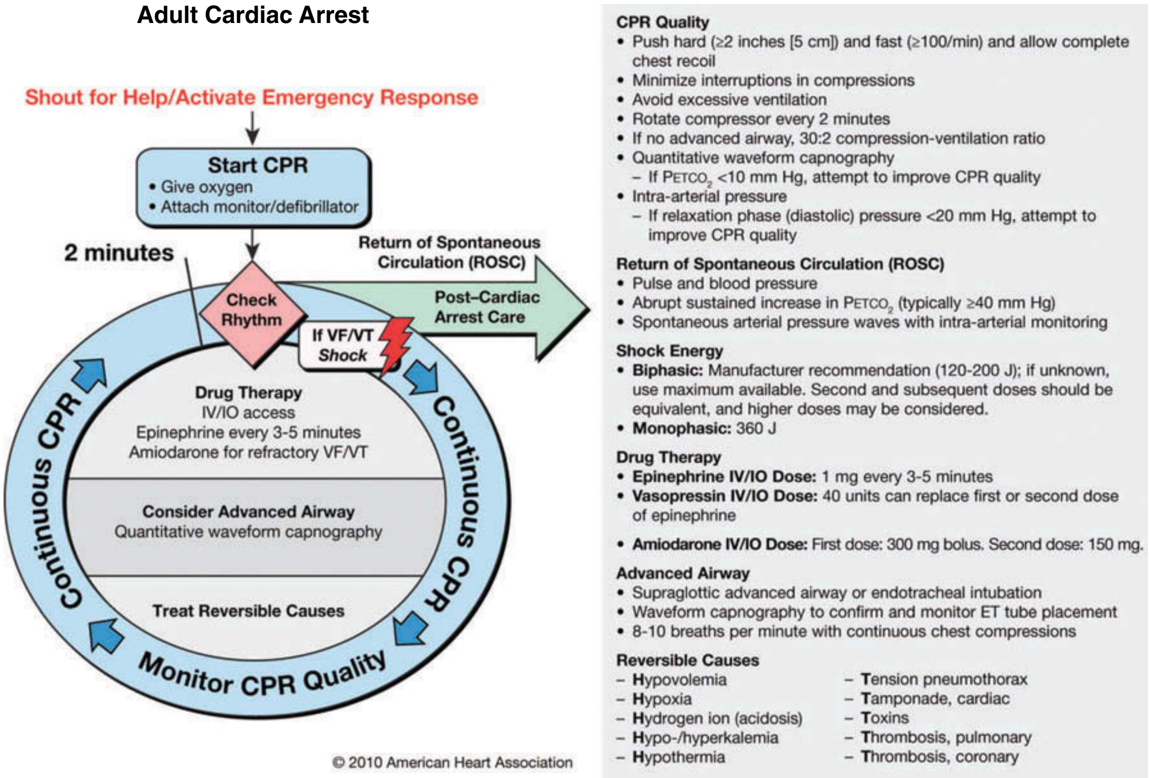
Initial survey of the airway focuses on ensuring airway patency. Patency can be rapidly evaluated by asking the patient to say his or her name. If the patient is able to verbalize, immediate airway management is unlikely necessary. In patients who do not respond to questioning, the gag reflex may be checked using a tongue blade or more preferably a suction catheter. The lack of a strong gag reflex implies inadequate airway protective mechanisms. A patient in full respiratory arrest or with inadequate airway protection requires immediate intervention. In the atraumatic, unconscious or obtunded patient, the airway is most often compromised by the tongue, which tends to fall posteriorly against the soft palate. Adjustment of the mandible using the head tilt-chin lift or jaw thrust maneuver (if cervical spine injury is suspected) is often sufficient to open the airway. Simple adjuncts such as the nasopharyngeal airway (in the semi-responsive patient who retains a gag reflex) or the oropharyngeal airway (in the unresponsive patient who has no airway protective reflexes) can greatly improve airway patency. Chapter 2 describes comprehensive airway and breathing assessment and management.

Breathing

Most cardiopulmonary arrest victims will not breathe spontaneously. However, in the peri-arrest period, agonal respirations and occasional gasps may be observed. Adequacy of respirations is determined by observation of the chest for symmetric rise and fall and by auscultation of the chest for bilateral air movement. In patients with absent or inadequate respirations, delivery of two positive pressure ventilations by bag-mask device or mouth-to-mouth with a protective barrier is indicated after the first round of 30 chest compressions has been completed. Successful bag-mask ventilation confirms airway patency and may stimulate spontaneous breathing. If breathing remains inadequate, additional supplemental breaths are administered in an age-appropriate rate with chest compressions until a definitive airway is established. Each breath should be delivered over 1 second with adequate volume to produce visible chest rise. Hyperventilation should be avoided, as breaths that are too large, too forceful, or too frequent can be counterproductive. Providers must remember that during CPR, pulmonary blood flow is significantly reduced, such that matching ventilation to perfusion may be accomplished with lower tidal volumes and respiratory rates. Furthermore, forceful, high-volume breaths may lead to increased intra-thoracic pressure, decreased cardiac venous return and gastric insufflation.

Defibrillation

Defibrillation is the delivery of a current of electricity to simultaneously depolarize the myocardium and terminate a non-perfusing ventricular rhythm. The goal of defibrillation is to allow the sinus node or other myocardial focus to resume pacing the heart, resulting in an organized perfusing rhythm. Defibrillation is the cornerstone of treatment in victims of cardiac arrest experiencing VF and pulseless VT (Figure 3.2). Patients in cardiac arrest with pulseless electrical activity (PEA) or asystole do not respond to defibrillation, as an organized rhythm already exists in PEA and the myocardial cells are unresponsive in asystole. In non-arrest patients with unstable tachycardias, synchronized cardioversion (rather than defibrillation) is indicated (Figure 4.7).


Adult Cardiac Arrest
Shout for Help/Activate Emergency Response
Start CPR
• Give oxygen
• Attach monitor/defibrillator
2 minutes
Return of Spontaneous Circulation (ROSC)
Check Rhythm
If VF/VT Shock
Post–Cardiac Arrest Care
Drug Therapy
IV/IO access
Epinephrine every 3-5 minutes
Amiodarone for refractory VF/VT
Consider Advanced Airway
Quantitative waveform capnography
Treat Reversible Causes
Continuous CPR
Continuous CPR
Monitor CPR Quality
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CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- **Biphasic:** Manufacturer recommendation (120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy
- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

Figure 3.2
2010 AHA Guidelines for CPR and ECC, Part 8: Adult Advanced Cardiovascular Life Support, Page S737, Figure 2. ACLS Cardiac Arrest Circular algorithm. Reprinted with permission, 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 8: Adult Advanced Cardiovascular Life Support. *Circulation* 2010;122[suppl 3]:S729–67 © 2010 American Heart Association, Inc.

Early recognition and defibrillation of VF or pulseless VT coordinated with appropriate, high-quality CPR provide the greatest opportunity for successful resuscitation of a cardiac arrest patient. When defibrillation can be successfully performed within the first minute or two, as many as 90% of patients return to their pre-arrest neurologic status. The longer the patient remains in cardiac arrest, the more likely that defibrillation and resuscitation will be unsuccessful. Survival rates are <10% when defibrillation is delayed ≥10 minutes after a patient's collapse. CPR can improve the responsiveness of VF and delay its degeneration to asystole, thus lengthening the window of time during which successful defibrillation may occur.

Because survival from VF or pulseless VT is exquisitely time-sensitive, defibrillation in witnessed VF or pulseless VT should preclude any other intervention. Defibrillation should be attempted with a single shock as soon as the diagnosis is made (Figure 3.2). Using gel or defibrillation pads, one paddle should be placed to the right of the sternum below the right clavicle and the other in the left mid-axillary line at the level of the nipple (Figure 3.3). Firm pressure (approximately 25 lb) should be applied to each paddle. Alternatively, "hands off" defibrillator pads can be used that are placed on the chest and the back, sandwiching the heart (Figure 3.4). CPR should be performed until the defibrillator is charged and ready to deliver a shock. Immediately following all attempts at defibrillation, an additional five cycles of CPR should be provided *before* performing a pulse or rhythm check.

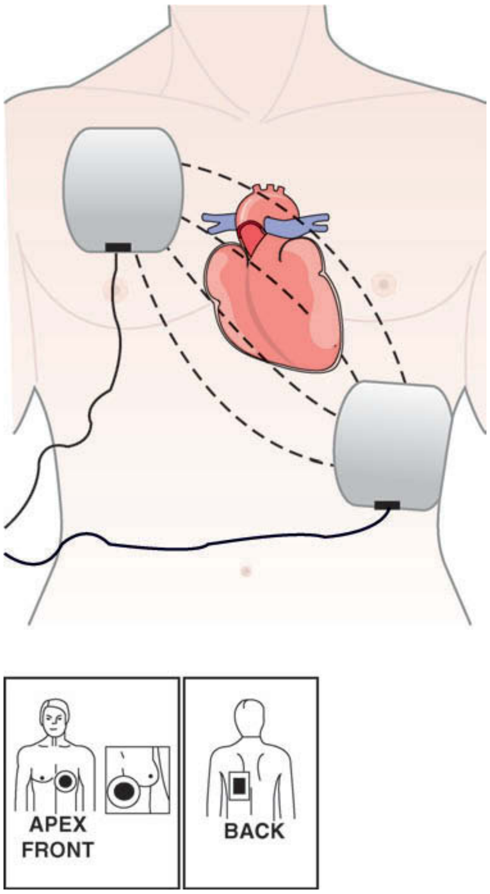

Figure 3.4
Anterior placement for defibrillation pads. Alternatively, defibrillation pads may be placed directly anterior and posterior to the heart, when accessible.

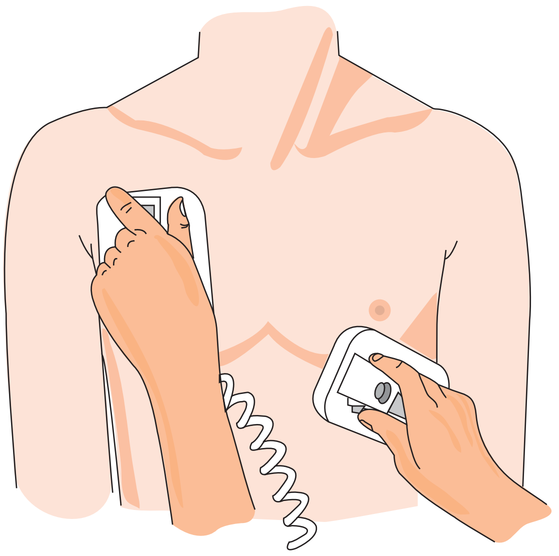

Figure 3.3
Proper positioning of defibrillation paddles for adult patients.

Defibrillators are available in two types of waveforms: monophasic and biphasic. Although no survivability superiority has been demonstrated between waveforms, biphasic defibrillators require less energy and are recommended. Consequently, most recently manufactured defibrillators are biphasic. However, many monophasic defibrillators remain in use. As with other critical resuscitation equipment, it is imperative to be familiar with the specific defibrillator available to avoid unnecessary delays during resuscitations. The energy level for each shock delivered by a biphasic defibrillator in treating VF/pulseless VT should be as specified by the product manufacturer. In the absence of knowledge of manufacturer recommendations, the maximal energy level should be used.

The term automatic external defibrillator (AED) refers to a sophisticated computerized device that incorporates rhythm analysis and a shock advisory system. AEDs are designed to recognize VF or VT and advise the user whether or not to deliver an electric shock. The AED will deliver a shock and hopefully convert a non-perfusing rhythm to a perfusing one. Placing AEDs in public access areas like airports, sports stadiums and restaurants allows quicker access to life-saving defibrillation. When police officers in Rochester, Minnesota were equipped with an AED, survival from out-of-hospital VF averaged 50% with a median time from collapse to defibrillation of 5 minutes. Similar statistics have been reported in public access trials in other states. These survival rates are twice those previously reported for many emergency medical systems.

Secondary survey

The ACLS secondary survey focuses on advanced interventions and a search for the etiology of cardiopulmonary arrest. It can be remembered using the C-A-B mnemonic.

Circulation: Continued delivery and monitoring of high quality CPR
Airway: Definitive airway management as appropriate
Breathing: Confirmation of adequate ventilation
Defibrillation: Continued interval rhythm analysis and treatment
Other actions, as appropriate: IV access, ACLS medications, IV fluids if suspicion of intravascular volume depletion

Airway

A definitive airway should be established, preferably by endotracheal intubation. A correctly placed endotracheal tube is the most effective method of ensuring adequate ventilation, oxygenation and airway protection. However, these benefits must be weighed against the risks, the experience of the provider considered, and the amount of interruption to chest compressions. As limited evidence exists to guide the appropriate timing of definitive airway management within the code situation, this decision is at the resuscitation leader's discretion.

Breathing

Following placement of a definitive airway, continuous high-flow oxygen should be initiated and ventilation evaluated by observing bilateral chest wall rise, auscultating equal and bilateral breath sounds, and identifying fog in the endotracheal tube on exhalation. Quantitative end-tidal CO_2 monitoring devices are recommended for correct endotracheal tube placement confirmation. This method is more accurate and allows for continual monitoring throughout the resuscitation. A chest X-ray can help determine the location of the tip of the endotracheal tube in relation to the carina. If the patient was intubated in the prehospital setting, visualization of tube location by direct laryngoscopy is advised. Movement of the patient during transfer can dislodge a properly placed endotracheal tube. Once the position of the definitive airway device has been confirmed, breaths should be delivered at a rate of one breath every 6–8 seconds (8–10 breaths per minute) without pauses in chest compression.

Circulation

The "C" component of the secondary survey focuses on continuing high-quality CPR along with rhythm recognition and management as dictated by ACLS guidelines. The adequacy of CPR should be assessed frequently; further monitoring through quantitative waveform capnography and intra-arterial pressure monitoring should be considered. Four cardiac rhythms may produce a pulseless arrest: VF, VT, PEA and asystole. ACLS divides management into those responsive to defibrillation (pulseless VT and VF [Figure 3.5]) and those unresponsive to defibrillation (PEA and asystole). A patient's cardiac rhythm may change multiple times during resuscitation; providers must change treatment plans accordingly. Intravenous (IV) access should be obtained by central venous catheter or two large-bore peripheral lines, and isotonic fluid administered rapidly by bolus infusion. In the event IV access cannot be obtained within 1–2 minutes, intraosseous (IO) access is recommended.

Ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT)

VF and pulseless VT are the most common underlying rhythms of cardiac arrest (Figure 3.2). Initial defibrillation of these non-perfusing rhythms is addressed in the primary survey. If VF and pulseless VT are refractory to an initial attempt at defibrillation, IV/IO epinephrine should be delivered for cardiac and blood pressure support. This should be repeated every 3–5 minutes until the patient has a perfusing cardiac rhythm. Vasopressin may be used as an alternative to epinephrine. In patients with initial refractory VF or pulseless VT, the administration of amiodarone, an antidysrhythmic medication, should be considered. Lidocaine may be substituted if amiodarone is unavailable, though current guidelines recommend the use of amiodarone. If the rhythm is determined to be *torsades de pointes*, a specialized form of polymorphic ventricular tachycardia, magnesium is recommended, as it may terminate this dysrhythmia. Following each five cycles of CPR (approximately 2 minutes), the rhythm and perfusion should be reassessed and defibrillation performed if indicated.

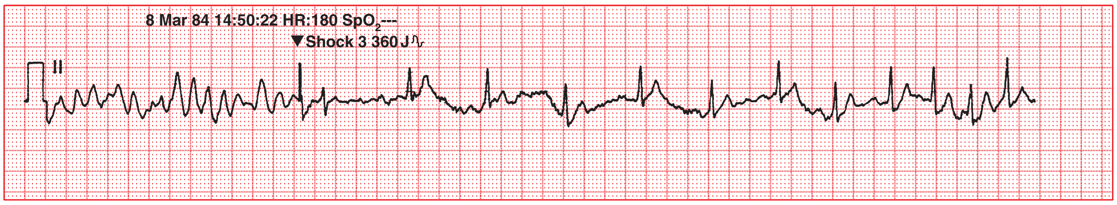

Figure 3.5
Rhythm strip of a patient with ventricular fibrillation (VF). Following the third defibrillation attempt at 360 J, the patient returned to sinus rhythm. HR: heart rate; J: joules; SpO_2: saturated pressure of oxygen. Courtesy: S.V. Mahadevan, MD.

Pulseless electrical activity (PEA) and asystole

Pulseless patients in any cardiac rhythm other than VF or VT are defined as having PEA. Asystole, a lack of myocardial electrical activity, is generally considered an end-stage rhythm and should be confirmed on cardiac monitor in at least two leads. PEA and asystole are discussed together as they are both unresponsive to defibrillation and follow the same evaluation and treatment algorithm. Other cardiac rhythms such as tachycardias and bradycardias are discussed in Chapter 4.

The key to successful treatment of PEA and asystole is delivering high-quality CPR with minimal interruptions, providing vasoactive medications, and, most importantly, searching for the underlying etiology of the patient's cardiac arrest (Figure 3.2). During the resuscitation process, a vigilant, systematic search by the treatment team for reversible causes of cardiac arrest should be undertaken. The most frequent causes are summarized by the "5 Hs and 5 Ts" mnemonic: hypovolemia, hypoxia, hydrogen ions (acidosis), hypo-/hyperkalemia, hypothermia, toxins, tamponade (cardiac), tension pneumothorax, thrombosis (coronary) and thromboembolism (pulmonary). The history, physical examination, diagnostic studies and management decisions should focus on identifying and treating these reversible causes of cardiopulmonary arrest.

History

Obtaining historical information about a patient in cardiac arrest is often difficult; thus, the resourceful physician must utilize alternative methods of data collection. Information must be gathered from prehospital providers, family, medical records, medication lists and primary care physicians. Clues on the patient's body (wallet, ID bracelet, traumatic injuries, needle marks, scars, dialysis shunts) must be identified. An attempt to learn the following information should be made:

What were the events surrounding the arrest?

Determine whether the patient had a witnessed arrest or was found unconscious. What was the approximate duration of time prior to initiation of CPR (downtime)? Ask whether the patient was having any concerning symptoms prior to the arrest such as chest pain, palpitations, or shortness of breath.

What has been the extent of the resuscitation thus far?

Determine the patient's initial cardiac rhythm and any subsequent changes in rhythm throughout the resuscitation. Ask which interventions have been made, such as defibrillation, airway intervention and medications, and the patient's response to them.

What is the patient's medical history?

Concentrate on the patient's cardiac history and risk factors for coronary artery disease. Heart disease is the most common cause of dysrhythmias and sudden cardiac death. Risk factors for pulmonary embolism, toxin exposure and drug overdose should also be obtained. Familiarity with the patient's medical problems or medications can suggest other possible causes of the cardiac arrest.

Historical information needs to be acquired in parallel with the resuscitation. An alternative member of the health care team may be assigned to search for clues if the physician is unable to leave the bedside. Much of this information may be obtained from family members. It is important to ask questions in a concise but sensitive manner. Communicate the critical nature of the situation while providing reassurance that care is being provided and the patient is not suffering. If appropriate, it is important to reassure the family that they did not cause or contribute to the situation.

Physical examination

Following the secondary survey, the physical examination should focus on vital organ systems and additional clues suggesting a reversible cause of cardiac arrest. The physician should begin by reassessing the baseline rhythm and vital signs, including a temperature. A quick head-to-toe survey should concentrate on the findings listed in Table 3.3. Subsequent examinations focus on assessing for any response and the potential complications of interventions.

Diagnostic studies

Diagnostic testing should focus on rapid evaluation of the 5 Hs and 5 Ts. Continuous cardiac monitoring and a 12-lead electrocardiogram (ECG) are important to evaluate for ST-segment elevation consistent with an acute myocardial infarction, T-wave changes concerning for hyperkalemia, or other ECG changes compatible with various toxins.

Bedside laboratory testing is readily available for hypoglycemia and, increasingly, for potassium abnormalities (hypo-/hyperkalemia) and acidosis. An acidosis is common in patients suffering cardiac arrest and less frequently the primary cause. Determining and correcting the underlying etiology of the acidemia is critical. In a patient with spontaneous circulation on mechanical ventilation, the arterial blood gas (ABG) can additionally guide ventilator settings and ongoing resuscitation measures. Other laboratory analyses to consider in the appropriate clinical situation include a hemoglobin level, cardiac markers, serum and urine toxicologic screens, and other specific toxin levels (such as aspirin). These test results are unlikely to return during the initial resuscitation, so empiric therapy should be initiated if clinical suspicion is high.

A chest radiograph is an important diagnostic study that may help establish a definitive diagnosis in a patient with cardiopulmonary arrest. It can also confirm correct endotracheal tube, central access catheters and nasogastric (NG) tube placement.

Table 3.3 Physical examination findings indicating potential cause of cardiac arrest and complications of therapy

Physical examination	Abnormalities	Potential causes
General	Pallor Cold	Hemorrhage Hypothermia
Airway	Secretions, vomitus, or blood	Aspiration Airway obstruction
	Resistance to positive-pressure ventilation	Tension pneumothorax Airway obstruction Bronchospasm
Neck	Jugular venous distention	Tension pneumothorax Cardiac tamponade Pulmonary embolus
	Tracheal deviation	Tension pneumothorax
Chest	Median sternotomy scar	Underlying cardiac disease
Lungs	Unilateral breath sounds	Tension pneumothorax Right mainstem intubation Aspiration
	Distant or no breath sounds, or no chest expansion	Esophageal intubation Airway obstruction Severe bronchospasm
	Wheezing	Aspiration Bronchospasm Pulmonary edema
	Rales	Aspiration Pulmonary edema Pneumonia
Heart	Audible heart tones	Hypovolemia Cardiac tamponade Tension pneumothorax Pulmonary embolus
Abdomen	Distended and dull	Ruptured abdominal aortic aneurysm Ruptured ectopic pregnancy
	Distended, tympanitic	Esophageal intubation Gastric insufflation
Rectal	Blood, melena	Gastrointestinal hemorrhage
Extremities	Asymmetric pulses	Aortic dissection
	Arteriovenous shunt or fistula	Hyperkalemia
Skin	Needle tracts or abscesses	Intravenous drug abuse
	Burns	Smoke inhalation Electrocution

Adapted from Marx J (ed). *Rosen's Emergency Medicine Concepts and Clinical Practice.* Mosby, St. Louis, MO, 2010.

The use of focused bedside ultrasonography in patients with cardiac arrest is common. Ultrasound (US) can identify the presence or absence of cardiac activity in the pulseless patient. This information can be used to distinguish asystole from VF, and confirm PEA. Furthermore, US can assist with the rapid evaluation and determination of the cause of cardiac arrest. Identification of a pericardial effusion (cardiac tamponade), pneumothorax, focal cardiac wall motion abnormality (myocardial infarction), dilated right ventricle (pulmonary embolus), flattened inferior vena cava (hypovolemia), and abdominal aortic aneurysm (hypovolemia) provides critical information to the care team. Finally, US can assist with the decision to cease resuscitation efforts. A "silent" heart on bedside US is an ominous finding, making recovery highly improbable.

General treatment principles

The cornerstone of treatment is quality BLS and ACLS. Other therapies must focus on the rapid correction of potential underlying causes of cardiac arrest. Hypovolemia and hypoxia should be empirically addressed by the administration of IV fluid and high-flow oxygen. Severely hypothermic patients should be aggressively warmed prior to termination of resuscitative efforts. If rapid testing of potassium is unavailable and there is suspicion for hyperkalemia, empiric therapy with calcium, bicarbonate, glucose, insulin and beta agonists is appropriate. The proper antidote should be administered prior to confirmation if the clinical scenario suggests toxin exposure.

Empiric thrombolytic use is supported by existing guidelines if clinical suspicion is high for massive pulmonary embolus. In rare circumstances, emergency percutaneous coronary intervention (PCI) may be indicated. Otherwise, appropriate supportive measures and interventions should be employed as indicated based on the likely etiology of the cardiac arrest.

Post-resuscitation care

Following return of spontaneous circulation (ROSC), the resuscitation is far from over. The post-arrest patient remains critically ill and continues to require aggressive evaluation and management (Figure 3.6). The goals during the post-cardiac arrest period are:

1. Optimize hemodynamic status and perfusion to vital organ systems.
2. Institute measures to prevent recurrence.
3. Identify and definitively treat the underlying cause of arrest.
4. Initiate therapeutic hypothermia.
5. Transfer the patient to an appropriate critical care setting.

Initially, oxygenation and ventilation of the patient should be optimized. *Airway* management includes reconfirming tracheal intubation, obtaining a chest radiograph if not previously completed, and placing a nasogastric tube to decompress the stomach. *Breathing* management involves placing the patient on a ventilator and refining their respiratory parameters. Data from the cardiopulmonary examination, ABG and ETCO$_2$ measurements can guide adjustments to ventilator settings. *Circulation* management should entail reassessment of hemodynamic status and monitoring. This includes measures to prevent recurrence of hypotension, including IV fluid boluses and vasopressor therapy as needed. A Foley catheter should be placed and urine output recorded. Invasive monitoring by arterial lines (BP), central venous catheters (central venous pressure [CVP]), central venous oxygen saturation (ScvO2), and Swan-Ganz catheterization (CVP, mixed venous oxygen saturation, pulmonary artery pressure, pulmonary artery wedge pressure) should be considered, although their true utility during ED management is controversial. Further cardiovascular care includes a repeat ECG searching for underlying abnormalities that place the patient at risk for ventricular dysrhythmias. Finally, it is reasonable to maintain infusions of any antidysrhythmic previously given and thought to be of benefit.

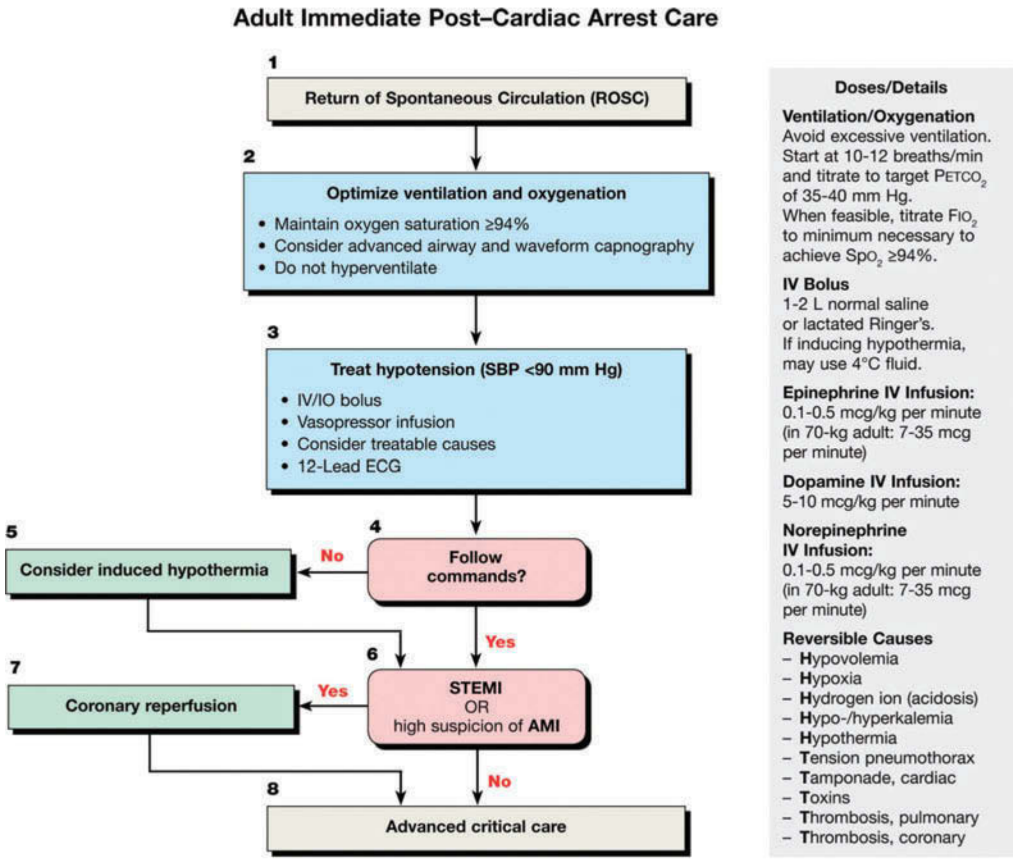
Adult Immediate Post–Cardiac Arrest Care
1
Return of Spontaneous Circulation (ROSC)
2
Optimize ventilation and oxygenation
• Maintain oxygen saturation ≥94%
• Consider advanced airway and waveform capnography
• Do not hyperventilate
3
Treat hypotension (SBP <90 mm Hg)
• IV/IO bolus
• Vasopressor infusion
• Consider treatable causes
• 12-Lead ECG
4
Follow commands?
No
5
Consider induced hypothermia
Yes
6
STEMI OR high suspicion of AMI
Yes
7
Coronary reperfusion
No
8
Advanced critical care
Doses/Details
Ventilation/Oxygenation
Avoid excessive ventilation. Start at 10-12 breaths/min and titrate to target PETCO$_2$ of 35-40 mm Hg.
When feasible, titrate FIO$_2$ to minimum necessary to achieve SpO$_2$ ≥94%.
IV Bolus
1-2 L normal saline or lactated Ringer's.
If inducing hypothermia, may use 4°C fluid.
Epinephrine IV Infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)
Dopamine IV Infusion:
5-10 mcg/kg per minute
Norepinephrine IV Infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)
Reversible Causes
– Hypovolemia
– Hypoxia
– Hydrogen ion (acidosis)
– Hypo-/hyperkalemia
– Hypothermia
– Tension pneumothorax
– Tamponade, cardiac
– Toxins
– Thrombosis, pulmonary
– Thrombosis, coronary

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Figure 3.6
2010 AHA Guidelines for CPR and ECC, Part 9: Post-Cardiac Arrest Care, Page S769, Figure. Post-cardiac arrest care algorithm. Reprinted with permission, 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 9: Post-Cardiac Arrest Care. *Circulation* 2010;122[suppl 3]:S768–86 © 2010 American Heart Association, Inc.

Subsequent to hemodynamic stabilization, a thorough search for additional history, meticulous physical examination, and careful review of all labs and studies should be performed. Of particular note, glucose levels require close monitoring, as untreated abnormalities may worsen neurologic outcomes. Other organ systems should be evaluated vigilantly, as the post-cardiac arrest patient is at high risk for multiorgan dysfunction syndrome.

In the post-cardiac arrest patient who remains comatose (unable to rouse and follow commands), therapeutic hypothermia should be initiated. Cardiac arrest victims may benefit from induced hypothermia during the post-resuscitation period. Improved outcomes and metabolic end points have been observed in a number of early studies, including both in- and out-of-hospital cardiac arrest victims. The exact mechanism of action of induced hypothermia and determination of those patients most likely to benefit is unclear at this time. However, based on existing data, many hospitals have instituted therapeutic hypothermia protocols in patients who remain comatose after initial resuscitation using specialized cooling blankets, endovascular cooling catheters, cooled saline intravenous infusions and ice packs. Most protocols target cooling to a core temperature of 32–34°C, but the optimal

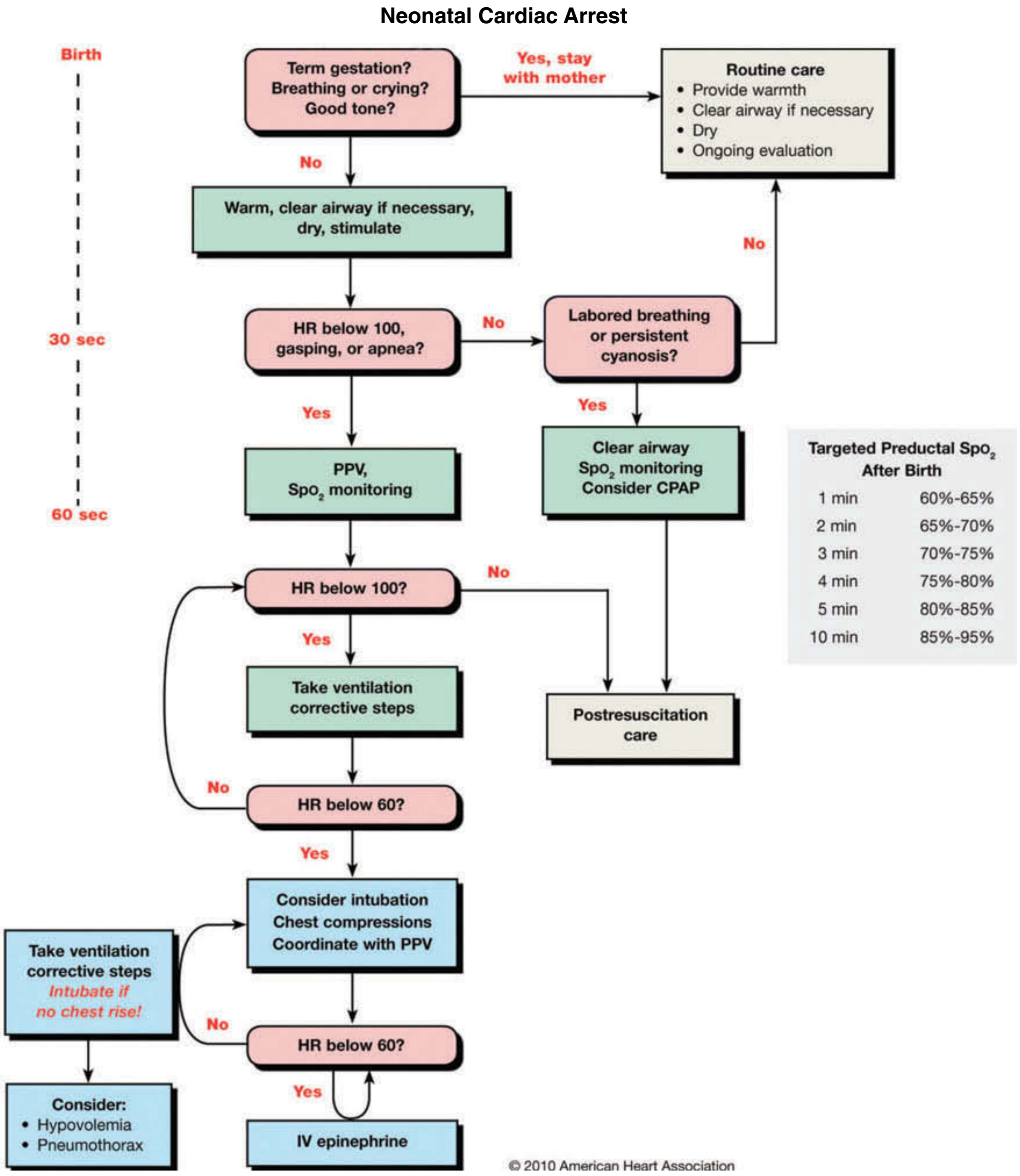
Neonatal Cardiac Arrest
Birth
30 sec
60 sec
Term gestation? Breathing or crying? Good tone?
Yes, stay with mother
Routine care
• Provide warmth
• Clear airway if necessary
• Dry
• Ongoing evaluation
No
Warm, clear airway if necessary, dry, stimulate
No
HR below 100, gasping, or apnea?
No
Labored breathing or persistent cyanosis?
Yes
Yes
PPV, SpO2 monitoring
Clear airway SpO2 monitoring Consider CPAP
Targeted Preductal SpO2 After Birth
1 min 60%-65%
2 min 65%-70%
3 min 70%-75%
4 min 75%-80%
5 min 80%-85%
10 min 85%-95%
HR below 100?
No
Yes
Take ventilation corrective steps
Postresuscitation care
No
HR below 60?
Yes
Consider intubation Chest compressions Coordinate with PPV
Take ventilation corrective steps Intubate if no chest rise!
No
HR below 60?
Yes
Consider:
• Hypovolemia
• Pneumothorax
IV epinephrine
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Figure 3.7
2010 AHA Guidelines for CPR and ECC, Part 15: Neonatal Resuscitation, Page S910, Figure. Newborn Resuscitation algorithm. Reprinted with permission, 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 15: Neonatal Resuscitation. *Circulation* 2010;122[suppl 3]:S909–19 © 2010 American Heart Association, Inc.

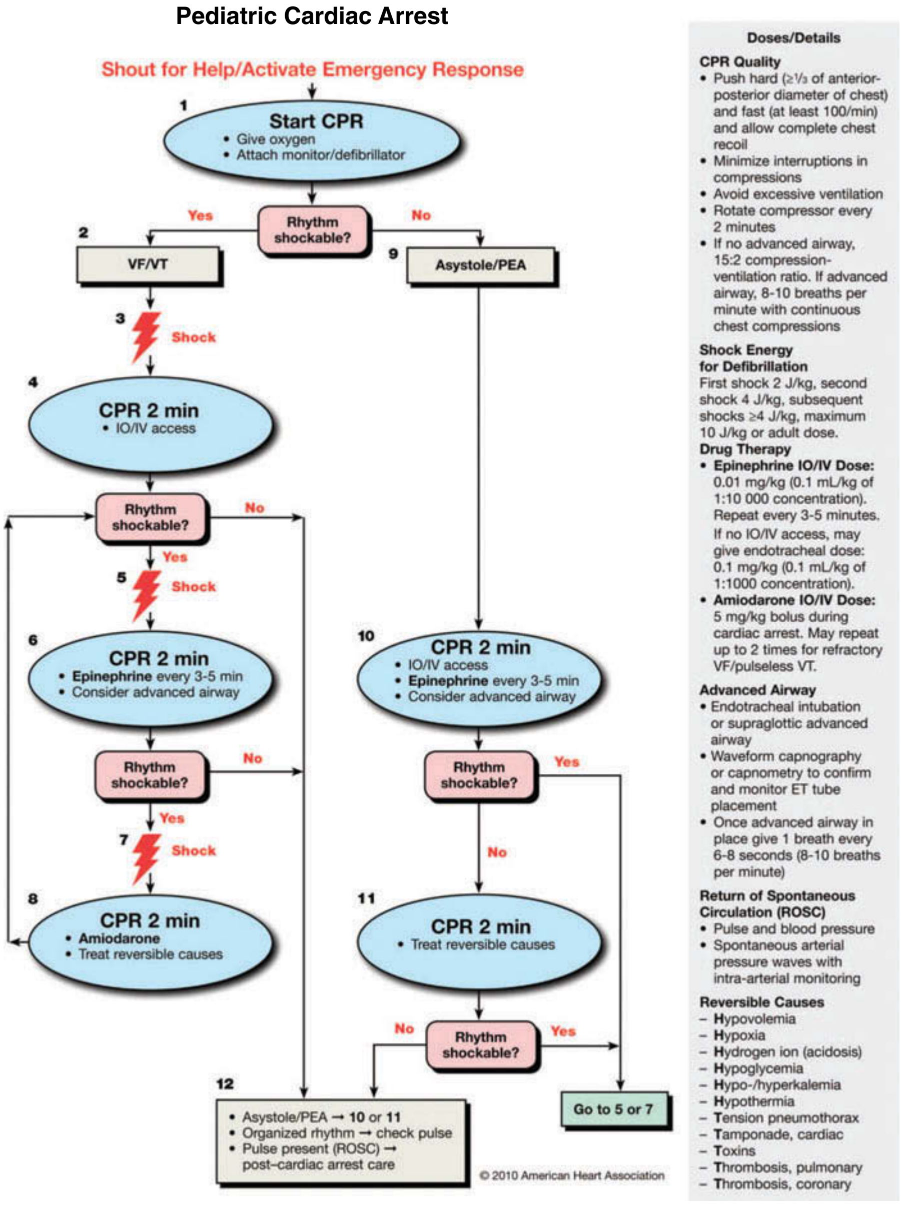
rate of cooling, target temperature and duration of therapy has yet to be determined. Physicians and hospitals not practicing therapeutic induced hypothermia should note that permissive hypothermia, which often develops naturally in the post-arrest victim, does not require treatment, and factors contributing to hyperthermia (fever, shivering) should be aggressively treated with antipyretics, sedation and paralysis as needed.

Ultimately, admission or transfer to an intensive care unit will be required for the care and further assessment of post-cardiac arrest patients. Although it is common for patients to remain comatose, requiring ventilatory support during the immediate post-arrest period, it is very difficult for clinicians to predict a patient's neurologic outcome and survival during this time. It may take 12–72 hours before measurement of brainstem and cortical responses reveal the patient's true neurologic prognosis.

Ethical considerations

Withholding and termination of efforts

Deciding to withhold or terminate resuscitation efforts can be a challenging decision for emergency providers.


Pediatric Cardiac Arrest
Shout for Help/Activate Emergency Response
1
Start CPR
• Give oxygen
• Attach monitor/defibrillator
Yes
Rhythm shockable?
No
2
VF/VT
9
Asystole/PEA
3
Shock
4
CPR 2 min
• IO/IV access
Rhythm shockable?
No
Yes
5
Shock
6
CPR 2 min
• Epinephrine every 3-5 min
• Consider advanced airway
10
CPR 2 min
• IO/IV access
• Epinephrine every 3-5 min
• Consider advanced airway
Rhythm shockable?
No
Rhythm shockable?
Yes
Yes
7
Shock
No
8
CPR 2 min
• Amiodarone
• Treat reversible causes
11
CPR 2 min
• Treat reversible causes
No
Rhythm shockable?
Yes
12
• Asystole/PEA → 10 or 11
• Organized rhythm → check pulse
• Pulse present (ROSC) → post-cardiac arrest care
Go to 5 or 7
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Doses/Details

CPR Quality
- Push hard (≥1/3 of anterior-posterior diameter of chest) and fast (at least 100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 15:2 compression-ventilation ratio. If advanced airway, 8-10 breaths per minute with continuous chest compressions

Shock Energy for Defibrillation
First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥4 J/kg, maximum 10 J/kg or adult dose.

Drug Therapy
- **Epinephrine IO/IV Dose:** 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 concentration).
- **Amiodarone IO/IV Dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.

Advanced Airway
- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place give 1 breath every 6-8 seconds (8-10 breaths per minute)

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes
– Hypovolemia
– Hypoxia
– Hydrogen ion (acidosis)
– Hypoglycemia
– Hypo-/hyperkalemia
– Hypothermia
– Tension pneumothorax
– Tamponade, cardiac
– Toxins
– Thrombosis, pulmonary
– Thrombosis, coronary

Figure 3.8
2010 AHA Guidelines for CPR and ECC, Part 14: Pediatric Advanced Life Support, Page S885, Figure 1. PALS Pulseless Arrest algorithm. Reprinted with permission, 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 14: Pediatric Advanced Life Support. *Circulation* 2010;122[suppl 3]:S876–S908 © 2010 American Heart Association, Inc.

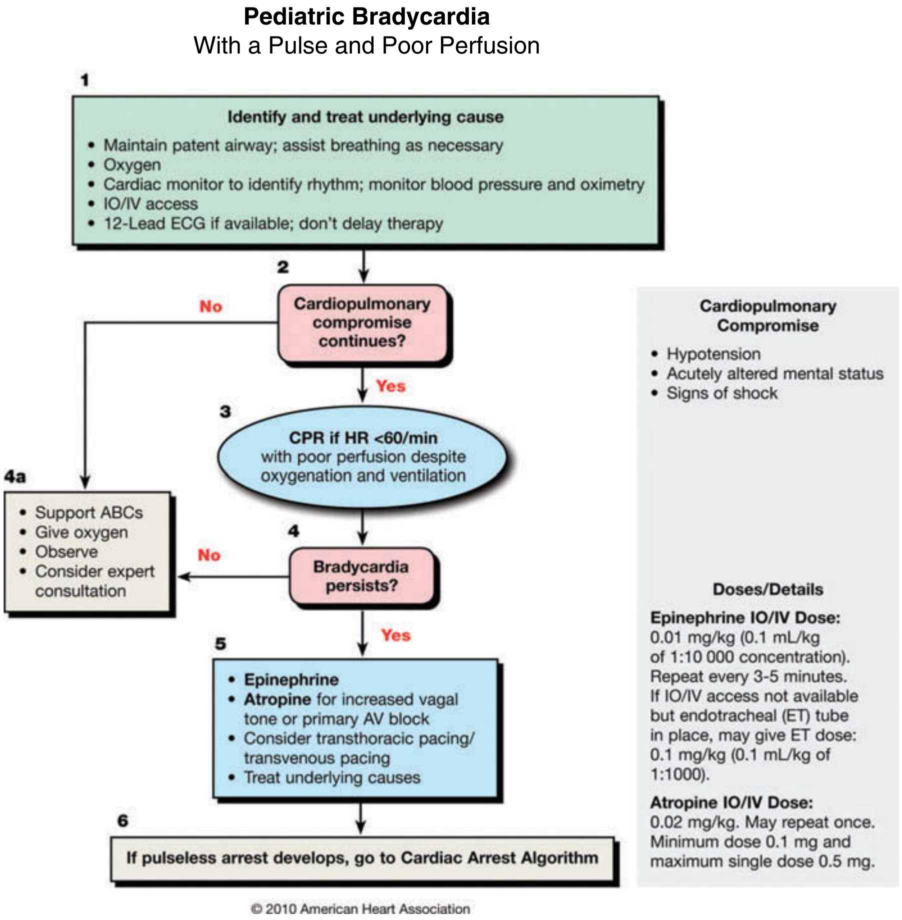
Pediatric Bradycardia
With a Pulse and Poor Perfusion
1
Identify and treat underlying cause
• Maintain patent airway; assist breathing as necessary
• Oxygen
• Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
• IO/IV access
• 12-Lead ECG if available; don't delay therapy
2
Cardiopulmonary compromise continues?
No
Yes
3
CPR if HR <60/min with poor perfusion despite oxygenation and ventilation
4a
• Support ABCs
• Give oxygen
• Observe
• Consider expert consultation
4
Bradycardia persists?
No
Yes
5
• Epinephrine
• Atropine for increased vagal tone or primary AV block
• Consider transthoracic pacing/ transvenous pacing
• Treat underlying causes
6
If pulseless arrest develops, go to Cardiac Arrest Algorithm
Cardiopulmonary Compromise
• Hypotension
• Acutely altered mental status
• Signs of shock
Doses/Details
Epinephrine IO/IV Dose:
0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If IO/IV access not available but endotracheal (ET) tube in place, may give ET dose: 0.1 mg/kg (0.1 mL/kg of 1:1000).
Atropine IO/IV Dose:
0.02 mg/kg. May repeat once. Minimum dose 0.1 mg and maximum single dose 0.5 mg.
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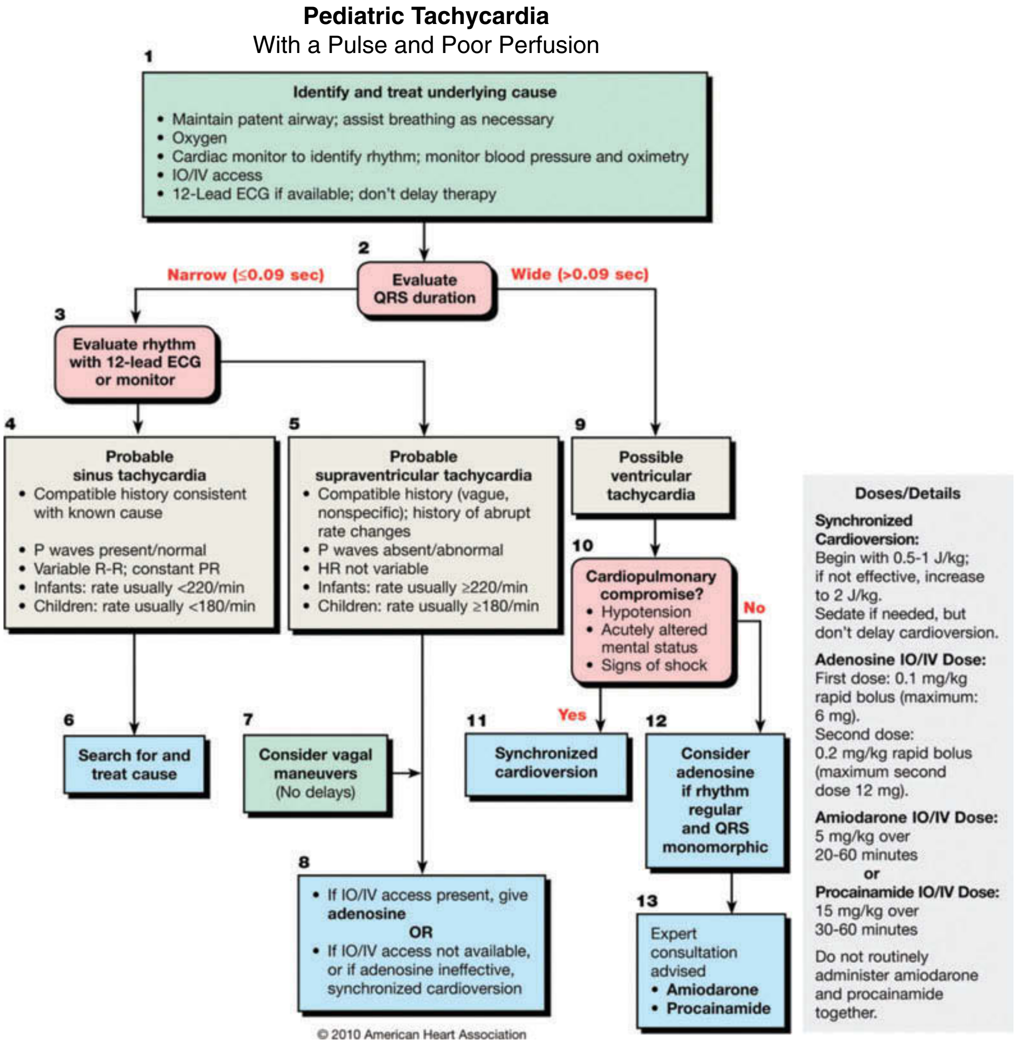
Figure 3.9
2010 AHA Guidelines for CPR and ECC, Part 14: Pediatric Advanced Life Support, Page S887, Figure 2. PALS Bradycardia algorithm. Reprinted with permission, 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 14: Pediatric Advanced Life Support. *Circulation* 2010;122[suppl 3]:S876–S908 © 2010 American Heart Association, Inc.

The care team must take into account the wishes of the patient and family, their own desires to aid sick patients, the likelihood of meaningful survival, and the need to avoid unnecessary suffering and medically futile interventions. Unfortunately, when this decision is made in the emergency setting, limited information about the patient or their pre-arrest condition is typical.

For adult patients in cardiac arrest, it is generally accepted that resuscitation efforts be withheld in patients with a valid "Do Not Resuscitate" (DNR) order, signs of irreversible death (rigor mortis, dependent lividity, decapitation), and in whom no benefit is expected because the patient's condition has deteriorated despite maximal therapy. In newborns, resuscitation should be attempted unless extreme prematurity (gestational age <23 weeks or birth weight <400 g) or congenital abnormalities predict almost certain early mortality or extremely high morbidity. In general, resuscitative efforts should be initiated for all other patients.

Once attempts at resuscitation have been initiated, the decision to terminate efforts can be even more difficult. Emergency providers must weigh multiple factors, the most important of which is the duration of cardiac arrest. The chance of neurologic recovery and discharge from the hospital alive diminishes as resuscitation time increases. Available scientific studies have shown that prolonged resuscitation efforts are unlikely to be successful in the absence of ROSC at any time during 30 minutes of cumulative ACLS. Other factors include historical elements predicting anoxic brain injury, such as pre-arrest state, time until initiation of CPR and defibrillation, and initial arrest rhythm. Additional elements, such as underlying comorbid illness, predict a patient's ability to tolerate resuscitative efforts with a meaningful outcome, but not reliably. Studies in children and neonates show justification for ceasing efforts if the patient shows no signs of life after 10 minutes of adequate resuscitation. Reversible causes of cardiac arrest, such as drug overdose, electrolyte abnormalities or profound hypothermia, should be taken into account when considering termination of efforts. Treatment of these conditions may improve the efficacy of the resuscitation effort and the patient's chances of survival.


Pediatric Tachycardia
With a Pulse and Poor Perfusion
1
Identify and treat underlying cause
• Maintain patent airway; assist breathing as necessary
• Oxygen
• Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
• IO/IV access
• 12-Lead ECG if available; don't delay therapy
2
Evaluate QRS duration
Narrow (≤0.09 sec)
Wide (>0.09 sec)
3
Evaluate rhythm with 12-lead ECG or monitor
4
Probable sinus tachycardia
• Compatible history consistent with known cause
• P waves present/normal
• Variable R-R; constant PR
• Infants: rate usually <220/min
• Children: rate usually <180/min
5
Probable supraventricular tachycardia
• Compatible history (vague, nonspecific); history of abrupt rate changes
• P waves absent/abnormal
• HR not variable
• Infants: rate usually ≥220/min
• Children: rate usually ≥180/min
9
Possible ventricular tachycardia
10
Cardiopulmonary compromise?
• Hypotension
• Acutely altered mental status
• Signs of shock
No
Yes
6
Search for and treat cause
7
Consider vagal maneuvers (No delays)
11
Synchronized cardioversion
12
Consider adenosine if rhythm regular and QRS monomorphic
8
• If IO/IV access present, give adenosine
OR
• If IO/IV access not available, or if adenosine ineffective, synchronized cardioversion
13
Expert consultation advised
• Amiodarone
• Procainamide
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Doses/Details
Synchronized Cardioversion:
Begin with 0.5-1 J/kg; if not effective, increase to 2 J/kg. Sedate if needed, but don't delay cardioversion.
Adenosine IO/IV Dose:
First dose: 0.1 mg/kg rapid bolus (maximum: 6 mg).
Second dose: 0.2 mg/kg rapid bolus (maximum second dose 12 mg).
Amiodarone IO/IV Dose:
5 mg/kg over 20-60 minutes
or
Procainamide IO/IV Dose:
15 mg/kg over 30-60 minutes
Do not routinely administer amiodarone and procainamide together.

Figure 3.10 2010 AHA Guidelines for CPR and ECC, Part 14: Pediatric Advanced Life Support, Page S888, Figure 3. PALS Tachycardia algorithm. Reprinted with permission, 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 14: Pediatric Advanced Life Support. *Circulation* 2010;122[suppl 3]:S876–S908 © 2010 American Heart Association, Inc.

Following any resuscitation, a timely debriefing should be conducted for as many members of the resuscitation team as possible. These are best lead by personnel specifically trained in psychology, psychiatry, or critical debriefing.

Involvement of the family

Traditionally, family members have not been present during resuscitation. Because most resuscitations are unsuccessful, providers generally assume that family members do not wish to witness a loved one's death. Additionally, providers might fear families will misunderstand events, interfere with resuscitations, and be more likely to pursue litigation if death occurs. Experience and research have demonstrated these assumptions to be incorrect. Allowing selected family members to witness resuscitation efforts may help the family accept their loss and aid in their grieving process. The resuscitation care team must be adequately prepared to provide real-time support to the family members, including assigning a team member to the family to offer simple explanations, answer questions and provide comfort. They should be seated if possible and positioned so as not to interrupt or delay critical procedures. Providers should note that most family members assume they are not allowed to be present, and will remain outside the room unless invited in.

When all BLS and ACLS measures have been reasonably attempted and the likelihood of survival is minimal, resuscitation efforts should be discontinued. Informing family members of the death of a loved one is an extremely difficult yet critical responsibility faced by emergency providers. Prior to such disclosure, family members should be gathered in a quiet and private area. Social service personnel and nursing staff should be asked to assist. It is best to be honest and straightforward, using language that is appropriate for the family's education level and culture. Ask them their understanding of events up to this point. Briefly relate the circumstances regarding the resuscitation efforts, ending with the news that their loved one is dead. Avoid terminology such as "passed away," "is gone," or "in another place," which may lead to confusion. Family will often want to know what, if anything, they could have done to change the outcome. It is important to reassure them that they did nothing wrong and that their loved one did not suffer, if these are appropriate. Enlist the support of social services, clergy, or other culturally appropriate personnel to assist with important issues of autopsy, organ donation and viewing. Express sympathy, answer questions and make sure that there is reasonable social support before leaving.

Special patients

Pediatric

The evaluation and treatment of cardiopulmonary arrest in the pediatric population is particularly challenging. Unlike adults, pediatric cardiac arrests most commonly result from respiratory causes. Recent guidelines, however, have increased emphasis on optimizing CPR and circulation. Reduced familiarity with procedures as well as anatomic issues (i.e., decreased size of structures) make definitive airway management and vascular access more challenging in pediatric patients. In addition, psychosocial issues are generally more complex in these patients. Neonatal advanced life support (NALS), pediatric advanced life support (PALS) and advanced pediatric life support (APLS) courses exist to teach these differences. The cardiopulmonary arrest algorithms are similar between children and adults, although the energy of defibrillation and medication dosing are weight-based. The Broselow tape, which bases a neonate's or child's weight on their length, is an essential piece of equipment for pediatric resuscitations. It has the appropriate medication doses, equipment sizes and defibrillation energies listed for the appropriate length (weight), and is color-coded. Many EDs arrange pediatric resuscitation equipment by these colors in order to make the proper equipment more readily accessible during resuscitation. Although detailed discussion of these scenarios is beyond the scope of this chapter, the algorithms for neonatal and pediatric resuscitations are provided (Figures 3.7–3.10).

Acknowledgment

Drs. Lin and Strehlow wish to acknowledge Robert R. Leschke, MD for his contribution to the first edition.

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4 Cardiac dysrhythmias

Swaminatha V. Gurudevan, MD

Scope of the problem

Cardiac dysrhythmias are an important first manifestation of cardiovascular disease. Heart disease is the leading single cause of death in the United States, accounting for 25.4% of all deaths. A total of 435,687 Americans died of coronary heart disease in 2005, amounting to approximately one death every 60 seconds. Even more striking was the fact that nearly half of these deaths occurred before the patient reached a hospital. Most of these were sudden deaths, usually resulting from ventricular fibrillation. Despite the strong link between cardiac dysrhythmias and cardiovascular disease, rhythm disturbances may also occur in the absence of structural heart disease or as a result of generalized systemic illness.

Proper identification of cardiac dysrhythmias is a vital skill for emergency providers. A critical aspect is the differentiation of benign from malignant dysrhythmias. The appropriate identification of the rhythm disturbance and a solid understanding of the underlying disease process are critical to the appropriate short- and long-term management of the patient. Dysrhythmias can be broadly divided into three categories: tachydysrhythmias, bradydysrhythmias, and disorders of conduction. The 2010 Advanced Cardiovascular Life Support (ACLS) guidelines place a great emphasis not only on identification of the rhythm disturbance, but also on recognition of patients with left ventricular (LV) systolic dysfunction, as these patients are known to have a significantly higher mortality from each dysrhythmia.

Anatomic essentials

The sinoatrial node

Normal cardiac conduction is initiated by the dominant pacemaker of the heart, the sinoatrial (SA) node (Figure 4.1). The SA node is located at the junction of the right atrium and superior vena cava, and its vascular supply is from the SA nodal artery, which originates from the right coronary artery in 55% of patients and the left circumflex artery in the remaining 45% of patients. The SA node is innervated by parasympathetic fibers from the vagus nerve and sympathetic fibers from the thoracic sympathetic trunk. Its normal discharge rate is between 60 and 100 times per minute.

The atrioventricular node

In the normal heart, conduction proceeds through the atrial fibers to the atrioventricular (AV) node, which is located beneath the right atrial endocardium directly above the insertion of the septal leaflet of the tricuspid

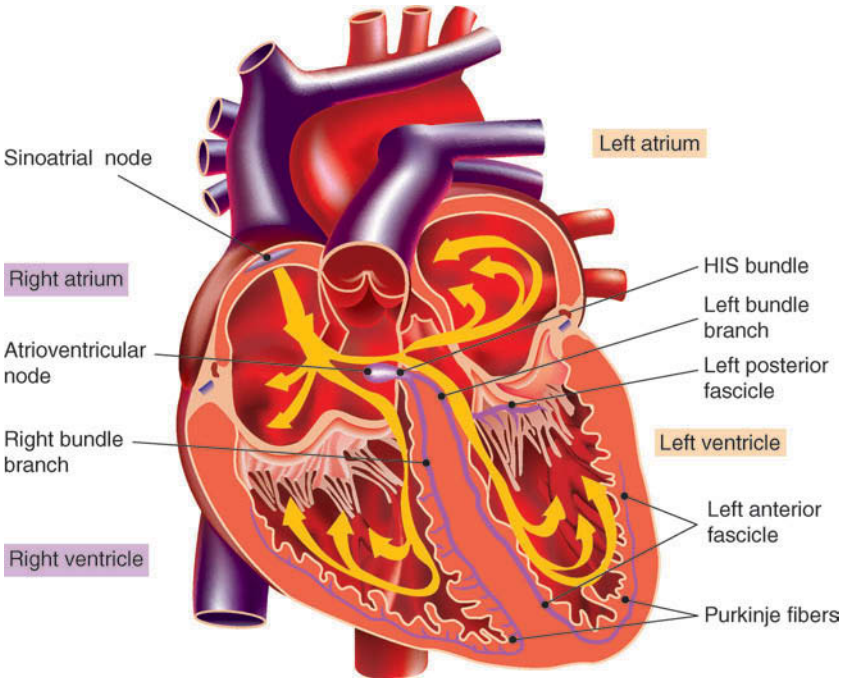
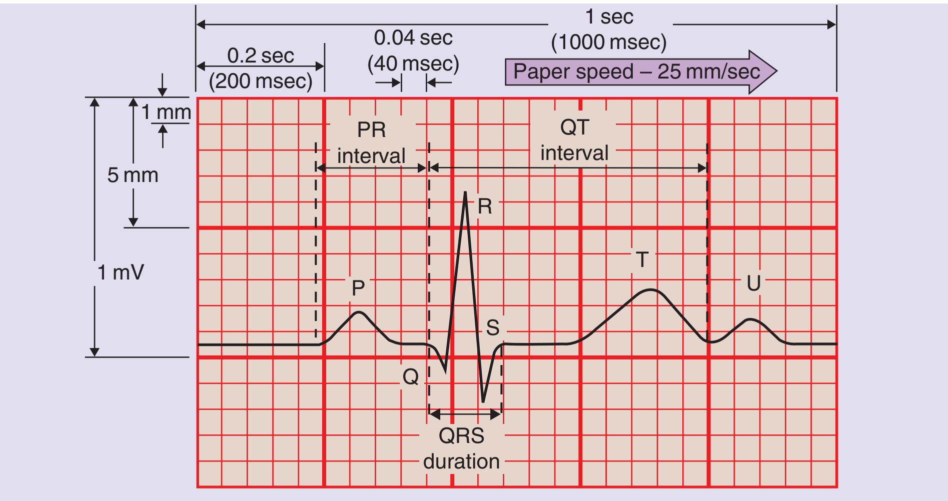


Figure 4.1
The cardiac conduction system. Copyright © 2000, General Electric.



Vertical axis	
Vertical axis	1 small square = 1 mm (0.1 mV)
	1 large square = 5 mm (0.5 mV)
	2 large squares = 1 mV

Horizontal axis	
Horizontal axis	1 small square = 0.04 sec (40 msec)
	1 large square = 0.2 sec (200 msec)
	5 large squares = 1 sec (1000 msec)

Figure 4.2
Components of the electrocardiogram. Copyright © 2000, General Electric.

valve. On the electrocardiogram (ECG), atrial depolarization is represented by the P wave (Figure 4.2). The AV nodal artery provides the blood supply for the AV node, arising in the majority of cases (90%) from the right coronary artery. In patients with a left-dominant or co-dominant coronary circulation (10%), the AV nodal artery may arise from the left circumflex. Physiologically, the AV node slows conduction velocity to allow greater time for ventricular filling during diastole. In addition, its long refractory period protects the ventricles from excessively rapid stimulation, which could cause inadequate diastolic filling time and acute cardiac failure. The AV node is innervated by the same parasympathetic and sympathetic fibers as the SA node. On the ECG, the PR interval represents the time between the onset of depolarization in the atria and the onset of depolarization in the ventricles, and is used as an estimation of AV nodal conduction time. The normal PR interval is between 0.12 and 0.20 seconds. Prolongation of the PR interval may occur as a result of excessive vagal stimulation, drugs affecting the AV node, AV nodal ischemia or underlying conduction system disease.

The His–Purkinje system

Depolarization proceeds from the AV node to the bundle of His, which is composed of rapidly conducting Purkinje fibers. The bundle divides in the muscular interventricular septum into two major branches: the left and right bundle branches, which innervate the left and right ventricles (LV and RV), respectively. The left bundle branch divides into the left anterior and left posterior fascicles. Ventricular conduction and depolarization through the His–Purkinje system are represented on the surface ECG by the QRS complex. Normal QRS width is 0.06-0.1 seconds. Widening of the QRS complex beyond 0.12 seconds represents ventricular conduction delay, which can occur as a result of bundle branch blocks, aberrant conduction, electrolyte abnormalities, drugs affecting the myocardium, or rhythms that originate in the ventricular myocardium. Infranodal tissue does not have parasympathetic innervation, an important consideration when administering anticholinergic drugs such as atropine. Atropine will not improve a conduction block that originates below the AV node in the His–Purkinje system, and may actually worsen it.

Ventricular repolarization

Repolarization of the ventricular myocardium is represented by the T wave. Mechanical contraction typically follows depolarization through excitation–contraction coupling and fractional shortening of cardiac myocytes. The QT interval, which represents ventricular depolarization and repolarization time, is dependent to some extent on heart rate. A corrected QT interval (QT_c) is obtained by dividing the measured QT interval by the square root of the RR interval. A normal QTc is less than 0.47 seconds. Prolongation of the QT interval can occur secondary to drug effects, electrolyte abnormalities and congenital abnormalities. A prolonged repolarization period increases the “vulnerable period” of the ventricle, during which premature ventricular contraction can trigger a reentrant ventricular tachydysrhythmia.

Tachycardias and bradycardias

The normal range of heart rates in a healthy adult with an intact sinus node is 60-100 beats per minute. *Bradycardia* is defined as a heart rate less than 60 beats per minute, whereas *tachycardia* is defined as a heart rate greater than 100 beats per minute.

History

The history is the most important component in the evaluation of a patient with a cardiac dysrhythmia. Patients with cardiac dysrhythmias may complain of palpitations, or the sensation of a rapid or irregular heart rhythm. They may not notice the abnormal heart rhythm and may instead complain of chest pain, shortness of breath, lightheadedness, fatigue, presyncope, syncope or convulsions.

Do you have chest pain or shortness of breath?

Chest pain or shortness of breath may reflect underlying cardiac ischemia. Patients with underlying coronary artery disease (CAD) can experience symptoms of demand-related ischemia during a tachydysrhythmia or a bradydysrhythmia. Alternatively, ischemia can cause both tachydysrhythmias and bradydysrhythmias. In fact, sudden death from unstable tachydysrhythmias such as ventricular fibrillation (VF) and ventricular tachycardia (VT) represent the leading cause of out-of-hospital death from cardiac causes and is the most common initial presenting symptom of CAD in the United States. A history of exertional or rest angina may provide clues to an acute coronary syndrome. Similarly, a history of orthopnea, paroxysmal nocturnal dyspnea or lower extremity edema may suggest underlying LV dysfunction, which would increase the risk of sudden death from ventricular tachydysrhythmias.

Did you feel lightheaded or dizzy, or lose consciousness?

Syncope is an important finding, as it portends a poorer prognosis in patients with either tachydysrhythmias or bradydysrhythmias, mandating an aggressive work-up to exclude a dysrhythmogenic cause of syncope. Cerebral hypoperfusion from lack of cardiac output is the mechanism of cardiogenic syncope; cerebral hypoperfusion can also manifest as a seizure or an alteration in level of consciousness.

When did your symptoms begin, and if they are episodic, how often do they occur and how long do they last?

If the patient complains of chest pain or palpitations, it is crucial to know how long these symptoms last (seconds, minutes, or hours). How many times a day or week do they occur? During the work-up of a cardiac dysrhythmia, it is important to correlate the presence of the dysrhythmia with concomitant clinical symptoms – chest pain, shortness of breath, or presyncope/syncope. The frequency of symptom occurrence may dictate the type of monitoring device (Holter or event monitor) to use when planning an outpatient work-up.

Do you have a previous history of coronary artery disease (CAD), congestive heart failure (CHF), dysrhythmias or valvular heart disease? Have you had prior cardiac surgery?

Given that CAD, CHF, and primary dysrhythmias are recurrent illnesses, it is important to identify those patients with a prior history of CAD or CHF, as these patients may be more likely to manifest certain dysrhythmias. Valvular heart diseases such as mitral stenosis and mitral regurgitation can predispose to atrial tachydysrhythmias. Atrial fibrillation, AV nodal reentrant tachydysrhythmias and ventricular tachydysrhythmias can have a relapsing and remitting course, and often recur. Certain dysrhythmias, especially atrial fibrillation and VT, can occur following cardiac surgery. These rhythm disturbances have a different management and prognosis in this setting.

Do you have a pacemaker or implanted defibrillator?

Pacemakers are typically implanted for symptomatic bradydysrhythmias such as sinus bradycardia, sick sinus syndrome, or high-degree AV block. A four- or five-letter code assigned to each type of pacemaker describes the chamber paced, the chamber sensed, the response to sensing and the rate adaptation programmability of the pacemaker. For example, a VVIR pacemaker is a single-chamber pacemaker that paces the ventricle (V), senses the ventricle (V), inhibits pacing (I) if a native beat is sensed, and has rate modulation programmability (R). A DDDR pacemaker is a dual-chamber pacemaker that paces both the atrium and the ventricle (D), senses both the atrium and the ventricle (D), either inhibits pacing of or triggers pacing of both the atrium and the ventricle (D) in response to sensing, and is rate-modulation programmable (R). Biventricular pacemakers employed for cardiac resynchronization therapy (CRT) in heart failure patients have a third lead in the coronary sinus that allows for synchronized pacing of the LV and RV.

Implantable cardioverter defibrillators (ICDs) are more common today given recent clinical trials demonstrating their effectiveness in preventing sudden death in certain patients. These include patients with CAD and LV dysfunction, patients with prior episodes of VT, or patients resuscitated after ventricular fibrillation arrest (termed sudden cardiac death). They are almost always dual-chamber devices and also function as pacemakers.

Both pacemakers and ICDs have a stored memory that can be interrogated by the pacemaker company representative or a skilled cardiologist. This information can be extremely helpful in the analysis of a current or recent dysrhythmia, and can serve as a "continuous telemetry box" for the patient. The patient typically carries a card with the pacemaker company and the model; a company representative is generally available 24 hours a day to interrogate the pacemaker if necessary.

Are you taking cardiac, non-prescription, herbal or alternative medications? What about illicit substances?

Medications and drug interactions are an important cause of bradydysrhythmias, tachydysrhythmias and conduction system disorders. Beta-blockers (atenolol, metoprolol, carvedilol) and calcium channel blockers (verapamil, diltiazem) can be negatively chronotropic and contribute to bradydysrhythmias and conduction system disorders. Digoxin toxicity can be responsible for bradydysrhythmias, conduction system disturbances and tachydysrhythmias by increasing the parasympathetic tone at the SA and AV nodes and by increasing automaticity in the ventricular myocardium. Antihistamines, neuroleptics and gastrointestinal (GI) medications such as metoclopramide can prolong the QT interval and thereby predispose to ventricular tachydysrhythmias. Herbal medications such as ephedra (Ma Huang) or jimsonweed tea can have sympathomimetic or anticholinergic effects, respectively. Excessive caffeine intake can have a sympathomimetic effect and can shorten the refractory period in the slow pathway of the AV node, predisposing one to AV nodal reentrant tachycardia (AVNRT). Sympathomimetic drug abuse (cocaine, crystal methamphetamine) usually causes tachydysrhythmias. Injection drug use can contribute to the development of infectious endocarditis, which may manifest as heart block.

Do you have any other medical problems, such as chronic obstructive pulmonary disease, renal failure or thyroid disease?

Chronic pulmonary diseases can predispose patients to atrial tachydysrhythmias, especially multifocal atrial tachycardia (MAT). Inhaled beta-agonists and anticholinergic agents used to treat chronic obstructive pulmonary disease (COPD) and asthma can contribute to tachydysrhythmias as well. Renal failure can contribute to hyperkalemia, which may cause heart blocks and bradydysrhythmias, and worsens digoxin toxicity. Hypocalcemia from chronic renal failure can cause prolongation of the QT interval. Hypothyroidism can present with significant sinus bradycardia. Thyrotoxicosis can present with sinus tachycardia and is an important cause of atrial fibrillation.

Is there a family history of dilated cardiomyopathy, sudden cardiac death or early coronary artery disease?

Patients with a strong family history have a higher risk of similar diseases and carry a poorer prognosis. Dilated cardiomyopathy is known to have both X-linked and autosomal dominant inheritance, whereas hypertrophic cardiomyopathy is an autosomal dominant condition. Certain hereditary conditions such as Brugada syndrome predispose individuals to VT and sudden cardiac death. A history of a first-degree relative with CAD before the age of 50 years is an independent risk factor for coronary events.

Physical examination

General appearance

This is perhaps the most important part of the physical examination in terms of guiding the management of a cardiac dysrhythmia. Does the patient appear ill? Is the patient clinically stable or unstable? A patient is clinically unstable if they have evidence of end-organ hypoperfusion as a direct result of the dysrhythmia. This may be manifested as severe chest pain, hypotension due to myocardial ischemia, or respiratory failure with pulmonary edema. Patients who are clinically unstable require immediate aggressive, focused management of their dysrhythmia, including medications, cardioversion, defibrillation or pacing according to ACLS guidelines. Patients who are clinically stable can be evaluated and treated in a more methodical fashion.

Vital signs

Is the patient hypertensive or hypotensive? The absolute blood pressure may be deceiving, and comparing the current blood pressure with previous normal blood pressures should be done. For example, a blood pressure of 100/50 mmHg in an elderly patient with hypertension and CAD whose normal blood pressure is 160/90 mmHg may be more significant than a blood pressure of 85/50 mmHg in a young, healthy female without prior history of cardiac disease. Assess the heart rate as well as the caliber and regularity of the pulses. Atrial fibrillation typically presents with an irregularly irregular pulse.

Skin

Inspect the skin for pallor, cyanosis or duskiness, which reflect tissue hypoperfusion. Palpate the skin to assess the temperature and moisture. In thyrotoxicosis, the skin is typically warm and moist; cool or clammy skin suggests hypoperfusion.

Head, eyes, ears, nose and throat

Look for exophthalmos, which may be a physical finding of Graves disease. Look for nasal flaring, which may reflect acute respiratory distress and air hunger. Examination of the oral mucous membranes provides clues towards the patient's hydration status. Look for perioral cyanosis, another sign of tissue hypoperfusion.

Neck

Inspect the level of the jugular venous pulsations to assess the patient's volume status. Press below the costal margin to assess for hepatojugular reflux. When seen, cannon A waves in the jugular venous pulse suggest AV dissociation, which can occur in third-degree heart block and VT. The cannon A waves reflect atrial contraction against a closed tricuspid valve. Inspect the thyroid gland for a goiter, thyroidectomy scar or any nodularity.

Cardiovascular

Inspect the chest wall for the point of maximal impulse (PMI). Palpate the PMI and note any displacement. The normal position of the PMI is the 5th intercostal space in the midclavicular line. Inferior and lateral displacement of the PMI to the anterior axillary or midaxillary line can occur with progressive LV dilation and failure. Palpate for an RV parasternal heave, which can reflect RV failure. Next, auscultate the heart, listening for the regularity of rhythm, the loudness and splitting of S1 and S2, and systolic or diastolic murmurs. Atrial fibrillation is most commonly associated with an irregularly irregular rhythm. A left bundle branch block can cause S2 to be paradoxically split (A2 will come after P2, and inspiration will cause the split to come together). A right bundle branch block can cause wider splitting of S2. Listen for an S3 gallop, which reflects LV failure and high filling pressures. Mitral stenosis and aortic insufficiency have distinct diastolic murmurs, whereas aortic stenosis and mitral regurgitation have systolic murmurs. Finally, assess the quality of the pulses and evaluate capillary refill.

Chest and lungs

Look for a midline sternotomy scar that may reflect prior cardiac surgery. Inspect for accessory muscle use for breathing. Pulmonary rales or wheezes may reflect volume overload and LV failure. Percuss the chest wall for dullness and listen for decreased breath sounds; these findings may suggest a pleural effusion and volume overload.

Abdomen

Inspect for any evidence of abdominal distention or ascites. Palpate the liver edge; a pulsatile liver may reflect pulmonary hypertension with significant tricuspid regurgitation.

Extremities

Inspect the extremities for their degree of warmth. The presence of cyanosis or clubbing may indicate chronic pulmonary disease. Pitting edema may reflect volume overload.

Neurologic

Assess the level of consciousness. Is the patient's mental status different from baseline? Is there a focal neurologic deficit that warrants investigation of a cerebrovascular accident related to the dysrhythmia?

Diagnostic testing

Electrocardiogram with rhythm strip

A 12-lead ECG is an essential part of the initial evaluation of a patient with a cardiac rhythm disturbance. All patients with a cardiac dysrhythmia should be on continuous telemetry monitoring and have a 12-lead ECG performed on arrival to the emergency department.

Radiologic studies

All patients should have a portable chest X-ray performed to evaluate the cardiac silhouette, assess for pulmonary vascular congestion and confirm the appropriate placement of pacemaker or defibrillator leads, if present. Pacemaker lead fractures, although difficult to identify, may be a cause for pacemaker malfunction or failure.

Laboratory studies

Cardiac enzymes

Serum creatine kinase (CK), CK-MB and troponin I should be considered in all patients in whom myocardial ischemia is suspected. CK-MB begins to rise 4 hours after myocardial injury, but is not always specific for myocardial injury. Troponin I rises 6 hours after myocardial injury, and remains elevated for several days following the injury. It is nearly 100% specific for myocardial injury, and can establish that myocardial necrosis has occurred so that appropriate disposition and treatment of the patient can be carried out. In addition, troponin I helps to risk stratify patients presenting with a cardiac dysrhythmia, as those with elevated troponin I are likely to have myocardial damage.

Electrolytes

A stat serum electrolyte panel should be obtained in every patient with a new cardiac dysrhythmia. In particular, serum potassium, calcium and magnesium should be evaluated. If the patient is clinically unstable, some of these tests can be ordered as part of an arterial blood gas analysis, with the results available more rapidly. Hyperkalemia predisposes the patient to bradydysrhythmias and heart block. It causes flattening of the P wave, peaking of the T wave and widening of the QRS complex. Conversely, hypokalemia may predispose individuals to ventricular tachydysrhythmias. Severe hypokalemia results in a more prominent P wave, a flattened T wave and a prominent U wave seen following the T wave on the ECG. Hypocalcemia may prolong the QT interval, whereas hypercalcemia can result in shortening of the QT interval. Serum magnesium levels are also important, as levels influence the body's potassium homeostasis. Hypomagnesemia can cause prolongation of the QT interval and predispose a patient to torsades de pointes.

Thyroid function tests

A serum thyroid-stimulating hormone (TSH) should be obtained in patients with new onset atrial fibrillation or inappropriate sinus tachycardia. If the TSH is abnormal, a complete thyroid panel should also be obtained. Similarly, patients with unexplained sinus bradycardia should have a TSH drawn to rule out significant hypothyroidism. Although these results are rarely available during a patient's emergency department course, they are of use to the physician who admits the patient or sees the patient in follow-up.

Drug levels

A digoxin level should be obtained in patients taking this medication. Digoxin toxicity should be suspected if the patient presents with symptomatic bradycardia, high-grade AV block, atrial tachycardias with block or bidirectional VT. A urine toxicology screen should also be obtained, especially from those patients in whom illicit sympathomimetic substance abuse is suspected.

Management of bradydysrhythmias

General management

After assessing and securing (if necessary) the airway, breathing and circulation; obtaining intravenous (IV) access; and placing the patient on a cardiac monitor, a 12-lead ECG should be obtained. Stat blood tests should be ordered as discussed.

First, assess for the presence of serious signs or symptoms due to the bradydysrhythmia. These include hypotension, impaired tissue perfusion or any alteration in sensorium. If present, treatment should be initiated immediately.

IV fluids should be started if there is no overt evidence of CHF. Atropine should be given as it is effective in reversing supranodal causes of bradycardia and may reverse functional AV nodal conduction block. Two milligrams of atropine causes complete vagal blockade, so it is unlikely that higher doses will contribute to improvement. However, research is ongoing about the appropriate maximum dose. Current ACLS guidelines recommend a maximum dose of 0.04 mg/kg for symptomatic bradycardia.

If symptomatic bradycardia persists, the next medication to administer is IV dopamine at the beta-receptor dosing range of 2–10 mcg/kg/min. Dopamine is both positively chronotropic and ionotropic, and may assist with hypotension. It should be given through a central line if possible to avoid dopamine-induced skin necrosis. IV epinephrine, a beta-predominant sympathomimetic agent, may be necessary at an infusion rate of 2–10 mcg/kg/min. Isoproterenol is no longer part of the 2010 ACLS guidelines (Figure 4.3).

If serious signs and symptoms of bradycardia persist despite appropriate medical therapy, transcutaneous pacing should be initiated with preparations for urgent temporary transvenous pacemaker placement.

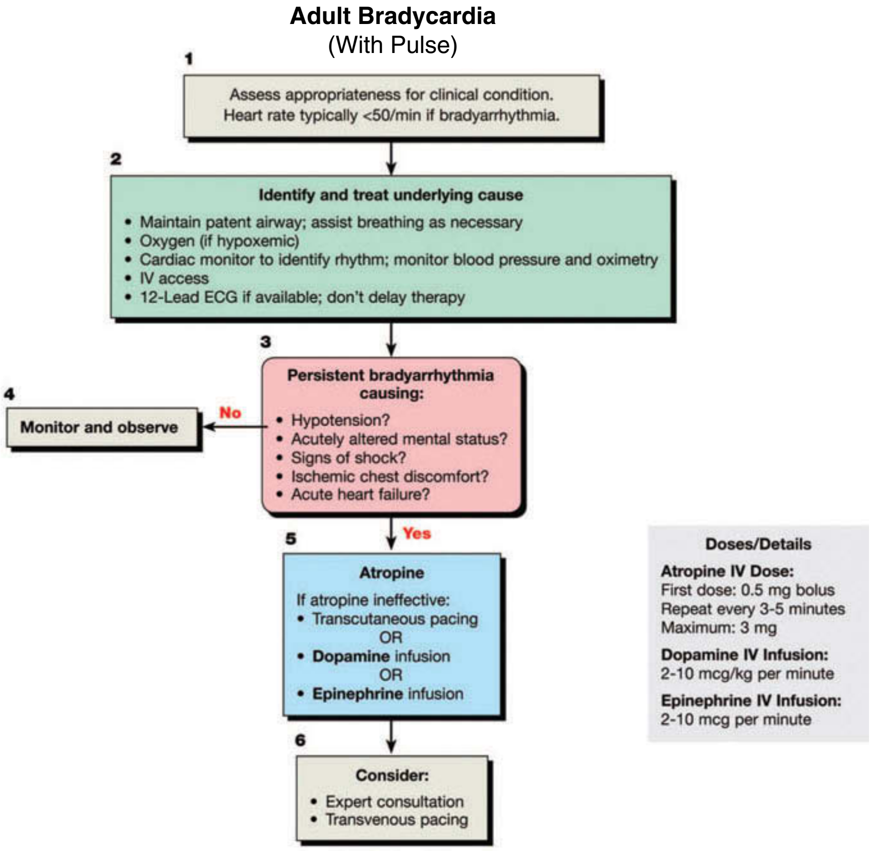

Adult Bradycardia
(With Pulse)
1
Assess appropriateness for clinical condition.
Heart rate typically <50/min if bradyarrhythmia.
2
Identify and treat underlying cause
• Maintain patent airway; assist breathing as necessary
• Oxygen (if hypoxemic)
• Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
• IV access
• 12-Lead ECG if available; don't delay therapy
3
Persistent bradyarrhythmia causing:
• Hypotension?
• Acutely altered mental status?
• Signs of shock?
• Ischemic chest discomfort?
• Acute heart failure?
4
Monitor and observe
No
Yes
5
Atropine
If atropine ineffective:
• Transcutaneous pacing
OR
• Dopamine infusion
OR
• Epinephrine infusion
6
Consider:
• Expert consultation
• Transvenous pacing
Doses/Details
Atropine IV Dose:
First dose: 0.5 mg bolus
Repeat every 3-5 minutes
Maximum: 3 mg
Dopamine IV Infusion:
2-10 mcg/kg per minute
Epinephrine IV Infusion:
2-10 mcg per minute

Figure 4.3
Bradycardias. bpm: beats per minute; ABCD: Airway, Breathing, Circulation, Defibrillation. Reproduced with permission, ACLS Provider Manual, © 2010 American Heart Association.

Management of specific bradydysrhythmias

Sinus bradycardia

Sinus rates of less than 60 beats per minute are termed sinus bradycardia. Sinus bradycardia is commonly observed in individuals with a high resting vagal tone (athletes) or patients on negatively chronotropic medications (beta-blockers, calcium channel blockers, digoxin, amiodarone and clonidine). Sinus bradycardia can also be seen early in the course of an acute inferior wall myocardial infarction, triggered by parasympathetic stimulation known as the Bezold–Jarisch reflex.

Sinus bradycardia should only be treated if there are associated symptoms. Elimination of reversible aggravating factors is an essential first step in management. This includes discontinuing negatively chronotropic medications and considering administration of reversal agents (IV glucagon for beta-blockers, IV calcium gluconate for calcium channel blockers, and Digoxin Immune Fab for digoxin). If the symptoms persist despite discontinuation of all bradycardia-aggravating medications or if the medications are essential to the patient's overall management, permanent pacemaker placement is indicated. Patients with an inappropriate sinus bradycardia should be investigated to rule out myocardial ischemia, significant hypothyroidism, adrenal insufficiency, overmedication, or certain uncommon infectious diseases.

Ectopic atrial rhythm or wandering atrial pacemaker

This dysrhythmia is caused by an ectopic atrial focus distinct from the sinus node that represents the dominant sinus rhythm. On the ECG, ectopic P waves are recognized as being different from those in the patient's usual rhythm, and the PR interval may vary from the patient's baseline PR interval, depending on the location of the ectopic atrial focus. If three or more different atrial foci are seen, the rhythm is termed a *wandering atrial pacemaker*. These rhythms do not have clinical significance, and no specific treatment is required unless warranted by symptoms.

Sinoatrial block (sinus exit block)

Sinoatrial (SA) block is characterized by the absence of atrial depolarization. This can occur due to the SA node's failure to generate an impulse or failure of the SA nodal impulse to conduct to the atria. On the ECG, P waves are typically absent. The most common factors that predispose to SA block are ischemia, hyperkalemia, excessive vagal tone, or negative chronotropic drugs.

Typically, an alternate region of myocardium becomes the dominant pacemaker and manifests an escape rhythm. Junctional escape rhythms are usually narrow-complex rhythms at 45-60 beats per minute, whereas escape rhythms originating from the His–Purkinje system are wide-complex with a rate of 30-45 beats per minute. Treatment of SA block with escape rhythms is indicated based on the patient's symptoms.

Sick sinus syndrome

Sick sinus syndrome is a syndrome of abnormalities in cardiac impulse formation and AV conduction that manifest as combinations of tachydysrhythmias and bradydysrhythmias. It is also referred to as *tachy–brady syndrome*. Most patients present to the emergency department with symptomatic bradydysrhythmias and a history of episodic palpitations. The ECG manifestations include SA block and sinus or atrial bradycardia with bursts of an atrial tachydysrhythmia (usually atrial fibrillation). Treatment of this syndrome is directed towards the specific manifestation of the syndrome – either augmentation of rate with atropine if the patient has bradycardia, or rate control of an atrial tachydysrhythmia with a beta-blocker, calcium channel blocker or digoxin. One must exercise caution with these agents, as they may lead to excessive tachycardia or bradycardia. In the long term, most patients require a permanent pacemaker for support during excessive sinus bradycardia and an antidysrhythmic medication to suppress tachydysrhythmias.

First-degree atrioventricular block

AV block is divided into three grades, based on the ECG characteristics and the degree of the block. AV block is the result of impaired conduction through the atria, AV node, or His–Purkinje system.

First-degree AV block is defined as prolonged AV conduction without loss of conduction of any single atrial impulse. On the ECG, it is manifested by a PR interval greater than 0.20 seconds. In first-degree AV block, the ventricular rate is not slow unless there is concomitant sinus bradycardia. No specific treatment is indicated. Negatively chronotropic medications should be used with caution in these patients.

Second-degree atrioventricular block

In second-degree AV block, most but not all atrial impulses are conducted to the ventricles. It is divided into two subtypes based on the ECG appearance and the underlying pathophysiology.

Type I second-degree

Type I second-degree AV block (referred to as the Wenckebach phenomenon or Mobitz type I block) is caused by a conduction defect within the AV node itself (Figure 4.4). On the ECG there is progressive lengthening of the PR interval on successive cardiac cycles until eventually a P wave is not conducted ("dropped"). This results in an irregular rhythm with "grouped beating," usually in pairs or triplets, but occasionally larger groups. Progressive lengthening of the PR intervals occurs because each successive atrial impulse arrives earlier and earlier in the refractory period of the AV node, and therefore takes longer and longer to conduct to the ventricle. Another feature is a progressive shortening of the RR interval in the grouped beats preceding the dropped beat. Type I second-degree AV block can occur following inferior wall myocardial infarction, and occasionally requires

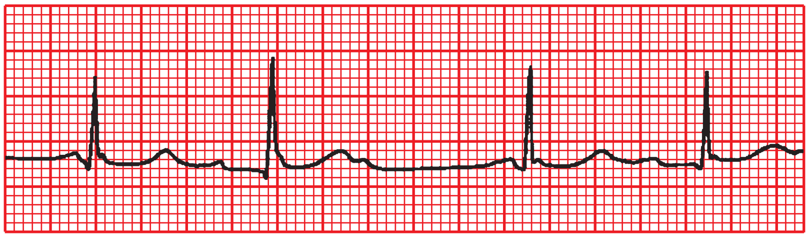


Figure 4.4
Second-degree AV block, Mobitz type I. From Da Costa D, Brady WJ, Edhouse J. Bradycardias and atrioventricular conduction block. *Br Med J* 2002;324(7336):535–8. Printed with permission.

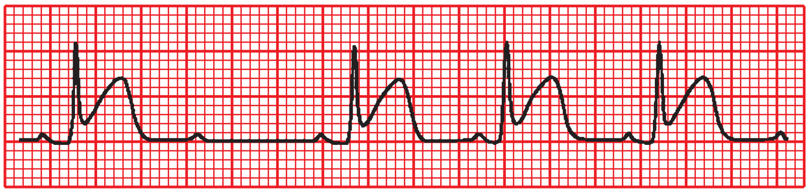


Figure 4.5
Second-degree AV block, Mobitz type II. From Da Costa D, Brady WJ, Edhouse J. Bradycardias and atrioventricular conduction block. *Br Med J* 2002;324(7336):535–8. Printed with permission.

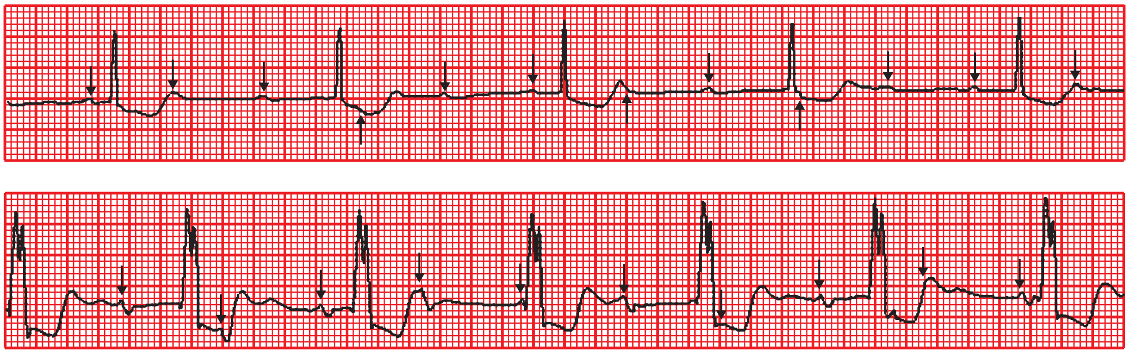


Figure 4.6
Third-degree AV block. From Da Costa D, Brady WJ, Edhouse J. Bradycardias and atrioventricular conduction block. *Br Med* J 2002;324(7336):535–8. Printed with permission.

temporary pacing in this setting. In the majority of cases, however, it is asymptomatic and requires no treatment.

Type II second-degree

Type II second-degree AV block (or Mobitz type II block) suggests a conduction block below the level of the AV node (Figure 4.5). This finding is much more ominous than type I block, as there is a significant risk of progression to complete heart block. It is caused by degenerative disease of the conduction system, termed *Lev* or *Lenegre disease*. On the ECG there is preservation of a constant PR interval on conducted beats with sudden loss of P wave conduction. There is often a concomitant bundle branch block or baseline first-degree AV block reflecting underlying conduction system disease. Patients with this form of AV block can present with symptomatic bradydysrhythmias or syncope. As parasympathetic innervation is absent below the level of the AV node, atropine is not effective in treating bradycardia associated with this type of conduction block. Permanent pacemaker placement is indicated.

2:1 block

A third type of AV block exists that cannot be definitively classified as type I or type II. It is known as *2:1 block* and is characterized by two P waves for every QRS complex. The location of the block cannot be determined with certainty based on the ECG alone, as it may represent a 2:1 Mobitz type I block or high-grade conduction system disease. This type of conduction block can occur with digoxin toxicity or AV nodal ischemia. Further invasive electrophysiologic (EP) testing involving measurement of H–V conduction times is necessary to clarify the location of the block and determine further treatment.

Third-degree atrioventricular block

Third-degree AV block occurs when there is absolutely no conduction of atrial impulses to the ventricle (Figure 4.6). Atrial and ventricular impulses may be present, but each occur independent of the other. This phenomenon is also termed *AV dissociation*. With third-degree AV block, a secondary pacemaker below the AV node assumes control and produces an escape rhythm. This escape pacemaker can originate from low in the AV node or from the His–Purkinje system. This is usually evident from the width of the QRS complex. On the ECG, there are visible P waves with a constant PP interval that continuously march through the strip. In addition, there are visible QRS complexes with a constant RR interval that also march through. As there is AV dissociation, and the atria and ventricles beat independent of each other, the PR interval is variable. In some instances, it may be difficult to identify AV dissociation, as the atrial and ventricular rates may be similar (termed *isorhythmic AV dissociation*), and longer rhythm strips may be necessary.

Management of third-degree AV block depends on the patient's clinical status. Drugs that can cause AV nodal block should be reversed. If there is evidence of significant hemodynamic compromise, transcutaneous or transvenous pacemaker placement is indicated. Unless a clearly reversible cause of third-degree block is present, most patients will require permanent pacemaker placement. With all forms of AV block, the patient should be questioned regarding risk factors for Lyme disease, myocarditis, endocarditis or lupus erythematosus, as these systemic illnesses can contribute to disease of the cardiac conduction system.

Management of tachydysrhythmias

General management

As with bradydysrhythmias, assessment and stabilization of the airway, breathing and circulation should occur rapidly. IV access should be obtained, the patient should be placed immediately on a cardiac monitor, and a 12-lead ECG should be performed and reviewed. Stat serum electrolytes should be ordered as dictated.

First, assess for the presence of serious signs or symptoms due to the tachydysrhythmia. These include hypotension, impaired tissue perfusion, chest pain, hypoxemia, other signs of worsening myocardial ischemia, or altered sensorium. If there are serious signs and symptoms, treatment should be initiated immediately, as determined by current ACLS guidelines (Figure 4.7).

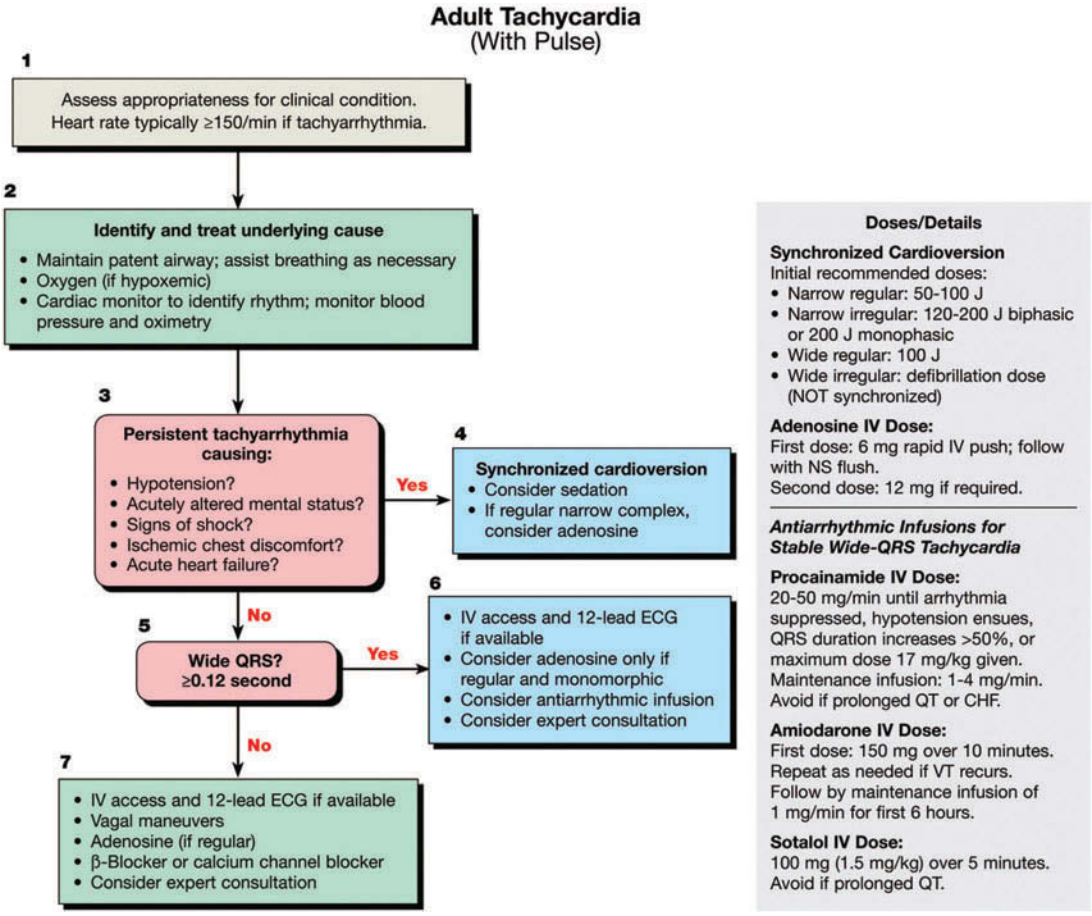

Adult Tachycardia
(With Pulse)
1
Assess appropriateness for clinical condition.
Heart rate typically ≥150/min if tachyarrhythmia.
2
Identify and treat underlying cause
• Maintain patent airway; assist breathing as necessary
• Oxygen (if hypoxemic)
• Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
3
Persistent tachyarrhythmia causing:
• Hypotension?
• Acutely altered mental status?
• Signs of shock?
• Ischemic chest discomfort?
• Acute heart failure?
Yes
4
Synchronized cardioversion
• Consider sedation
• If regular narrow complex, consider adenosine
No
5
Wide QRS? ≥0.12 second
Yes
6
• IV access and 12-lead ECG if available
• Consider adenosine only if regular and monomorphic
• Consider antiarrhythmic infusion
• Consider expert consultation
No
7
• IV access and 12-lead ECG if available
• Vagal maneuvers
• Adenosine (if regular)
• β-Blocker or calcium channel blocker
• Consider expert consultation
Doses/Details
Synchronized Cardioversion
Initial recommended doses:
• Narrow regular: 50-100 J
• Narrow irregular: 120-200 J biphasic or 200 J monophasic
• Wide regular: 100 J
• Wide irregular: defibrillation dose (NOT synchronized)
Adenosine IV Dose:
First dose: 6 mg rapid IV push; follow with NS flush.
Second dose: 12 mg if required.
Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia
Procainamide IV Dose:
20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given.
Maintenance infusion: 1-4 mg/min.
Avoid if prolonged QT or CHF.
Amiodarone IV Dose:
First dose: 150 mg over 10 minutes.
Repeat as needed if VT recurs.
Follow by maintenance infusion of 1 mg/min for first 6 hours.
Sotalol IV Dose:
100 mg (1.5 mg/kg) over 5 minutes.
Avoid if prolonged QT.

Figure 4.7
Tachycardias: overview algorithm. bpm: beats per minute; WPW: Wolff–Parkinson–White syndrome; CHF: congestive heart failure; SVT: supraventricular tachycardia; VT: ventricular tachycardia. Reproduced with permission, ACLS Provider Manual, © 2010 American Heart Association.

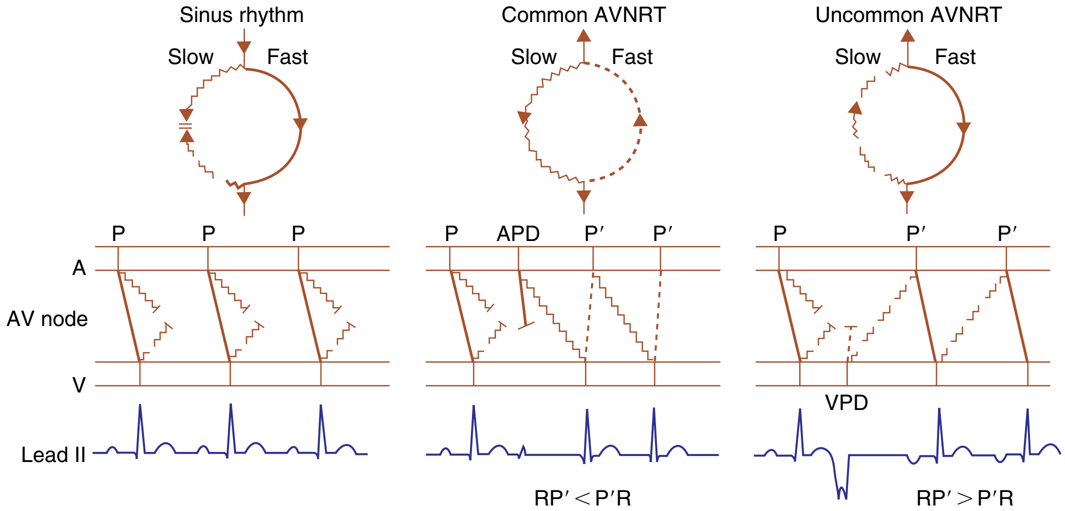


Figure 4.8
Ladder diagrams showing normal sinus conduction pattern through AV node, conduction pattern with slow–fast atrioventricular nodal reentrant tachycardia (AVNRT), and conduction pattern with fast–slow AVNRT. Corresponding ECG tracings are shown below. Each panel shows the AV node (top), a Lewis diagram (middle), and a surface ECG lead (bottom). Solid lines indicate anterograde AV nodal conduction, and broken lines retrograde conduction; straight lines indicate conduction over the fast pathway, and wavy lines conduction over the slow pathway. P denotes sinus P wave. P′, atrial echoes resulting from AV nodal reentry; APD, atrial premature depolarization; VPD, ventricular premature depolarization, and R, R waves. During sinus rhythm the presence of the slow pathway is concealed because the impulse traveling over the fast pathway turns around after traversing the AV node and retrogradely penetrates the slow pathway, colliding with the oncoming impulse moving anterogradely over the slow pathway. Note the simultaneous registration of P′ waves and QRS complexes during common AVNRT, with RP′ < P′R. Retrograde P′ waves result in the appearance of pseudo waves in the inferior ECG leads. During uncommon AVNRT, inverted P′ waves are visible, with RP′ < P′R. Diagram from Ganz LI, Friedman PL. Supraventricular tachycardia. *N Engl J Med* 1995;332(3):162–73. Printed with permission.

Management of specific tachydysrhythmias

Tachydysrhythmias can be best understood by grouping them broadly into two categories: *narrow-complex tachydysrhythmias* (defined as those with a QRS duration less than 120 milliseconds) and *wide-complex tachydysrhythmias* (QRS duration greater than 120 milliseconds). Narrow-complex tachydysrhythmias are best understood when grouped into those that are *regular*, with a relatively constant RR interval, and those that are *irregular*, with a highly variable RR interval. The regular narrow-complex tachydysrhythmias include sinus tachycardia, paroxysmal atrial tachycardia (PAT), AV nodal reentrant tachycardia (AVNRT), AV reentrant tachycardia (AVRT) and non-paroxysmal junctional tachycardia. Irregular narrow-complex tachycardias are those in which the RR interval is irregular. These include atrial fibrillation, atrial flutter and multi-focal atrial tachycardia (MAT). Atrial flutter can present with either an irregular or regular ventricular response rate (or both).

Sinus tachycardia

In sinus tachycardia, a P wave precedes each QRS complex with a relatively uniform morphology and constant PR interval. The heart rate usually ranges from 100-160 beats per minute. Small variations in PR interval may be present, related to physiologic sinus dysrhythmia.

As a general rule, sinus tachycardia is not a disease in itself; rather, it is a response to an extracardiac stimulus. As such, the rate itself does not require treatment; rather, the underlying cause should be addressed. An important exception to this rule is sinus tachycardia that occurs in the setting of acute myocardial ischemia. In this setting, reducing the heart rate with beta-blockade is indicated, in order to reduce myocardial oxygen demand and improve mortality. Systemic causes of sinus tachycardia include pain, hypovolemia, anemia, fever, hypoxemia, anxiety, sympathomimetic drugs, pregnancy and pulmonary embolism.

Sinus tachycardia can be mistaken for other causes of regular narrow-complex tachycardia, especially in children and young adults. Sinus tachycardia that is near 150 beats per minute should be re-examined closely to ensure that it is not atrial flutter with 2:1 block.

Paroxysmal atrial tachycardia (PAT)

PAT is associated with a reentrant ectopic atrial focus distinct from the sinus node. It is characterized by a heart rate of 100-160 beats per minute, having a different P-wave morphology from the patient's normal P wave. There may be 1:1 conduction or variable degrees of AV block present.

No specific therapy is indicated for PAT. It occurs in association with underlying electrolyte disturbances, drug toxicity, hypoxemia and fever. Digoxin toxicity should be investigated in any patient presenting with PAT with 2:1 block or complete AV block, as these are classic dysrhythmias of digoxin toxicity.

Atrioventricular nodal reentrant tachycardia (AVNRT)

AVNRT is a reentrant tachydysrhythmia that involves a micro-reentrant circuit within the AV node (Figure 4.8). There are typically two anatomic pathways for transit of atrial impulses through the AV node, a fast pathway (through which sinus impulses normally travel) and a slow pathway (which is typically blocked due to a long inherent refractory period). AVNRT is triggered when a premature atrial impulse passes through one of the pathways then travels retrograde up the other pathway, causing depolarization of the atrium. The impulse returns to the AV node and the cycle repeats.

In the majority of cases, the impulse travels down the fast pathway and up the slow pathway ("fast–slow" AVNRT), whereas in the remainder of cases, the impulse travels down the slow pathway and up the fast pathway ("slow–fast" AVNRT). On the ECG, a regular narrow-complex tachycardia is present. Due to the timing of atrial depolarization, the P waves are usually buried within the QRS complexes and are therefore not visible. With the slow–fast AVNRT, inverted P waves may be present before the QRS complex.

Treatment of AVNRT is predicated on temporarily interrupting the reentrant circuit, or converting the unidirectional block to a bidirectional block. This can be accomplished through vagal maneuvers or through drugs that prolong the AV nodal refractory period, such as adenosine, beta-blockers or calcium channel blockers. AVNRT may also be corrected through radiofrequency catheter ablation of the slow pathway.

Atrioventricular reentrant tachycardia (AVRT)

AVRT involves a macro-reentrant circuit including the AV node and a coexistent accessory pathway of conduction from the atria to the ventricles (Figure 4.9). In 90% of cases, the impulse starts in the atria and travels antegrade down the AV node, then retrograde up the accessory pathway (referred to as *orthodromic* AVRT) before depolarizing the atrium again. In the remaining 10% of cases, the impulse travels antegrade down the accessory pathway, then retrograde through the His–Purkinje system and the AV node (referred to as *antidromic* AVRT) before depolarizing the atria. On the ECG, orthodromic AVRT appears as a regular narrow-complex tachycardia with inverted retrograde P waves appearing after the QRS complex. Antidromic AVRT, however, appears as a wide-complex tachycardia given the antegrade conduction down the accessory pathway. Retrograde P waves are sometimes visible, so this dysrhythmia may be

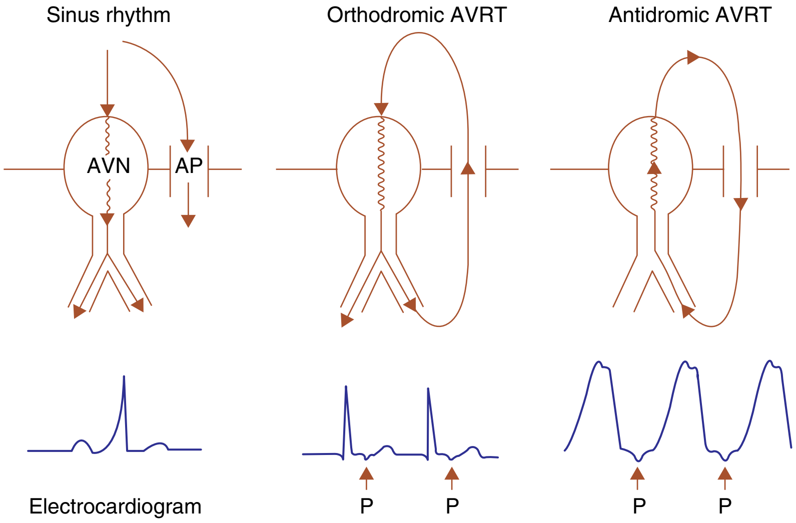


Figure 4.9
Ladder diagrams showing conduction pattern with sinus rhythm and Wolff–Parkinson–White syndrome (WPW), orthodromic and antidromic AVRT (atrioventricular reentrant tachycardia). Corresponding ECG tracings are shown below. During sinus rhythm, the slurred initial portion of the QRS delta wave is due to early activation of part of the ventricles through rapid anterograde conduction over the AP (accessory pathway). During orthodromic AVT (atrioventricular tachycardia), no delta wave is seen because all anterograde conduction is over the AV node (AVN) and through the normal His–Purkinje system. Retrograde P waves are visible shortly after each QRS. During antidromic AVT, there is maximal pre-excitation with wide, bizarre QRS complexes, because ventricular activation results entirely from anterograde conduction over the AP. Diagram from Ganz LI, Friedman PL. Supraventricular tachycardia. *N Engl J Med* 1995;332(3):162–73. Printed with permission.

mistaken for VT. AVRT can also be treated with adenosine or calcium channel blockers, which block conduction through the AV node and break the macro-reentrant circuit. Extreme caution should be used if calcium channel blockers are given to individuals with wide-complex tachycardias.

Non-paroxysmal junctional tachycardia

Junctional tachycardia occurs when there is increased automaticity of the AV node and a coexistent AV block. This results in a narrow- or wide-complex tachycardia, depending on where in the AV node the impulse originates. It can occur in the setting of digoxin toxicity, inferior myocardial infarction or acute rheumatic fever. Treatment is supportive. In a patient with chronic atrial fibrillation on digoxin therapy, the finding of a *regular* ventricular response rate despite underlying atrial fibrillation should raise the suspicion of digoxin toxicity causing complete AV block with a junctional escape pacemaker.

Atrial fibrillation

Atrial fibrillation is characterized by chaotic, disorganized depolarization of the atria with multiple impulses from the atrial tissue (Figure 4.10). Mechanically, there is no effective contraction of the atria, only a quivering of the atrial muscle. The atrial impulses travel to the AV node, where the majority are blocked and the remainder are conducted to the ventricles. This produces a heart rate from 100-180 beats per minute in patients with a healthy AV node. On the ECG, the hallmark is the absence of definitive atrial activity with either coarse or fine atrial fibrillatory waves. The ventricular response rate is almost always irregular.

Atrial fibrillation usually occurs in the setting of underlying heart disease, with systemic hypertension being the most common coexistent condition. Other associated conditions include valvular heart disease (especially mitral stenosis) and ischemic heart disease. It can be triggered by extracardiac conditions as well, including thyrotoxicosis, underlying infection or pulmonary embolism. The immediate hemodynamic consequence of atrial fibrillation is the loss of the atrial contribution (called the “atrial kick”) to diastolic filling of the LV. This should not significantly affect individuals with a normal heart, as diastolic filling in a normal ventricle results predominantly from relaxation of the ventricular myocardium. However, some patients with systolic or diastolic CHF depend on the atrial kick for a large part of diastolic filling. In these patients, atrial fibrillation can result in hypotension or pulmonary edema, especially if there is a rapid ventricular response that limits time for passive diastolic ventricular filling.

In clinically stable patients, the treatment of atrial fibrillation is predicated on slowing the ventricular response rate, which allows more time for diastolic filling. This can be accomplished with beta-blockers, calcium channel blockers or digoxin. However, if atrial fibrillation with pre-excitation is suspected, these agents are contraindicated. In such cases, they can accentuate conduction through the accessory pathway by prolonging the AV nodal refractory period. In clinically unstable patients manifesting hypotension, worsening cardiac ischemia, acute pulmonary edema or alteration in sensorium, the treatment of choice is immediate synchronized direct current (DC) cardioversion.

Due to the disorganized atrial contraction associated with this dysrhythmia, atrial fibrillation predisposes individuals to thrombus formation in the left atrial appendage. Accordingly, atrial fibrillation carries an increased risk of thromboembolic stroke. The risk is greatest during the first 48 hours following DC cardioversion. Anticoagulation with adjusted-dose warfarin can reduce the risk of thromboembolism. Stable patients should be anticoagulated for at least 3 weeks before undergoing elective DC cardioversion. If atrial fibrillation has been present for less than 48 hours, DC cardioversion or chemical cardioversion (with agents such as amiodarone, procainamide or ibutilide) can be performed without anticoagulation, provided that post-cardioversion anticoagulation is given. An alternative approach is to screen patients for thrombus with transesophageal echocardiography (TEE). If the TEE shows no evidence of thrombus, cardioversion can be performed without pre-procedural anticoagulation, but post-cardioversion anticoagulation should still be administered for 3 weeks if atrial fibrillation has been present for more than 48 hours.

All patients with atrial fibrillation should be referred for elective echocardiography to evaluate their left atrial size and LV systolic function, and should have a TSH level drawn to exclude underlying thyrotoxicosis. The decision to place a patient on long-term anticoagulation for atrial fibrillation is made based on the patient’s risk factors for stroke, which can be determined using the CHADS_2 scoring system. One point is assigned for a

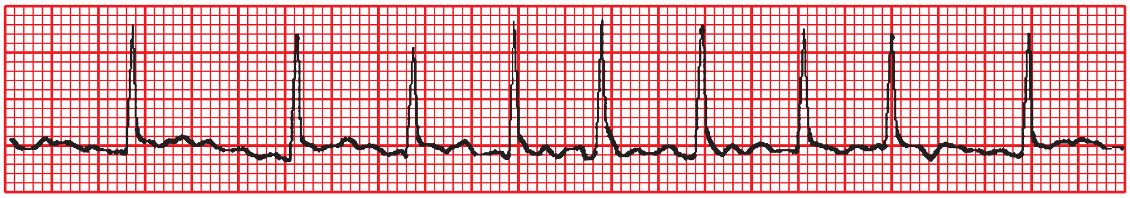


Figure 4.10
Atrial fibrillation. From Edhouse J, Morris F. ABC of clinical electrocardiography: broad complex tachycardia – Part II. *Br Med J* 2002;324(7340):776–9. Printed with permission.

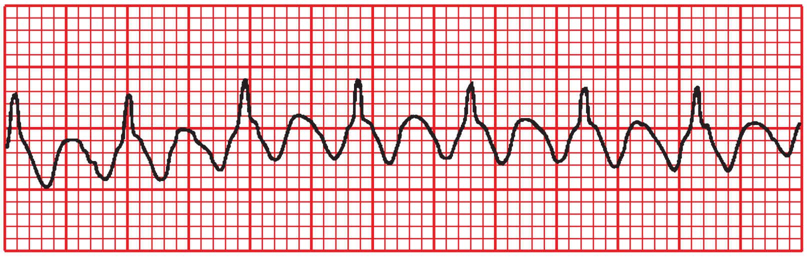


Figure 4.11
Atrial flutter. From Goodacre S, Irons R. ABC of clinical electrocardiography: atrial dysrhythmias. *Br Med J* 2002;324(7337):594–7. Printed with permission.

history of congestive heart failure (C), one point for a history of hypertension (H), one point for age greater than 75 years (A), one point for diabetes mellitus (D), and two points for a prior stroke or TIA (S). Patients with a total $CHADS_2$ score of 2 or greater have the greatest benefit from anticoagulation and should be treated with warfarin. Any decision regarding whether to place an atrial fibrillation patient on anticoagulation should be made by the patient's primary care physician and cardiologist after a detailed discussion with the patient.

Atrial flutter

Atrial flutter is characterized by a macro-reentrant dysrhythmia involving the atria, with "flutter waves" being generated at 280-320 beats per minute (Figure 4.11). It typically presents with 2:1 block and can be mistaken for sinus tachycardia. It can also present with 4:1 or variable AV block. On the ECG, flutter waves are best seen in lead II as an inverted sawtooth pattern, but may be concealed within T waves or QRS complexes.

Atrial flutter is commonly associated with underlying heart disease, such as ischemic heart disease or dilated cardiomyopathy. It is considered to have the same pathologic spectrum as atrial fibrillation, and patients with atrial flutter often have concomitant atrial fibrillation. Less commonly, it is associated with myocarditis, blunt chest trauma, or pulmonary embolism.

The treatment of atrial flutter in stable patients is rate control with a beta-blocker or calcium channel blocker. In unstable patients or patients with refractory atrial flutter, synchronized DC cardioversion with 50 joules of energy often converts atrial flutter to sinus rhythm. Ibutilide, amiodarone or procainamide can also be used to convert atrial flutter to sinus rhythm. Ibutilide should be used with caution in patients with structural heart disease or hypomagnesemia, as there is a higher risk of torsades de pointes in these patients. Atrial flutter carries with it a lower risk of thromboembolism than atrial fibrillation, but anticoagulation should be considered in patients with coexistent atrial fibrillation, patients greater than 70 years of age, patients with prior thromboembolism or patients with structurally abnormal hearts based on their $CHADS_2$ score. Atrial flutter is curable through radiofrequency catheter ablation, so these patients should be referred to a cardiologist for further evaluation.

Multi-focal atrial tachycardia (MAT)

MAT occurs when there are numerous ectopic atrial foci that simultaneously depolarize, producing at least three different P-wave morphologies, a narrow QRS complex (unless a coexistent bundle branch block is present), variable PR intervals, and a heart rate between 100 and 180 beats per minute (Figure 4.12). It is commonly associated with chronic lung disease and can be a manifestation of theophylline toxicity. Fortunately, it is seldom life-threatening. Treatment should be directed primarily at the underlying chronic lung disease, although judicious use of calcium channel blockers may provide symptomatic relief through rate control. Electrical cardioversion is not effective given the numerous sites of atrial ectopy present.

Ventricular tachycardia (VT)

VT is defined as three or more consecutive QRS complexes originating from the ventricles and occurring at a rapid rate. It may develop in a sporadic, intermittent fashion that interrupts the patient's underlying sinus rhythm (non-sustained VT), or as a consistent, uninterrupted wide-complex rhythm (sustained VT). It is typically regular or only slightly irregular. VT is almost always associated with underlying structural heart disease, and is therefore more common in older patients. It can have a single reentrant focus as the nidus for the dysrhythmia (*monomorphic* VT) or multiple reentrant foci (*polymorphic* VT), especially in the setting of ischemia (Figure 4.13). The most common underlying causes of VT are chronic ischemic heart disease and acute myocardial infarction. VT is important to recognize and treat as it has the potential to degenerate into ventricular fibrillation. All patients with VT require an aggressive work-up for possible cardiac ischemia and admission to a coronary care unit.

Perhaps the most challenging aspect of cardiac rhythm analysis is the differentiation of VT from supraventricular tachycardia (SVT) with aberrant conduction. Although no set of criteria will absolutely differentiate the two, several criteria have been developed to assist clinicians. There are several characteristics on the 12-lead ECG that strongly suggest the diagnosis of VT rather than another cause of wide-complex tachycardia. These include the following:

1. *QRS-complex duration greater than 140 milliseconds*: If the QRS complex is greater than 140 milliseconds, this strongly suggests that the rhythm is ventricular in

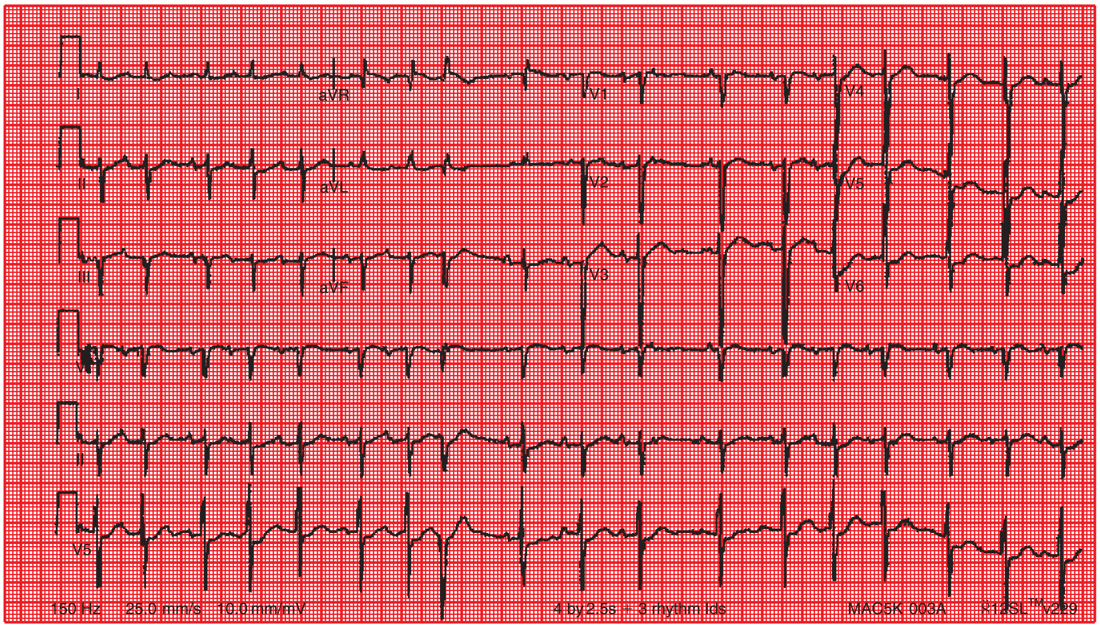


Figure 4.12
Multifocal atrial tachycardia. Note three different P wave morphologies. From Pollack ML, Brady WJ, Chan TC. Electrocardiographic manifestations: narrow QRS complex tachycardias. *J Emerg Med* 2003;24(1):35–43. Printed with permission.

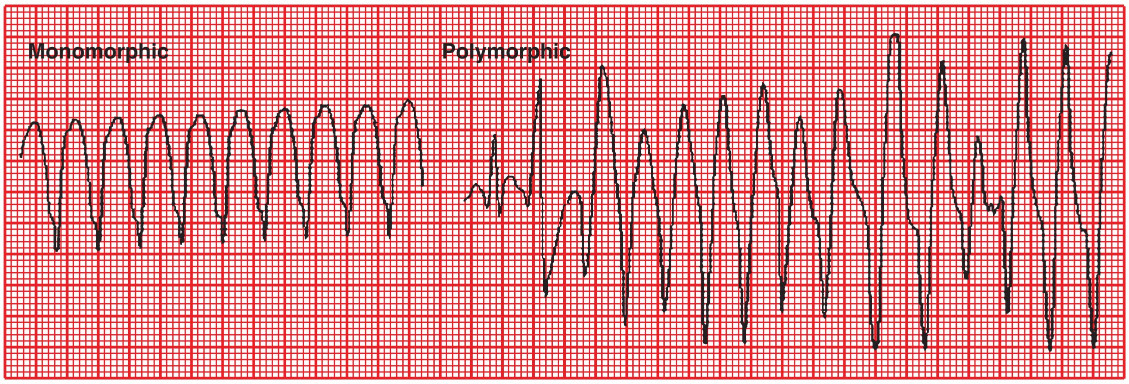


Figure 4.13
Monomorphic and polymorphic ventricular tachycardia. From Edhouse J, Morris F. Broad complex tachycardia – Part I. *Br Med J* 2002;324(7339):719–22. Printed with permission.

origin. An important exception to this rule would be a patient with a baseline bundle branch block in whom the baseline QRS duration for normal sinus beats is 140 milliseconds or greater. This is a rare scenario, however.

2. *Precordial QRS concordance*: When the initial deflection in all of the ventricular complexes from V1 through V6 are either positive or negative, this strongly suggests VT. This is very specific for VT but not very sensitive. Negative precordial QRS concordance suggests the origin of the tachycardia is the posterior wall of the LV and always connotes VT. Positive precordial QRS concordance suggests an origin from the anterior wall of the LV and may connote VT.
3. *Presence of AV dissociation*: Given that VT is caused by an independent pacemaker within the ventricle, there is no relation of the atrial rhythm to the faster ventricular rhythm. Therefore, P waves and unrelated wide QRS complexes can often be seen in the same rhythm strip. Clinically, intermittent cannon A waves may be present in the patient's jugular venous pulse; these represent right atrial contraction against a closed tricuspid valve. This clinical finding is a hallmark of AV dissociation.
4. *Presence of capture beats and/or fusion beats*: These beats represent interruptions of the underlying ventricular rhythm by atrial impulses that depolarize the ventricle, and strongly suggest VT. A *capture beat* (Figure 4.14) occurs when a P wave arrives before a ventricular impulse and results in a normal-appearing narrow-complex QRS in the midst of a group of wide-complex ventricular beats. The P wave temporarily "captures" the ventricle, but the underlying wide-complex rhythm

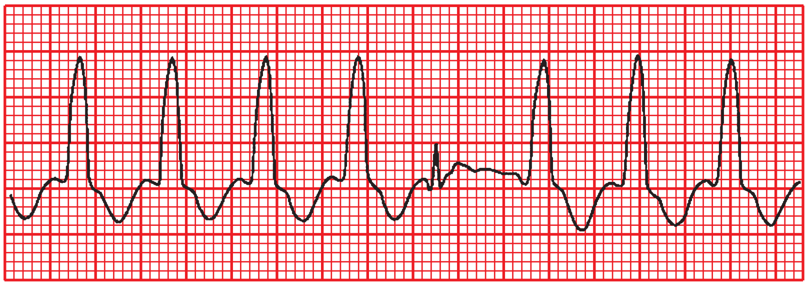


Figure 4.14
Capture beat. From Edhouse J, Morris F. Broad complex tachycardia – Part I. *Br Med J* 2002;324(7339):719–22. Printed with permission.

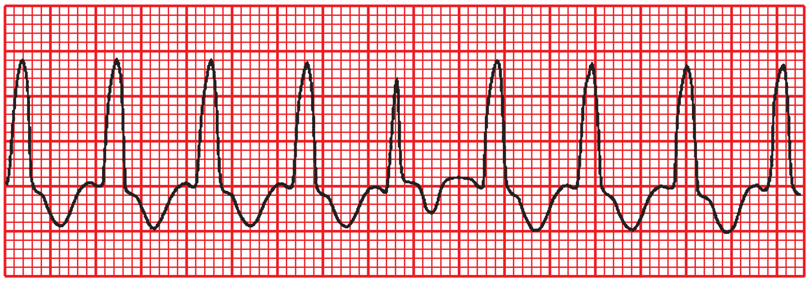


Figure 4.15
Fusion beat. From Edhouse J, Morris F. Broad complex tachycardia – Part I. *Br Med J* 2002;324(7339):719–22. Printed with permission.

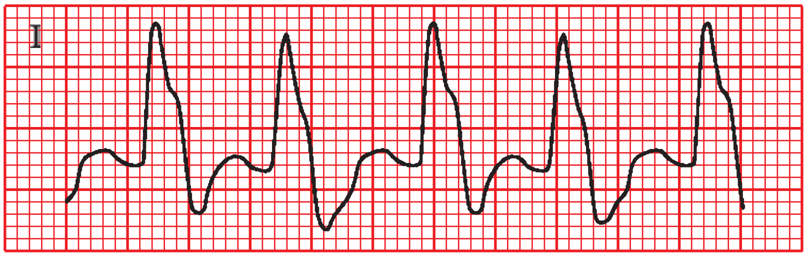


Figure 4.16
Supraventricular tachycardia (SVT) with aberrant conduction. From Edhouse J, Morris F. ABC of clinical electrocardiography: broad complex tachycardia – Part II. *Br Med J* 2002;324(7340):776–9. Printed with permission.

eventually takes over. A *fusion beat* (Figure 4.15) occurs when a P wave arrives at the same time as the ventricular impulse. The result is a QRS complex that is a hybrid between the normal narrow-complex and the wide-complex QRS of the ventricular rhythm.

Neither the rate alone nor the clinical scenario determines whether the rhythm is VT. However, a useful general principle is to treat any wide-complex QRS rhythm as VT until proven otherwise, especially if the patient is clinically unstable, has known structural heart disease or has had a previous MI.

Treatment of VT depends on the stability of the patient. Unstable patients require immediate DC cardioversion. Pulseless VT is treated as ventricular fibrillation, with immediate defibrillation. Stable patients can be treated with IV antidysrhythmic medications such as amiodarone, procainamide or lidocaine. Serum electrolytes should be drawn in all patients, and hypokalemia and hypomagnesemia should be corrected if present.

Supraventricular tachycardia (SVT) with aberrant conduction

Any of the previously discussed narrow-complex tachydysrhythmias can be accompanied by aberrant conduction (Figure 4.16). This can be either a left or right bundle branch block pattern. The following criteria suggest a supraventricular rhythm with aberrancy rather than VT:

1. A bundle branch morphology identical to that of the previous 12-lead ECG
2. An ectopic P wave that precedes the QRS complex
3. Variable coupling intervals between beats
4. Response to adenosine or carotid sinus massage (SVT with aberrancy will usually respond with a slowing of the heart rate and possible termination of the dysrhythmia, whereas VT typically does not)

When in doubt, it is safest and most appropriate to assume the rhythm disturbance is VT and treat accordingly. The treatment for all unstable patients is synchronized electrical cardioversion.

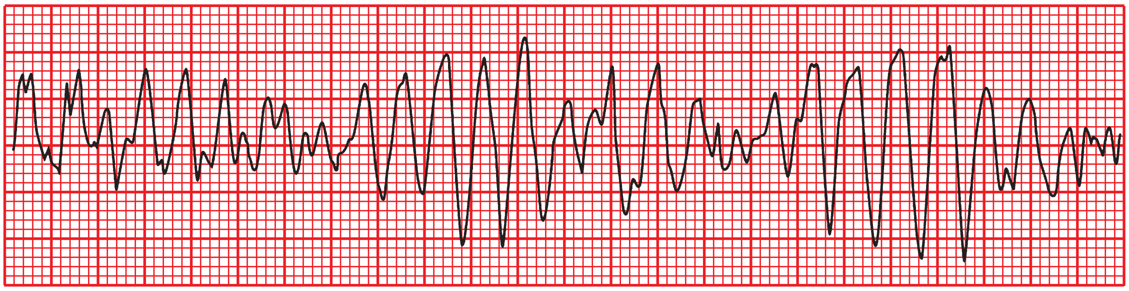


Figure 4.17
Torsades de pointes. From Edhouse J, Morris F. ABC of clinical electrocardiography: broad complex tachycardia – Part II. *Br Med J* 2002;324(7340):776–9. Printed with permission.

Torsades de pointes

Torsades de pointes ("twisting of the points") is a special type of polymorphic VT that arises in patients with preexisting prolongation of the QT interval (Figure 4.17). It is a wide-complex tachycardia with an undulating amplitude that varies above and below the baseline. Its rate varies from 180-250 beats per minute. Prolongation of the QT interval can occur as a result of the prodysrhythmic effects of numerous drugs, including quinidine, procainamide, ibutilide, amiodarone, sotalol, phenothiazines, certain antihistamines and tricyclic antidepressants. Electrolyte abnormalities such as hypomagnesemia, hypokalemia and hypocalcemia can cause prolongation of the QT interval as well.

Treatment of torsades de pointes is aimed at interrupting the ventricular rhythm and restoring sinus rhythm. As the majority of patients with this dysrhythmia are clinically unstable, DC cardioversion is the treatment of choice. Electrolyte abnormalities such as hypokalemia, hypocalcemia and hypomagnesemia should be aggressively corrected. In more stable patients, other treatment options include IV isoproterenol and overdrive pacing of the ventricle. Some clinicians empirically administer IV magnesium sulfate to treat this condition.

Atrial fibrillation with pre-excitation

This is a special case of atrial fibrillation in which conduction occurs antegrade down a pre-existing bypass tract (Figure 4.18a and 4.18b). As there is no inherent refractory period in bypass tract tissue unlike in the AV node, ventricular response rates are usually much higher. On the ECG, there is a wide-complex, irregular tachycardia with a rate ranging from 150-300 beats per minute. Delta waves (which may be seen on previous ECGs), a previous history of Wolff–Parkinson–White (WPW) syndrome, or irregular and short R-R intervals that reflect heart rates as fast as 300 beats per minute are major clues to this diagnosis.

Conventional treatment of atrial fibrillation with AV nodal blocking agents is contraindicated in the presence of a bypass tract. Instead, stable patients should receive IV procainamide, which attempts to chemically cardiovert the patient to sinus rhythm without slowing conduction through the AV node. Unstable patients should undergo synchronized electrical cardioversion. Patients who manifest this rhythm should be referred to a cardiologist for radiofrequency catheter ablation of the bypass tract to prevent recurrences.

Accelerated idioventricular rhythm (AIVR)

Accelerated idioventricular rhythm (AIVR) is a wide-complex dysrhythmia of ventricular origin that occurs at a rate of 40-100 beats per minute (Figure 4.19). It is characterized by regular, wide QRS complexes that are not preceded by P waves. It is commonly seen following acute myocardial infarction, and is known as a "reperfusion dysrhythmia." AIVR is a stable rhythm that usually produces no symptoms and therefore requires no treatment. In some instances, the ventricular pacemaker may be the only functioning pacemaker in the heart, and suppressing it with antidysrhythmics such as lidocaine can lead to asystole.

Pearls, pitfalls and myths

- Consider the etiology of the cardiac dysrhythmia, not just the rhythm itself.
- Certain medications and electrolyte abnormalities may predispose the patient to serious dysrhythmias.
- Learn to distinguish benign from malignant dysrhythmias.
- The patient's clinical (hemodynamic) stability is integral to the appropriate evaluation and management of dysrhythmias.
- Treat the patient, not the rhythm.
- Err on the side of caution, especially with regard to medication choices or rhythm interpretation.
- Serial administration of medications is generally safer than a single large bolus.
- When in doubt about the etiology of a wide-complex tachycardia, treat as ventricular tachycardia until proven otherwise.
- Before semi-elective cardioversion of atrial fibrillation, consider the risk of thromboembolic stroke and the need for anticoagulation.
- Beware of atrial fibrillation in the setting of pre-excitation, as treatment with AV nodal blockade can lead to ventricular fibrillation.

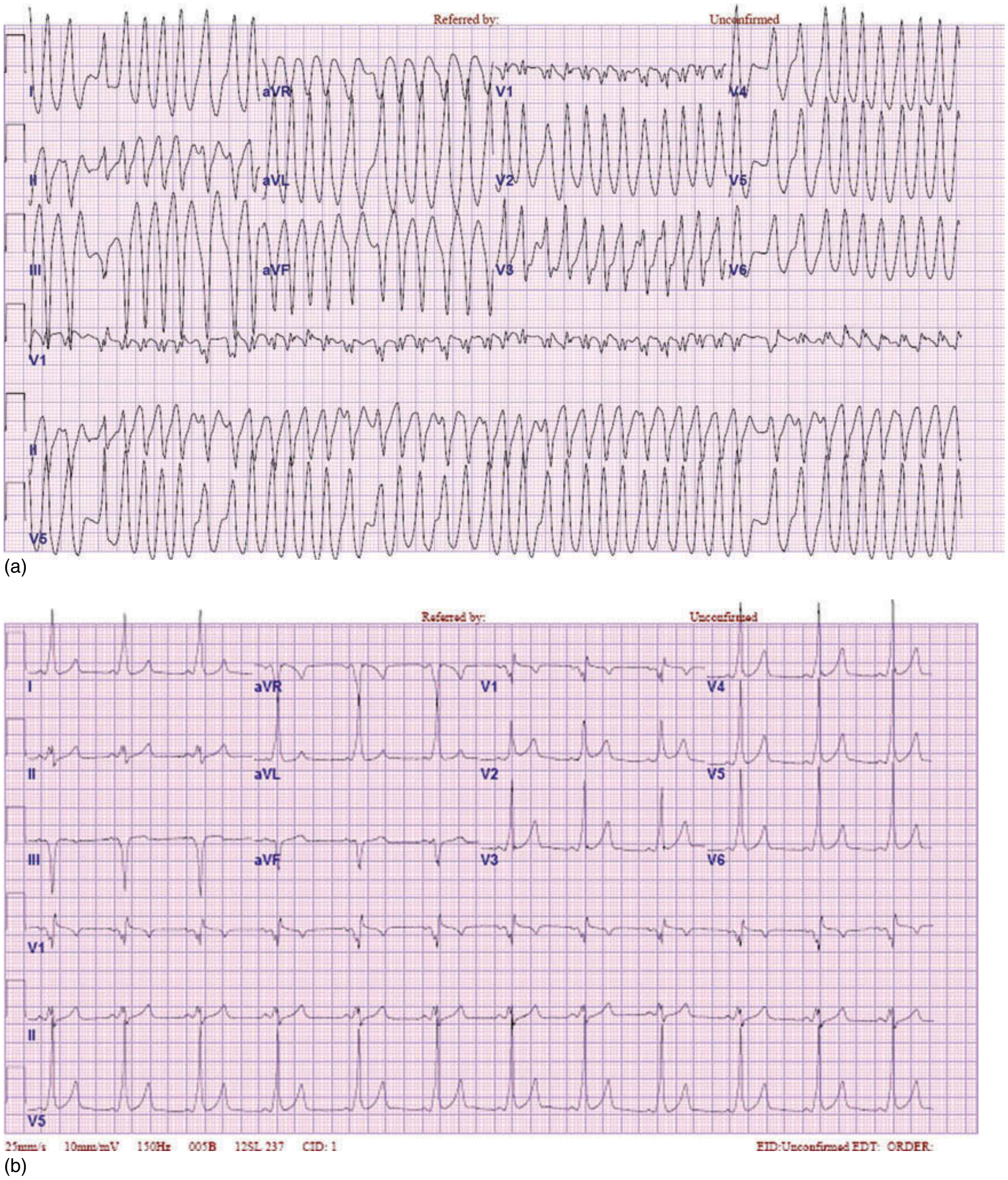

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Figure 4.18
(a) Atrial fibrillation where conduction occurs antegrade down a preexisting bypass tract, producing an irregular, wide-complex tachycardia. (b) Following cardioversion, the short P-R interval and delta waves are visible, consistent with Wolff-Parkinson-White syndrome.

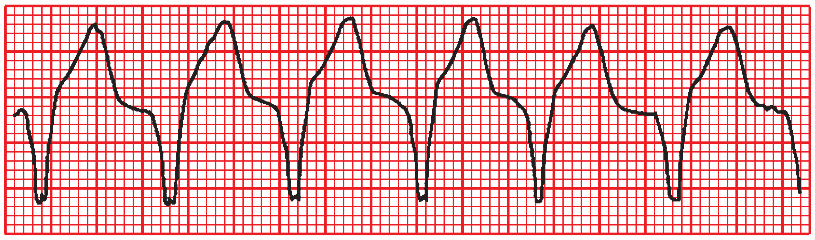

Figure 4.19
Accelerated idioventricular rhythm. From Edhouse J, Morris F. Broad complex tachycardia – Part I. *Br Med J* 2002;324(7339):719–22. Printed with permission.

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5 Severe sepsis and septic shock

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Scope of the problem

Of the 120 million patients presenting to the Emergency Department (ED) in the United States per year, over 600,000 (2.9%) carry a diagnosis of severe sepsis and septic shock. The overall hospital mortality for sepsis, severe sepsis and septic shock is 15%, 20% and 45%, respectively. Sepsis is responsible for 210,000 deaths (9%) per year in the United States. By comparison, 180,000 persons die of acute myocardial infarction and 200,000 die of lung or breast cancer annually. Many patients with severe sepsis and septic shock present to the ED, where long delays before transfer to an intensive care unit (ICU) bed may occur. Sepsis is the most expensive diagnosis among patients admitted to hospitals, accounting for over $50 billion in health care resource consumption each year. It is because of these aforementioned factors that the ED has become a focal point for sepsis diagnosis and treatment. Appropriate therapy during the first 6 hours of sepsis management can improve outcomes in one of every six patients presenting with the disease.

Anatomic essentials

A series of pathogenic events are responsible for the transition from simple infection or sepsis to severe sepsis and septic shock. When an organism enters the body, the reaction is a systemic response that creates a systemic inflammatory response syndrome (SIRS). This reaction may be self-limited or may create a generalized systemic response (Figure 5.1). SIRS is the result of a release of pro- and anti-inflammatory mediators. There is also a release of apoptotic proteins and an activation of the coagulation cascade. These processes can lead to malignant microvascular injury and thrombosis. Diffuse endothelial disruption follows, resulting in impaired tissue oxygenation. Furthermore, there is an imbalance between oxygen delivery and oxygen consumption. When organ dysfunction accompanies this response, severe sepsis begins. Global tissue hypoxia and cytopathic (cellular) hypoxia develops, leading to multiple organ dysfunction and irreversible shock (Figure 5.2).

Physiology of systemic oxygen transport and utilization

In order to diagnosis and treat early sepsis, a basic understanding of oxygen transport physiology is required. Oxygen is delivered to the tissues as a product of cardiac output and oxygen content (which is a product of hemoglobin oxygen saturation and hemoglobin). After oxygen is extracted at the tissue level, the remainder returns to

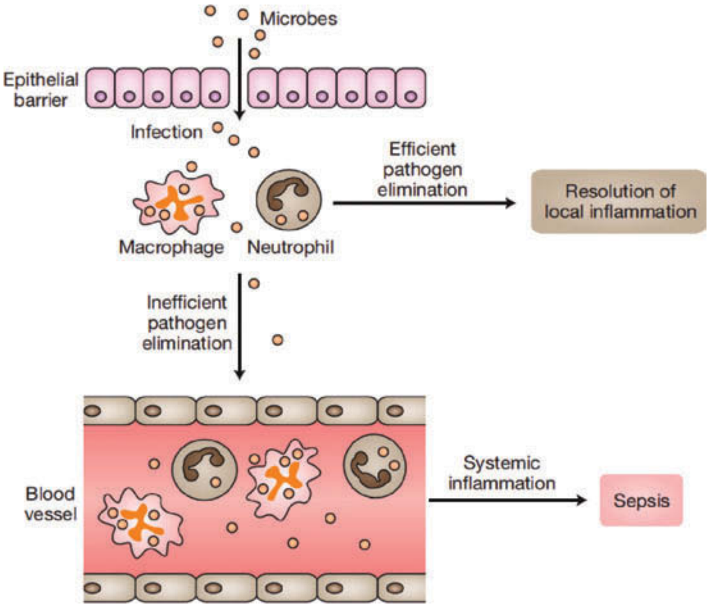

Microbes
Epithelial barrier
Infection
Efficient pathogen elimination
Resolution of local inflammation
Macrophage
Neutrophil
Inefficient pathogen elimination
Blood vessel
Systemic inflammation
Sepsis

Figure 5.1
Microbial trigger of local or systemic inflammatory response. With permission from Expert Review in Molecular Medicine, Cambridge University Press 2008.

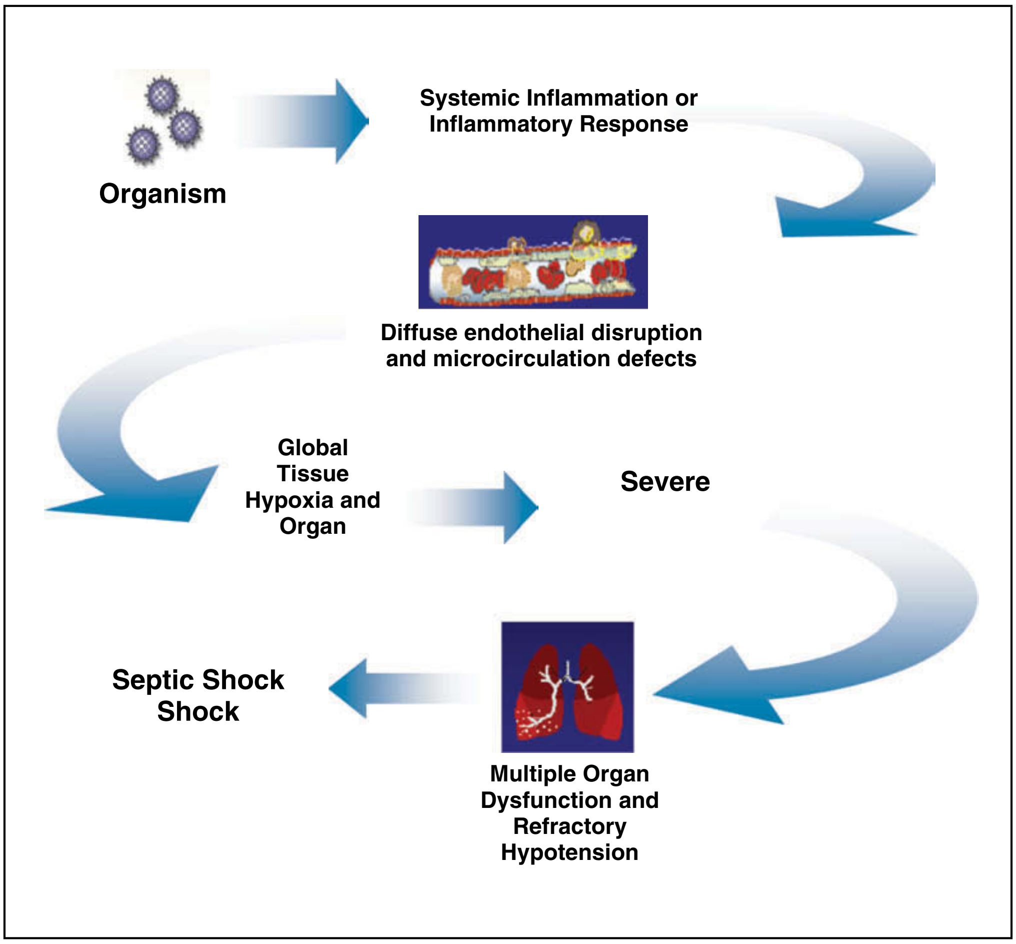

Organism
Systemic Inflammation or Inflammatory Response
Diffuse endothelial disruption and microcirculation defects
Global Tissue Hypoxia and Organ
Severe
Multiple Organ Dysfunction and Refractory Hypotension
Septic Shock Shock

Figure 5.2
Infectious cascade. From Nguyen HB, Rivers EP, Abrahamian FM, et al. Severe sepsis and septic shock: Review of the literature and emergency department management guidelines. *Ann Emerg Med 2006*;**48**: 28–54. Used with permission.

the venous circulation. The product of systemic oxygen delivery and the percentage of oxygen extracted (normally 25%) by the tissues is the systemic oxygen consumption. The balance between systemic oxygen delivery and consumption is reflected by the mixed venous hemoglobin oxygen saturation (SvO_2). Global tissue hypoxia results when there is an inability of systemic oxygen delivery to meet the oxygen requirements (i.e., consumption) of the tissues, resulting in lactic acidosis (Figures 5.3A, B).

Cardiovascular insufficiency and global tissue hypoxia

The development of cardiovascular insufficiency and resulting global tissue hypoxia is one of the most important events in the initial hours leading to morbidity and mortality. Global tissue hypoxia (or oxygen deprivation), which can occur prior to the development of hypotension, results in further endothelial activation and generalized inflammation. Global tissue hypoxia develops from multiple mechanisms that contribute to cardiovascular insufficiency (Figure 5.4). These mechanisms include (1) hypovolemia (decreased preload); (2) vasoregulatory dysfunction (hypotension); (3) myocardial depression; (4) increased metabolic demands; and (5) impaired tissue oxygen utilization resulting from microcirculatory dysfunction and cytopathic hypoxia.

History

To diagnose severe sepsis/septic shock as early as possible, it is necessary to identify historical, clinical and laboratory findings that increase the risk of infection and illness severity. Risk factors for sepsis include age <1 year or >65 years, malnutrition, hypothermia, indwelling catheter use, previous endotracheal intubation/mechanical ventilation, aspiration, chronic illness (e.g., diabetes, renal failure, hepatic failure, AIDS, alcoholism), high-risk behavior (intravenous drug use), chemotherapy and immunosuppressive drugs, surgery or invasive procedures (Figure 5.5). The elderly are particularly at high risk.

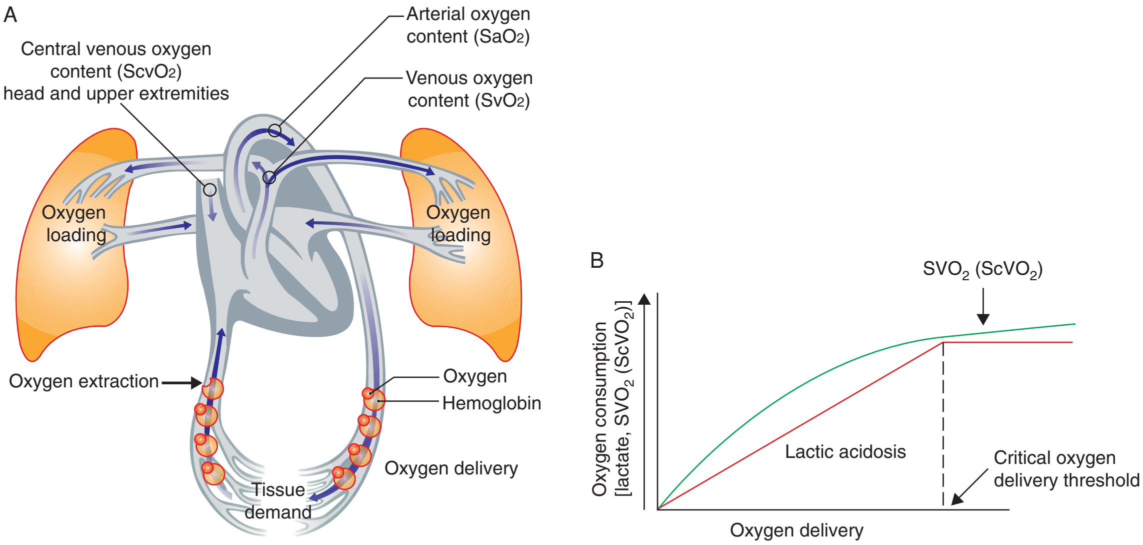

A
Central venous oxygen content (ScvO2) head and upper extremities
Arterial oxygen content (SaO2)
Venous oxygen content (SvO2)
Oxygen loading
Oxygen loading
Oxygen extraction
Oxygen
Hemoglobin
Oxygen delivery
Tissue demand
B
SVO2 (ScVO2)
Oxygen consumption [lactate, SVO2 (ScVO2)]
Lactic acidosis
Critical oxygen delivery threshold
Oxygen delivery

Figure 5.3
Physiology of systemic oxygen transport and utilization. From Rivers EP, McIntyre L, Morro DC, Rivers KK. Early and innovative interventions for severe sepsis and septic shock: Taking advantage of a window of opportunity. *CMAJ* 2005;**173**:1054–65. Used with permission.

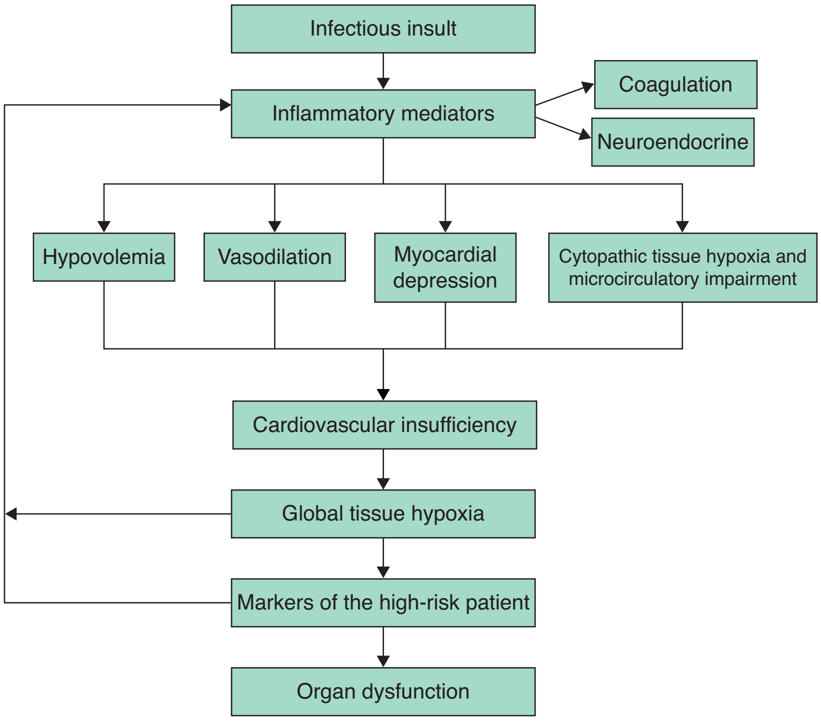

Infectious insult
Inflammatory mediators
Coagulation
Neuroendocrine
Hypovolemia
Vasodilation
Myocardial depression
Cytopathic tissue hypoxia and microcirculatory impairment
Cardiovascular insufficiency
Global tissue hypoxia
Markers of the high-risk patient
Organ dysfunction

Figure 5.4
Cardiovascular insufficiency and global tissue hypoxia. From Rivers EP, McIntyre L, Morro DC, Rivers KK. Early and innovative interventions for severe sepsis and septic shock: Taking advantage of a window of opportunity. *CMAJ* 2005;**173**:1054–65. Used with permission.

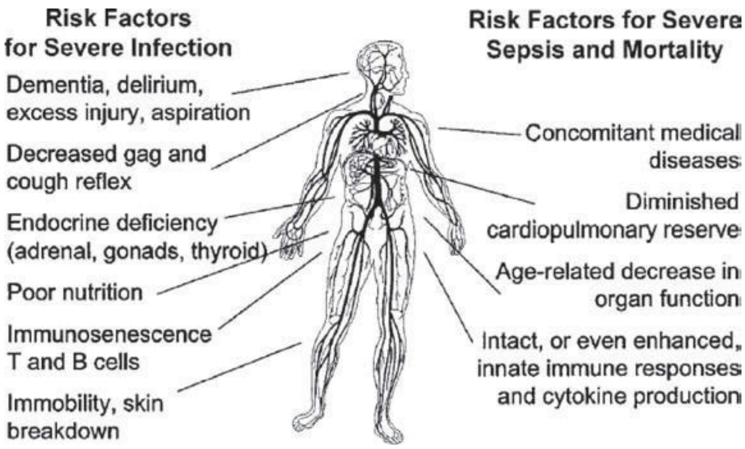

Risk Factors for Severe Infection
Dementia, delirium, excess injury, aspiration
Decreased gag and cough reflex
Endocrine deficiency (adrenal, gonads, thyroid)
Poor nutrition
Immunosenescence T and B cells
Immobility, skin breakdown
Risk Factors for Severe Sepsis and Mortality
Concomitant medical diseases
Diminished cardiopulmonary reserve
Age-related decrease in organ function
Intact, or even enhanced, innate immune responses and cytokine production

Figure 5.5
Risk factors for sepsis. From Girard TD, Opal SM, Ely EW. Insights into severe sepsis in older patients: From epidemiology to evidence-based management. *Clin Infect Dis* 2005;**40**:719–27. Used with permission.

Physical examination

Table 5.1 Physical examination

Temperature	The hallmark finding of infection is fever. Elderly patients tend to have lower temperatures than younger adults. Any oral temperature above 37.5°C or 99.0°F should be considered a fever in the elderly. Hypothermia (i.e., temperature < 36°C or 96.8°F) is associated with the presence of severe infection. Hyperthermia or hypothermia may be present.
Heart rate	Usually elevated. However, paradoxical bradycardia can be seen in patients with preexisting cardiac disease or those on beta-blockers. Increased heart rate variability is associated with poor outcomes.
SBP	May actually increase slightly when cardiac contractility increases in early shock before falling as shock advances.
DBP	Correlates with arteriolar vasoconstriction and may rise early in shock before falling when cardiovascular compensation fails.
Pulse pressure	SBP minus DBP, related to stroke volume and rigidity of the aorta. Increases early in shock and decreases before systolic pressure.
Pulsus paradoxus	The change in SBP with respiration. The rise and fall in intrathoracic pressure affects cardiac output. This can be seen in asthma, cardiac tamponade and severe cardiac decompensation.
MAP	DBP + (SBP − DBP)/3. The relationship between cardiac output and vascular resistance.
Shock index	Shock index = HR/SBP. Normal = 0.5 to 0.7. The shock index is related to left ventricular stroke work in acute circulatory failure. A persistent elevation of the shock index (>1) indicates impaired left ventricular function (as a result of blood loss and/or cardiac depression) and carries a high mortality rate.
Central nervous system	Acute delirium or brain failure, restlessness, disorientation, confusion and coma secondary to decrease in cerebral perfusion pressure (MAP falls below intracranial pressure). Patients with chronic hypertension may be symptomatic at normal blood pressures.
Skin	Pallor, pale, dusky, clammy, cyanosis, sweating, altered temperature and decreased capillary refill.
Cardiovascular	Neck vein distention or flattening, tachycardia and dysrhythmias. An S3 may result from high-output states. Decreased coronary perfusion pressures can lead to ischemia, decreased ventricular compliance and increased left ventricular diastolic pressure.
Respiratory	Tachypnea, increased minute ventilation, increased dead space, bronchospasm, hypocapnia with progression to respiratory failure and ARDS.
Splanchnic organs	Ileus, gastrointestinal bleeding, pancreatitis, acalculous cholecystitis and mesenteric ischemia can occur from low flow states.
Renal	Reduced GFR. Renal blood flow redistributes from the renal cortex toward the renal medulla, which may cause acute renal failure. Paradoxical polyuria can occur in sepsis, which may be confused with adequate hydration status.
Metabolic	Respiratory alkalosis is the first acid–base abnormality seen; as shock progresses, metabolic acidosis occurs. Hyperglycemia, hypoglycemia and hyperkalemia may occur.

ARDS: adult respiratory distress syndrome; DBP: diastolic blood pressure; GFR: glomerular filtration rate; HR: heart rate; MAP: mean arterial pressure; SBP: systolic blood pressure.

Table 5.2 Clinical sepsis definitions

Systemic Inflammatory Response Syndrome (SIRS)

The systemic inflammatory response to a wide variety of severe clinical insults, manifested by two or more of the following conditions:

1. Temperature >38°C or <36°C
2. Heart rate >90 beats/min
3. Respiratory rate >20 breaths/min or $PaCO_2$ <32 mmHg
4. White blood cell count >12,000/µL or <4,000/µL, or >10% immature (band) forms

Sepsis – The systemic inflammatory response to infection. The diagnosis of sepsis requires the presence of at least two SIRS criteria plus an infection. Signs of infection include an inflammatory response to the presence of microorganisms or the invasion of a normally sterile host tissue by those organisms.

Severe Sepsis/SIRS – Sepsis (SIRS) associated with organ dysfunction, hypoperfusion or hypotension. Hypoperfusion and perfusion abnormalities may include, but are not limited to, lactic acidosis, oliguria or an acute alteration in mental status.

Sepsis (SIRS) – Induced Hypotension – A systolic blood pressure <90 mmHg or a reduction of ≥40 mmHg from baseline in the absence of other causes for hypotension.

Septic Shock/SIRS Shock – A subset of severe sepsis with hypotension despite adequate fluid resuscitation along with the presence of perfusion abnormalities that may include, but are not limited to, lactic acidosis, oliguria or an acute alteration in mental status. Patients receiving inotropic or vasopressor agents may no longer be hypotensive by the time they manifest hypoperfusion abnormalities or organ dysfunction, yet they would still be considered to have septic (SIRS) shock.

Multiple Organ Dysfunction Syndrome (MODS) – Presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

From Bone RC, Balk RA, Cerra FB, et al. American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference: Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. *Chest* 1992;101:1644–55. Reproduced with permission.

Diagnostic testing

Table 5.3 Diagnostic adjuncts for sepsis

General variables

- Fever (core temperature >38.3°C)
- Hypothermia (core temperature <36°C)
- HR >90/min or >2 SD above the normal value for age
- Tachypnea
- Altered mental status
- Significant edema or positive fluid balance (>20 mL/kg over 24 hrs)
- Hyperglycemia (plasma glucose >120 mg/dL or 7.7 mmol/L) in the absence of diabetes

Inflammatory and hematologic variables

- Leukocytosis (WBC count >12,000 µ/L)
- Leukopenia (WBC count <4000 µ/L)
- Normal WBC count with >10% immature forms
- Döhle's bodies, toxic granulation and vacuoles
- Plasma CRP >2 SD above the normal value
- Plasma procalcitonin >2 SD above the normal value
- Hemoconcentration (dehydration)
- Thrombocytopenia
- D-dimer platelet
- PT
- PTT
- D-dimer
- Fibrin degradation products
- Fibrinogen
- CRP (if available)
- IL-6 (if available)
- Procalcitonin, Protein C

Hemodynamic variables

- Arterial hypotension (SBP <90 mmHg, MAP <70, or an SBP decrease >40 mmHg in adults or >2 SD below normal for age)
- Mixed venous oxygen saturation (SvO_2 >70%)
- Cardiac index >3.5 L/min/m^2

Organ dysfunction variables

- Arterial hypoxemia (PaO_2/FiO_2 <300)
- Acute oliguria (urine output <0.5 mL/kg/hr or 45 mmol/L for at least 2 hrs)
- Creatinine increase >0.5 mg/dL
- Coagulation abnormalities (INR >1.5 or aPTT >60 secs)
- Ileus (absent bowel sounds)
- Thrombocytopenia (platelet count <100,000/µL)
- Hyperbilirubinemia (plasma total bilirubin >4 mg/dL or 70 mmol/L)

Tissue perfusion variables

- Hyperlactatemia (>2 mmol/L)
- Decreased capillary refill or mottling
- Low bicarbonate level
- Elevated anion gap

aPTT: activated partial thromboplastin time; CRP: C-reactive protein; FiO_2: fraction of inspired Oxygen; HR: heart rate; IL-6: interleukin 6; INR: international normalized ratio; MAP: mean arterial blood pressure; PaO_2: partial pressure of Oxygen; PT: prothrombin time; PTT: partial thromboplastin time; SD: standard deviation; SBP: systolic blood pressure; SvO_2: mixed venous oxygen saturation; WBC: white blood cell.

Differential diagnosis

Patients presenting with undifferentiated sepsis may be challenging. Mental status changes, syncope, acute bronchospasm, diffuse joint and muscle pain, or abdominal pain can be general presentations or complaints. A broad differential diagnosis should be maintained, with preparation for an early and aggressive therapeutic approach.

Risk stratification

New scoring systems should be developed to accommodate the spectrum of severity from the ED to the ICU, or current systems should be recalibrated to this end. ED-specific scoring systems should consider ease of use and bedside availability, shorter time frame of data collection and comparability with existing scores. The MEDS score (Table 5.4), specifically developed for use in the ED, is designed to identify ED patients at risk for infection and categorize them based on mortality risk. In developing the MEDS score, independent predictors of mortality for ED patients were determined. These are terminal illness, tachypnea or hypoxia, septic shock, platelets <150,000/mm^3, bands >5%, age >65 years, lower respiratory infection, nursing home resident, and altered mental status. The MEDS score may prove to be useful in identifying patients who will benefit from aggressive intervention.

General treatment principles

Early hemodynamic optimization

A goal-directed hemodynamic resuscitation of severe sepsis/septic shock includes a systematic approach to restoration of systemic oxygen delivery through a manipulation of preload (volume), afterload (blood pressure), and contractility (stroke volume) in order to preserve effective tissue perfusion while avoiding excessive increases in myocardial oxygen consumption (i.e., avoiding tachycardia and maintaining coronary perfusion pressure). When this is performed within the first 6 hours of disease presentation, outcomes are improved. Specifically, patients are managed by (1) fluid resuscitation with either crystalloid or colloid to achieve a central venous pressure (CVP) goal of 8–12 mm Hg; (2) vasoactive agents to achieve a mean arterial pressure (MAP) goal of 65–90 mmHg; (3) blood transfusion to a hematocrit ≥30%; (4) inotrope therapy; and (5) intubation, sedation and paralysis as necessary to achieve a $ScvO_2$ of ≥70% as measured by intermittent or continuous central venous monitoring. Early goal-directed therapy (EGDT) should be employed as the first means of resuscitation, with simultaneous prioritization of appropriate empiric antimicrobials and source control.

Hemodynamic monitoring

Optimal titration of fluids and vasoactive therapy is performed more objectively with invasive monitoring.

Table 5.4 Mortality in Emergency Department Sepsis (MEDS) score

Variable	Odds ratio	95% CI	Points
Terminal illness (<30-day predicted mortality due to underlying illness – e.g., end-stage CA, AIDS)	6.1	(3.6–10.2)	6
Tachypnea (RR >20) or hypoxia (Pox <90%)	2.7	(1.6–4.3)	3
Septic shock (SBP <90 mmHg after fluid challenge)	2.7	(1.2–5.7)	3
Platelets <150,000/mm^3	2.5	(1.5–4.3)	3
Bands 5%	2.5	(1.5–3.5)	3
Age >65 years	2.2	(1.3–3.6)	3
Lower respiratory infection	1.9	(1.2–3.0)	2
Nursing home resident	1.9	(1.2–3.0)	2
Altered mental status	1.6	(1.0–2.5)	2

Mortality estimates

Mortality risk	Points	Mortality
Very low	0–4	0.9%
Low	5–7	2%
Moderate	8–12	7.9%
High	13–15	20%
Very high	>15	50%

MEDS score used to identify ED patients with suspected infection at increased risk of death. Mortality estimates are determined using a point system derived from the clinical variables.

AIDS: acquired immune deficiency syndrome; CA: cancer; CI: confidence interval; Pox: pulse oximetry; RR: respiratory rate; SBP: systolic blood pressure.

Table 5.5 Summary of rationale and recommendations for the ED management of an adult patient with severe sepsis or septic shock

Therapy	Significance
First 6 hours	
Cultures	Obtain appropriate cultures from all potential sources prior to antimicrobial treatment.
Timely and appropriate therapy	Administration of antimicrobials within 3 hours of ED arrival.
Source control	Early detection of the site of infection, drainage, debridement or removal of devitalized infected tissue or infected devices.
Hemodynamic assessment and risk stratification	Check lactate level. Administer minimum of 20 mL/kg of crystalloid (or colloid equivalent). For hypotension not responding to volume resuscitation, vasopressors are employed to maintain mean arterial pressure >65 mmHg.
Early goal-directed therapy	Within 6 hours of presentation, patients are managed by: (1) Fluid resuscitation to achieve a CVP goal of 8–12 mmHg (2) Vasoactive agents to achieve a MAP of 65–90 mmHg (3) Blood transfusion to a hematocrit ≥30% (4) Inotrope therapy, and then sedation, paralysis, and intubation as necessary to achieve an $ScvO_2$ of ≥70%.
Corticosteroids	Administer low-dose steroids (50 mg of hydrocortisone every 6 hours) after adequate resuscitation in patients who remain on vasopressors.
Subsequent 24 hours	
Low-tidal volume mechanical ventilation	Maintain inspiratory plateau pressure <30 cm H_2O for patients requiring mechanical ventilation. The tidal volume may be decreased to as low as 4 mL/kg if necessary.
Recombinant human activated protein C drotrecogin alfa (activated)	Consider for patients with severe sepsis or septic shock with APACHE II scores >25.
Glucose control	Maintain glucose control greater than or equal to the lower limit of normal, but <150 mg/dL.

ACTH: adrenocorticotropic hormone; APACHE: Acute Physiology and Chronic Health Evaluation; CVP: central venous pressure; MAP: mean arterial pressure; $ScvO_2$: central venous oxygen saturation.

Table 5.6 Empiric antimicrobial recommendations for adult ED patients with severe sepsis and septic shock

Sepsis Source	Recommended Antimicrobial Regimen (standard adult dosing)	Comments
Unknown source	Vancomycin[1] 1 g IV Q 12 hrs and levofloxacin[2] 750 mg IV Q 24 hrs and gentamicin[3] 7 mg/kg IV Q 24 hrs	Consider abdominal/pelvic imaging if physical examination, chest radiograph and urinalysis do not reveal an infection source.
Community-acquired pneumonia	Vancomycin[1] 1 g IV Q 12 hrs and levofloxacin[2] 750 mg IV Q 24 hrs and gentamicin[3] 7 mg/kg IV Q 24 hrs (if recent hospitalization/nursing home residence or bronchiectasis)	Consider *Pneumocystis carinii* pneumonia treatment in AIDS patients and obtain an echocardiogram to evaluate endocarditis with septic emboli in intravenous drug users.
Urinary tract infection	Piperacillin/tazobactam[4] 3.375 g IV Q 6 hrs and gentamicin[3] 7 mg/kg IV Q 24 hrs	Complicated urinary tract infections may be caused by *Enterococcus* species, *Pseudomonas aeruginosa* or *Staphylococcus aureus* (non-nitrite producers) for which gentamicin and piperacillin/tazobactam are preferred. If nitrite production or Gram stain suggests Enterobacteriaceae, levofloxacin or ceftriaxone can be substituted for gentamicin. Obtain imaging to rule out obstruction as soon as possible.
Skin and soft tissue infection/necrotizing infection	Vancomycin[1] 1 g IV Q 12 hrs and piperacillin/tazobactam[4] 3.375 g IV Q 6 hrs and clindamycin 900 mg IV Q 8 hrs	For suspected necrotizing infections, obtain surgical consultation for tissue debridement as soon as possible.
Meningitis	Vancomycin[1] 1 g IV Q 12 hrs and ceftriaxone 2 g IV Q 12 hrs (and ampicillin 2 g IV Q 4 hrs if immunocompromised or elderly) ***after*** dexamethasone 10 mg IV Q 6 hrs	If altered mental status or focal neurologic abnormalities, consider adding acyclovir (10 mg/kg IV Q 8 hrs) to treat herpes encephalitis.
Intra-abdominal/pelvic infection	Piperacillin/tazobactam[4] 3.375 g IV Q 6 hrs and gentamicin[3] 7 mg/kg IV Q 24 hrs	Obtain surgical consultation for surgical exploration, or, if indicated, imaging to identify infection focus and potential for percutaneous or open drainage.

Dosages are for approximately 70-kg adult with normal renal function.

[1] May substitute linezolid.

[2] May substitute gatifloxacin or moxifloxacin (community acquired pneumonia only).

[3] May substitute ceftazidime, cefepime, aztreonam, imipenem or meropenem.

[4] May substitute ampicillin/sulbactam, imipenem or meropenem.

Table 5.7 The stages of sepsis and therapies

	Mean arterial pressure	Central venous pressure	ScvO$_2$	Lactate	Cardiac index	Systemic vascular resistance	Treatment
Stage 1: Hypovolemia	Variable	↓	↓	↑	↓	↑	Volume
Stage 2: Compensated and vasodilatory	↓	Normal	↑	Variable	↑	↓	Vasopressors, corticosteroids
Stage 3: Myocardial suppression	Variable	↑	↓	↑	Normal or ↓	Normal or ↑	Inotropic therapy
Stage 4: Impairment of tissue O$_2$ utilization	↓	Normal	↑	↑	Variable	Variable	Vasodilators, r-APC

Central venous access allows measurement of CVP and ScvO$_2$ continuously or by intermittent venous blood gas sampling. When using vasopressor agents, intra-arterial pressure monitoring is preferred, with the femoral site being recommended over the radial artery due to a more accurate reflection of central aortic pressure. Additional tools that may be more practical in the ED are evolving.

Volume therapy

The first parameter to target in hemodynamic optimization is intravascular volume with the use of fluid therapy targeting a CVP of 8–12 mmHg. No outcome benefit has been demonstrated from using colloids compared with crystalloids with respect to mortality or hospital length of stay. However, there may be a trend to improved survival with the use of colloid (albumin) in sepsis. The volume of crystalloids required may be two to three times that required of colloids in order to restore the optimal volume. One liter of normal saline adds 275 mL to the plasma volume, whereas 1 L of 5% albumin will increase plasma volume by 500 mL. In patients with low CVP and concurrent pulmonary edema, a colloid may be combined with a crystalloid to avoid the large volume of crystalloids alone and to achieve rapid attainment of CVP goal.

Vasopressors should be administered when hypotension is persistent (MAP <65 mmHg) despite multiple 20 mL/kg crystalloid volume challenges, regardless of the CVP. In the presence of hypotension, organ perfusion cannot be maintained with fluids alone. Existing evidence does not clearly support the superiority of one vasopressor over another (Table 5.8). In the patient with refractory hypotension, vasopressin deficiency should be considered. Vasopressin, deficient in many septic shock patients, is an endogenously produced hormone. When administered in a relatively small, physiologic dose of 0.01–0.04 Units/min, supplemental vasopressin corrects the deficiency through a hypersensitive physiologic response. Although vasopressors may support blood pressure in the short term, long-term use of vasopressors is associated with increased mortality. If an increase in blood pressure is seen in early sepsis and there is an associated decrease in tissue oxygen delivery (decreased ScvO$_2$ or cardiac output), afterload reduction should be initiated.

Increasing oxygen-carrying capacity

A low ScvO$_2$ and elevated lactate level suggests a mismatch between systemic oxygen delivery and oxygen consumption of the tissues. When a low ScvO$_2$ is identified, therapies to augment one or more of the three components of oxygen delivery are recommended to restore the balance between systemic oxygen delivery and consumption: (1) oxygen-carrying capacity; (2) cardiac output; or (3) arterial oxygen saturation. This is the rationale for utilizing packed red blood cell (PRBC) transfusion, inotropic agents and supplemental oxygen with or without mechanical ventilation to increase ScvO$_2$. After MAP has been optimized, patients with inadequate oxygen delivery reflected by ScvO$_2$ <70% and hematocrit <30% should receive a transfusion of PRBCs in order to achieve a hematocrit ≥30%.

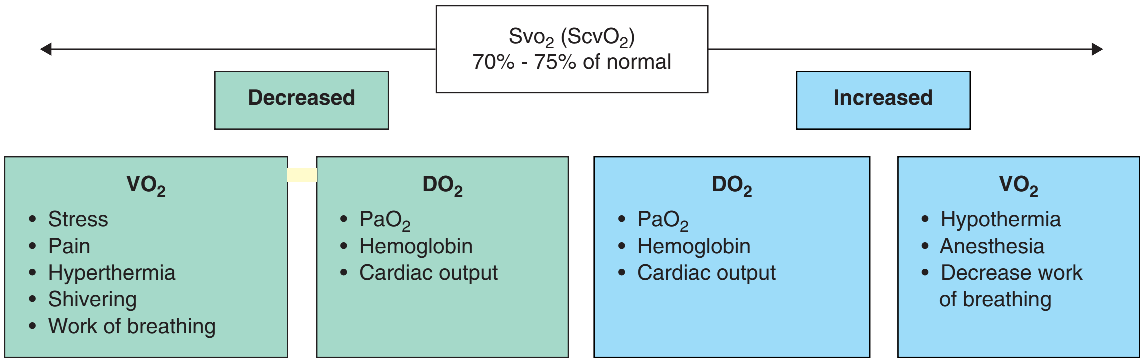


Figure 5.6
Changes in oxygen content. From Rivers EP, McIntyre L, Morro DC, Rivers KK. Early and innovative interventions for severe sepsis and septic shock: Taking advantage of a window of opportunity. *CMAJ* 2005;**173**:1054–65. Used with permission.

Table 5.8 Vasoactive agents

			Hemodynamic Effects				
Drug	**Dose (mixture)**	**Action**	**Cardiac stimulation**	**Vasoconstriction**	**Vasodilation**	**Cardiac output**	**Side effects and comments**
Norepinephrine	2–20 µg/min (4 mg/250 mL)	Primarily α-1, some beta-1	++	++++	0	Slight increase or no change	Dose-related reflex bradycardia; useful when loss of venous tone predominates, spares the coronary circulation
Dopamine	0.5–20 µg/kg/min (400 mg/250 mL)	α, β and dopaminergic	++ at 5–10 µg/kg/min	++ at 7 µg/kg/min	+ at 0.5–5 µg/kg/min	Usually increases	Tachydysrhythmias, increases myocardial oxygen consumption; a cerebral, mesenteric, coronary and renal vasodilator
Phenylephrine	40–200 µg/min (10 mg/250 mL)	Pure α	0	++++	0	Decrease	Reflex bradycardia, headache, restlessness, excitability, rarely dysrhythmias; ideal for patients in shock with tachycardia or supraventricular dysrhythmias
Vasopressin	0.01–0.04 Units/min (20 Units/100 mL)	α and V1	0	++++	+	Decrease	Outcome data of its use are lacking. Infusions of >0.04 Units/min may lead to adverse, likely vasoconstriction-mediated events. Reserved for refractory hypotension.
Epinephrine	1–10 µg/min (1 mg/250 mL)	α and β	++++ at 0.03–0.15 µg/kg/min	++++ at 0.15–0.30 µg/kg/min	+++	Increases	Causes tachydysrhythmias, leukocytosis; increases myocardial oxygen consumption and lactate production
Dobutamine	2.5–20 µg/kg/min (250 mg/250 mL)	β-1, some β- 2 and α-1 in large dosages	++++	+	++	Increase	Causes tachydysrhythmias, occasional GI distress, increases myocardial oxygen consumption, hypotension in volume-depleted patient. Has less peripheral vasoconstriction than dopamine.
Nitroglycerin	5–60 µg/min (100 mg/250 mL)	Smooth muscle relaxation of coronary and systemic vessels	0	0	+++	Slight decrease	Causes headache, dizziness, tachycardia, orthostatic hypotension, hypersensitivity reaction

0 = no effect, + = mild effect, ++ = moderate effect, +++ = marked effect, ++++ = very marked effect.

Inotropic therapy

If adequate volume, MAP and hematocrit goals are met yet $ScvO_2$ is <70%, dobutamine (2.5–20 μg/kg/min) should be started to improve contractility, and titrated to achieve $ScvO_2$ ≥70%. Patients with poor cardiac contractility may have elevated CVP and appear to be "volume overloaded," requiring diuresis. However, unresuscitated severe sepsis/septic shock patients will often also have underlying hypovolemia. Inotropic support with dobutamine in these patients may treat the myocardial depression and unmask hypovolemia. Volume resuscitation instead of diuresis will prevent subsequent cardiovascular collapse and vasopressor use in these situations. Because the vasodilatory effect of dobutamine could worsen hypotension, a vasopressor may be needed in combination with an inotrope for patients with persistent hypotension. In addition, dobutamine may also exacerbate tachycardia.

Decreasing oxygen consumption

When the goals of CVP, MAP and hematocrit are met, but $ScvO_2$ remains <70% despite a trial of dobutamine, or dobutamine caused an exaggerated response such as significant tachycardia and hypotension, one should consider reducing systemic oxygen demand and consumption. One of the greatest contributors to increased systemic oxygen demand is increased respiratory muscle use in breathing. In this situation, intubation and mechanical ventilation with sedation and paralysis decrease the work

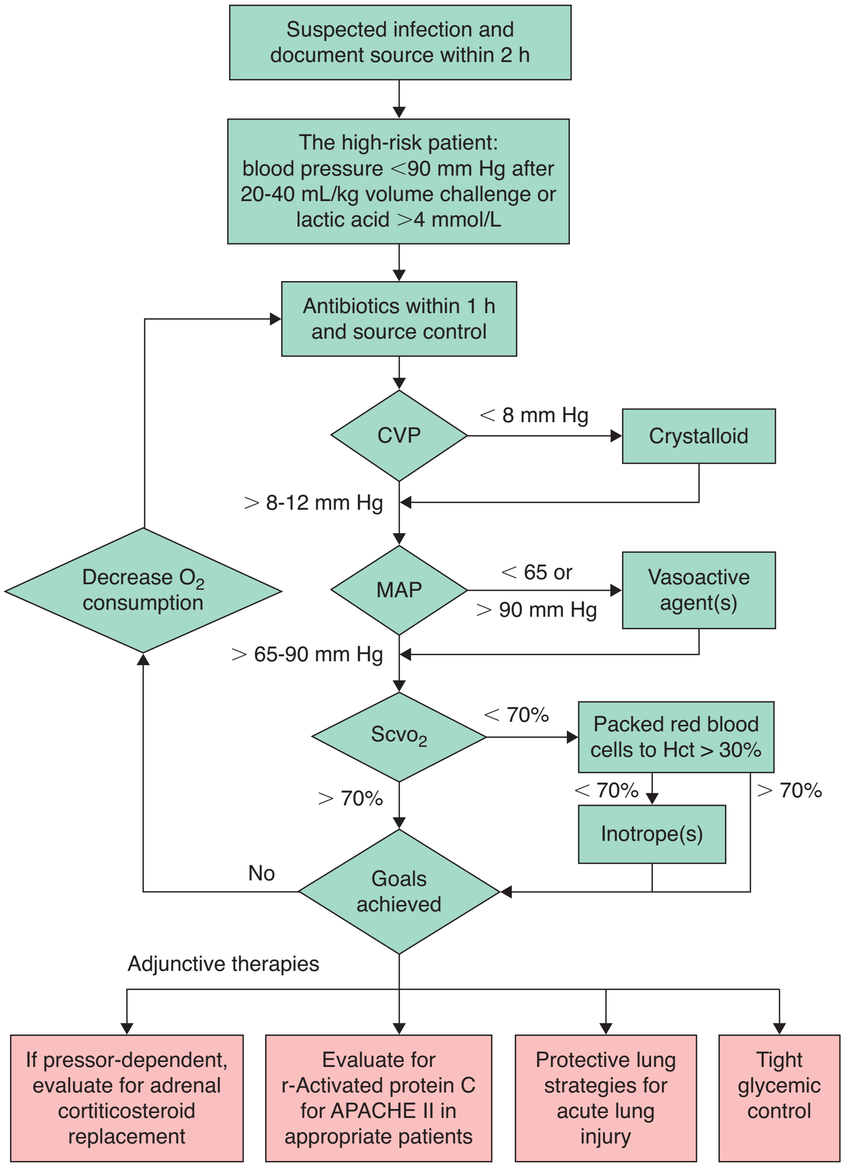

Suspected infection and document source within 2 h
The high-risk patient: blood pressure <90 mm Hg after 20-40 mL/kg volume challenge or lactic acid >4 mmol/L
Antibiotics within 1 h and source control
CVP
< 8 mm Hg
Crystalloid
> 8-12 mm Hg
Decrease O2 consumption
MAP
< 65 or
> 90 mm Hg
Vasoactive agent(s)
> 65-90 mm Hg
Scvo2
< 70%
Packed red blood cells to Hct > 30%
> 70%
< 70%
> 70%
Inotrope(s)
No
Goals achieved
Adjunctive therapies
If pressor-dependent, evaluate for adrenal corticosteroid replacement
Evaluate for r-Activated protein C for APACHE II in appropriate patients
Protective lung strategies for acute lung injury
Tight glycemic control

Figure 5.7
Treatment options in sepsis, summary algorithm. From Rivers EP, McIntyre L, Morro DC, Rivers KK. Early and innovative interventions for severe sepsis and septic shock: taking advantage of a window of opportunity. *CMAJ* 2005;173(9):1054–65 (p. 1061). Used with permission.

of breathing and redistribute blood flow from the respiratory muscles to the splanchnic and vital vascular beds.

Corticosteroids

The physiologic response to sepsis is a hypothalamic stimulation producing corticotrophin-releasing hormone (CRH). CRH stimulates the secretion of adrenocorticotropin-releasing hormone (ACTH), which stimulates the secretion of cortisol from the adrenal gland. An inflammatory mediator such as macrophage inhibitory protein inhibits the hypothalamic secretion of CRH, which suppresses the hypothalamic–pituitary axis and thus cortisol production.

Relative adrenal insufficiency is defined as a baseline cortisol less than 20 mcg/dL or an increase in serum cortisol of less than or equal to 9 mcg/dL one hour after administration of 250 mcg of ACTH. The clinical indications for considering adrenal insufficiency are when a patient remains vasopressor-dependent after adequate volume resuscitation. Up to 19% of hemodynamically unstable, vasopressor-dependent patients presenting to the ED will have adrenal insufficiency.

Recombinant human activated protein C

Cleavage of protein C by thrombin associated with thrombomodulin generates activated protein C, which has potent anticoagulant, profibrinolytic, anti-inflammatory and antiapoptotic effects. Recombinant human activated protein C (drotrecogin alfa [activated]) reduces mortality from severe sepsis/septic shock by 13% in septic patients with APACHE II scores of ≥25 or those with two or more sepsis-induced organ dysfunctions. The absolute contraindications to administration of drotrecogin alfa (activated) are active internal bleeding; recent hemorrhagic stroke within 3 months; recent intracranial or intraspinal surgery; severe head trauma within 2 months; trauma with an increased risk of life-threatening bleeding; presence of an epidural catheter; intracranial neoplasm, mass lesion or evidence of cerebral herniation; or known hypersensitivity to drotrecogin alfa (activated). With respect to the ED setting, especially in those patients with prolonged ED length of stay, the timing of drotrecogin alfa (activated) administration may be crucial for optimal outcome. Data suggest an improved mortality benefit is obtained if this drug is given in the first 24 hours of diagnosis.

Low-tidal volume mechanical ventilation

Many patients with severe sepsis/septic shock develop acute lung injury, defined as bilateral infiltrates consistent with pulmonary edema on chest radiograph, PaO_2/FiO_2 ratio ≤300, and no clinical evidence of left atrial hypertension (shown by physical examination or a pulmonary capillary wedge pressure ≤18 mmHg). The use of lower tidal volume mechanical ventilation when acute lung injury is present reduces mortality rates from 39.8% in conventionally ventilated patients to 31% in those who received low-tidal volume ventilation. In the low-tidal volume group, airway plateau pressures were kept ≤30 cm H_2O by decreasing the tidal volume to as low as 4 mL/kg if necessary; in the conventional tidal volume group, airway plateau pressures were not allowed to be >50 cm H_2O.

Intensive insulin therapy

Sepsis patients frequently have hyperglycemia without a previous history of diabetes. Although glucose control is associated with best outcomes in diabetic patients, strict glycemic control in the critically ill remains controversial. However, maintaining normal glucose levels is considered best practice in the management of the septic patient (Figure 5.7).

Special patients

Pediatric

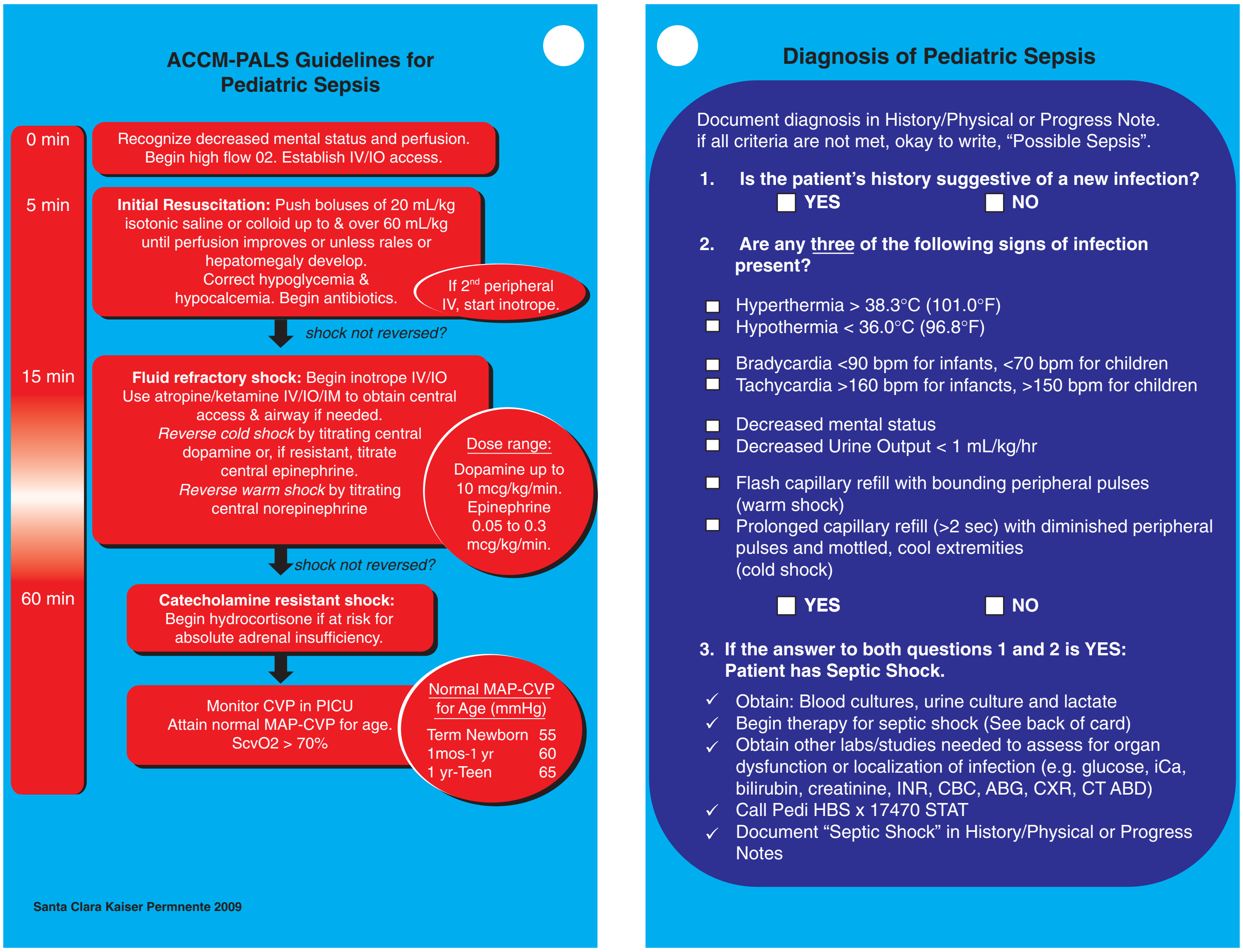
Aggressive treatment of pediatric sepsis has gained increasing importance in the literature. Similar to adult sepsis, pediatric sepsis treatment has time-sensitive goals. A flow diagram using ED goals is included (Figure 5.8). Refractory shock due to sepsis in this special population may benefit from extra-corporeal membrane oxygenation (ECMO).

Disposition

1. Patients with a lactate level ≥4 mmol/L are at high risk for mortality and are candidates for EGDT; these patients should be considered for ICU admission.
2. Patients who do not respond to an appropriate fluid challenge of 20–40 mL/kg are also at high risk for mortality and should be considered for ICU admission.

Pearls, pitfalls and myths

1. Sepsis can mimic many diseases upon presentation. The classic findings may not be present in many patients, especially the elderly.
2. Although the ED length of stay and the first 6 hours of ED management make up a relatively small portion of the overall hospitalization, these have significant impact on a patient’s morbidity and mortality.
3. The first steps of sepsis management include appropriate cultures, timely and recommended antibiotics, source control, and a lactic acid level. If the patient is at high risk, EGDT should be initiated immediately and not delayed until admission to an ICU.
4. After resuscitation, corticosteroids, glucose control, r-APC and protective lung strategies should be considered.



ACCM-PALS Guidelines for Pediatric Sepsis

0 min — Recognize decreased mental status and perfusion. Begin high flow 02. Establish IV/IO access.

5 min — **Initial Resuscitation:** Push boluses of 20 mL/kg isotonic saline or colloid up to & over 60 mL/kg until perfusion improves or unless rales or hepatomegaly develop. Correct hypoglycemia & hypocalcemia. Begin antibiotics.

If 2nd peripheral IV, start inotrope.

shock not reversed?

15 min — **Fluid refractory shock:** Begin inotrope IV/IO Use atropine/ketamine IV/IO/IM to obtain central access & airway if needed. *Reverse cold shock* by titrating central dopamine or, if resistant, titrate central epinephrine. *Reverse warm shock* by titrating central norepinephrine

Dose range:
Dopamine up to 10 mcg/kg/min.
Epinephrine 0.05 to 0.3 mcg/kg/min.

shock not reversed?

60 min — **Catecholamine resistant shock:** Begin hydrocortisone if at risk for absolute adrenal insufficiency.

Monitor CVP in PICU
Attain normal MAP-CVP for age.
ScvO2 > 70%

Normal MAP-CVP for Age (mmHg)	
Term Newborn	55
1mos-1 yr	60
1 yr-Teen	65

Santa Clara Kaiser Permnente 2009

Diagnosis of Pediatric Sepsis

Document diagnosis in History/Physical or Progress Note.
if all criteria are not met, okay to write, "Possible Sepsis".

1. **Is the patient's history suggestive of a new infection?**
   ☐ YES **☐ NO**

2. **Are any three of the following signs of infection present?**

☐ Hyperthermia > 38.3°C (101.0°F)
☐ Hypothermia < 36.0°C (96.8°F)

☐ Bradycardia <90 bpm for infants, <70 bpm for children
☐ Tachycardia >160 bpm for infancts, >150 bpm for children

☐ Decreased mental status
☐ Decreased Urine Output < 1 mL/kg/hr

☐ Flash capillary refill with bounding peripheral pulses (warm shock)
☐ Prolonged capillary refill (>2 sec) with diminished peripheral pulses and mottled, cool extremities (cold shock)

☐ YES **☐ NO**

3. **If the answer to both questions 1 and 2 is YES: Patient has Septic Shock.**

✓ Obtain: Blood cultures, urine culture and lactate
✓ Begin therapy for septic shock (See back of card)
✓ Obtain other labs/studies needed to assess for organ dysfunction or localization of infection (e.g. glucose, iCa, bilirubin, creatinine, INR, CBC, ABG, CXR, CT ABD)
✓ Call Pedi HBS x 17470 STAT
✓ Document "Septic Shock" in History/Physical or Progress Notes

Figure 5.8
Stepwise management of hemodynamic support with time-sensitive goals for infants and children in shock (proceed to next step if shock persists). Modified from Brierley J, Carcillo JA, Choong K, et al. *Crit Care Med* 2009;37(2);666–88 (p. 677). Used with permission from Kaiser Permanente.

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6 Shock

Jairo I. Santanilla, MD and Peter M.C. DeBlieux, MD

Scope of the problem

Shock is a state in which the oxygen (O_2) and metabolic demands of the body are not met by the cardiac output. When this process occurs in a single organ rather than throughout the body, organ ischemia and infarction ensue. When shock occurs on a more global level, multiorgan dysfunction and failure occur, ultimately leading to death if not corrected. Shock is most often accompanied by hypotension, termed *decompensated shock*. However, shock may also occur when the blood pressure is normal or elevated. Examples include hypertensive emergency with compromised cardiac output, or carbon monoxide poisoning with the inability to deliver O_2 despite normal hemodynamics. The approach to the patient in shock must proceed with the same urgency as the patient suffering from an acute myocardial infarction or cerebral vascular accident.

General approach to the patient in shock

The most important step in the approach to a patient in shock is early recognition of the shock state. If shock is defined by impaired global organ perfusion, then the signs of shock are derived from impaired organ function. Hypotension is an obvious sign of decompensated hemodynamics associated with shock. Alteration in mental status, diaphoresis, chest pain, evidence of cardiac failure, difficulty breathing, tachypnea, agonal respirations, abdominal pain from intestinal ischemia, low urinary output, and cold, clammy or mottled skin all suggest shock.

The approach to shock in this chapter is outlined in the traditional stepwise fashion of medicine (history, physical examination, diagnostic evaluation, differential diagnosis and treatment). However, in most cases of shock, multiple paths must be followed in parallel. As such, data from the history and physical examination need to be obtained as diagnostic and therapeutic measures are performed.

For some patients, the etiology of the shock state remains in question after the initial evaluation. Often, therapeutic intervention must be initiated without a verifiable diagnosis. The core principle for treatment of such patients is that O_2 delivery to the vital organs must be optimized, using any method possible.

The key concept is that in a state of shock, oxygen demands are not being met; therefore, the components of oxygen delivery are of utmost importance. The oxygen delivery equation includes oxygen saturation (SaO_2), hemoglobin (Hb), partial pressure of arterial oxygen (PaO_2) and cardiac output (CO).

$$DO_2 \text{ (Oxygen Delivery)} = CO \times [(1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)]$$

$$CO = \text{Stroke Volume (SV)} \times \text{Heart Rate (HR)}$$

Because stroke volume is related to preload, afterload and contractility, oxygen delivery (DO_2) is based on SaO_2, Hb, preload, afterload and cardiac contractility.

History

Obtaining an accurate history is essential to the evaluation and treatment of undifferentiated patients in shock. An incomplete history can lead to poor treatment choices and increased morbidity and mortality. Unfortunately, many patients in shock states are not able to provide an accurate and complete history. Paramedics, family members, friends, witnesses and medical records are invaluable resources in these situations. Time course and progression of illness provide important information regarding the rapidity of decline and may help narrow the differential diagnosis. The presence of trauma is an important consideration. Preexisting conditions, particularly limitations of the cardiopulmonary system and immune deficiencies, predispose patients to poor outcomes.

Obtaining a patient's complete medication list is vital to address the needs of a patient in shock. Medications that impair normal cardiac compensation in shock states, such as beta-blockers, calcium channel blockers or digitalis, may alter patient presentations and vital signs in profound shock. New medications may be involved in anaphylactoid reactions, drug–drug interactions, or toxidromes. Similarly, immunosuppressant agents (such as prednisone and chemotherapeutic drugs) may impair host immune response and mask serious or life-threatening infections. Social historical data focusing on alcohol, illicit drug use, work history and psychosocial support systems may offer insight into these complex patients.

Physical examination

Physical examination and rapid assessment of the patient in shock follow the basic tenants of emergency medicine. Airway, breathing, circulation, disability and exposure (ABCDE) are critical in the initial evaluation of complex patient presentations. One of the first steps in determining cardiopulmonary reserve is assessment of all the vital signs: heart rate, respiratory rate, blood pressure, pulse oximetry and temperature. An accurate set of vital signs is paramount in the management of a patient in shock. In shock states, blood pressure may need to be auscultated using a Doppler stethoscope, pulse oximetry may not capture secondary to cold extremities, and a rectal temperature is often required.

If the impairment of shock is the inability to adequately provide O_2 at the end organ, then the first critical appraisal must be airway, quickly followed by breathing and circulation. These first three steps comprise the critical care concept of "cardiopulmonary reserve." Cardiopulmonary reserve refers to the interdependence of the heart, lungs and O_2-carrying capacity of all patients. Those patients with an impaired cardiac pump, preexisting pulmonary disease, or abnormalities in hemoglobin may require more immediate intervention for milder shock states when compared with patients with normal cardiopulmonary physiology. Normal lungs, heart and hemoglobin permit a degree of physiologic reserve that allows patients to compensate for any given cardiopulmonary insult.

In evaluating and treating patients in shock, the goals are to maintain adequate tissue oxygenation and organ perfusion. Pulse oximetry can be utilized as an initial screening tool to determine the adequacy of oxygenation. Goal saturations during resuscitation and treatment should be maintained above 90%, although outcome data do not exist for this universally accepted goal. O_2 delivery devices may be required to reach the goal of 90%. If adequate O_2 saturations are not obtained with a 100% non-rebreather mask, patients should be endotracheally intubated and placed on mechanical ventilation. Overreliance on a normal oximetry measurement may result in management error, as certain states, such as carbon monoxide and cyanide poisoning, have normal or near-normal oximetry with relative hypoxia at the cellular level.

Once oxygenation has been addressed, the focus should be placed firmly on maintaining adequate cerebral and coronary perfusion pressures to prevent injury to these vital organs. Vital organ perfusion pressure is a function of mean arterial blood pressure (MAP), which can be approximated from the systolic blood pressure (SBP) and diastolic blood pressure (DBP). The critical nature of the DBP is evident from the formula to calculate MAP.

$$\text{MAP} = \text{DBP} + 1/3\,(\text{SBP} - \text{DBP})$$

Goals for resuscitation in the majority of shock states are a MAP of 65 to 75 mmHg, which provides adequate cerebral and coronary perfusion.

MAP can be better understood as it relates to preload and afterload. Physiologically, *preload* is defined as the left ventricular end diastolic wall tension. The patient's clinical presentation or physical exam findings may predict whether the preload is low, normal or high. Actively bleeding patients, trauma victims, or chronically dehydrated patients are virtually certain to have a low preload. An edematous patient with congestive heart failure (CHF) is likely to be volume overloaded. Estimation of the jugular venous pressure (JVP) on physical examination may suggest a patient's volume status; however, this technique is not always accurate, even in the hands of an experienced clinician. Auscultation of the heart and lungs is sensitive for detecting signs of volume overload (e.g., S3, crackles, rales), but does not distinguish a hypovolemic state. Assessment of skin turgor, capillary refill, and mucous membranes can likewise be misleading.

Afterload is the force that the heart must generate in order to eject blood into the arterial compartment. Because MAP is proportional to the product of systemic vascular resistance (SVR) and CO, SVR is one of the main determinants of afterload. A comprehensive review of the technique for insertion, calibration and collection of data from a pulmonary artery (PA) catheter is beyond the scope of this chapter.

It is essential to note that excessive heart rate (HR) increases myocardial O_2 consumption and may further compromise at-risk myocardium. Additionally, patients with normal vital signs can be in profound shock states despite normal calculated MAP, central venous pressure (CVP), HR and O_2 saturation.

After the assessment of cardiopulmonary reserve, a rapid neurologic assessment is performed, followed by complete exposure of the patient. Next, a comprehensive head-to-toe physical examination is performed to identify further evidence of decreased organ perfusion and to search for the etiology of shock. Altered mental status, cyanosis, delayed capillary refill and skin mottling may be early signs of decreased oxygenation and perfusion.

Diagnostic testing

Rapid bedside screening is the hallmark of the initial assessment of the undifferentiated patient in shock. Vital signs, pulse oximetry and continuous monitoring are standard measures. A Foley catheter with urometer should be inserted to assess hourly urinary output (0.5 ml/kg/hour). Initial screening studies for the undifferentiated patient include bedside blood sugar analysis, arterial blood gas analysis with hemoglobin, chest radiography and an electrocardiogram. A comprehensive metabolic profile, complete blood count and urinalysis may also be required. Consideration for toxicologic studies, urine pregnancy test, blood and urine cultures, cardiac profiles, and endocrine screening should be made on a case-by-case basis. Serum lactate levels can be used to screen for shock in the normotensive patient; they may also guide therapy and have prognostic value. This study is available as a rapid point-of-care test at many hospitals. Additional radiographic studies of the head, chest, abdomen, pelvis and extremities are second-tier studies and should only be obtained once the patient has been clinically stabilized.

Bedside ultrasonography has recently entered the mainstream in the evaluation of undifferentiated shock. A trained operator can use the bedside ultrasound machine to quickly evaluate for pericardial effusion and tamponade, cardiac hypokinesis, pneumothorax, inferior vena cava collapsibility (as a surrogate for volume responsiveness), and abdominal aortic aneurysm and intra-abdominal free fluid. The same machine can then be used to place intravascular resuscitation lines.

Several hemodynamic monitoring systems are available to assist with the diagnosis and management of patients in shock. Right heart catheterization is useful to determine the etiology of various shock states, but is rarely available in the emergency department (ED). Central venous lines offering continuous CVP and central venous oxygen saturation ($ScvO_2$) monitoring are being used increasingly in EDs. Arterial waveform analysis technologies that provide SV variability and SVR indices are currently employed in critical care units, and may soon be incorporated in the ED setting.

Differential diagnosis

History, physical examination and bedside ultrasound can often determine the etiology of a shock state (e.g., hypovolemic shock from trauma and exsanguination, neurogenic shock from a spinal cord injury, obstructive shock from pericardial tamponade). There are some shock states that are challenging to recognize, however (e.g., mixed syndromes, pulmonary embolism, septic and cardiogenic shock). Table 6.1 outlines physiologic parameters characterizing each shock state.

Table 6.1 Physiologic parameters in shock state

	CVP	PAOP	SVR (SVRI)	CO (CI)
Hypovolemic	↓	↓	↑	↓
Cardiogenic	↑↔	↑↔	↑	↓
Distributive				
Sepsis	↔↓	↔↓	↓↔	↕
Anaphylaxis	↔↓	↔↓	↓	↑
Neurogenic	↔	↔	↓	↔↑
Obstructive				
Tamponade	↑	↑	↑	↓
Tension PTX	↑↓[a]	↑↓[a]	↑	↓
Massive PE	↑	↑↓[b]	↑	↓

CVP: central venous pressure; PAOP: pulmonary artery occlusion pressure; SVR: systemic vascular resistance; SVRI: systemic vascular resistance index; CI: cardiac index; CO: cardiac output; PTX: pneumothorax; PE: pulmonary embolism.

[a] True CVP and PAOP are diminished due to impaired venous return. Measured pressure is falsely elevated, reflecting pleural pressure rather than vascular pressure.

[b] True left atrial pressure is low due to obstruction of flow through the pulmonary vasculature. Measured pressure may be falsely elevated, reflecting pulmonary vascular resistance rather than left heart filling pressure.

Classification

Shock states are classified according to their underlying physiologic derangement. Table 6.2 lists the most commonly used classification system. *Hypovolemic shock* is defined by decreased circulating blood volume, due to either blood or fluid loss, which compromises cardiac output. Impaired cardiac performance characterizes *cardiogenic shock.* Loss of vasomotor tone with hypotension is the hallmark of *distributive shock,* as in sepsis, anaphylaxis, or certain intoxications. Anatomic interruption of sympathetic output, usually secondary to spinal cord injury with disruption of the cervical sympathetic chain, leads to a paradoxical bradycardia with hypotension known as *neurogenic shock.* Obstruction of blood flow through the cardiopulmonary circuit is the etiology of *obstructive shock,* as occurs in tension pneumothorax, cardiac tamponade, or massive pulmonary embolus. Patients may present with a *mixed syndrome,* such as a patient with sepsis who develops gastrointestinal (GI) hemorrhage, or who suffers a concomitant myocardial infarction.

Table 6.2 Classification of shock states

Hypovolemic shock Hemorrhage Fluid loss/dehydration
Cardiogenic shock Pump failure Valvular disorders Cardiac dysrhythmia
Distributive shock Sepsis Anaphylaxis Intoxications
Neurogenic shock[a] Spinal cord injury
Obstructive shock Tension pneumothorax Pericardial tamponade/constrictive pericarditis[b] Massive pulmonary embolus Severe pulmonary hypertension Severe valvular stenosis

[a] Classified by some as distributive shock.

[b] Classified by some as cardiogenic shock.

Hypovolemic shock

Hypovolemic shock is defined by the loss of intravascular volume. CVP, pulmonary artery occlusion pressure (PAOP), and CO are low, whereas SVR is elevated. In the early compensated stages, the pulse pressure is narrowed due to vasoconstriction, but ultimately hypotension occurs with decompensation. Ultrasound examination may reveal a hyperdynamic heart, empty heart chambers, and a markedly collapsible inferior vena cava. The initial treatment of hypovolemic shock is aggressive volume expansion with crystalloid. Transfusion of blood products may be required if hemorrhage is the cause of hypovolemia.

Cardiogenic shock

The most common cause of cardiogenic shock is acute myocardial infarction, accounting for nearly half the cases.

Low CO and high SVR characterize cardiogenic shock. CVP and PAOP are most often elevated during acute exacerbations of CHF, but may be normal in patients who are adequately diuresed or volume-depleted from dehydration or concomitant sepsis. Bedside ultrasound may demonstrate an abnormally contracting heart (e.g., global hypokinesis from cardiomyopathy or segmental wall motion abnormalities from acute myocardial infarction).

The initial treatment of cardiogenic shock depends on the afterload and preload that the heart is experiencing. Patients may require afterload and preload reduction with vasodilators and diuresis, or may require fluids, vasopressors and inotropes. Strong evidence supporting selection of one vasopressor over another does not exist. Consensus committee (American College of Cardiology/American Heart Association) has recommended the use of dobutamine if SBP >90 mmHg, dopamine if SBP <90 mmHg, and norepinephrine if hypotension is severe or refractory to dopamine infusion. Pulmonary artery catheters may be needed to guide fluid management and vasoactive agent administration.

Urgent cardiology consultation may be warranted for intra-aortic balloon pump (IABP) placement in patients who do not respond to vasopressor therapy. An intra-aortic balloon inflates during diastole, augmenting MAP and systemic perfusion, and deflates during systole, effectively diminishing afterload and improving CO. Percutaneous coronary interventions (PCI) and/or coronary artery bypass grafting (CABG) should be performed in patients with acute ST-segment elevation myocardial infarction (STEMI) complicated by shock.

Distributive shock

In early sepsis, SVR is elevated. However, as sepsis progresses to septic shock, SVR drops precipitously. CO is increased in most cases, but a cytokine known as myocardial depressant factor is believed to be released from the pancreas, and may impair systolic function in later stages. Impaired cardiac perfusion will also adversely affect CO. Vascular permeability is increased. Fluid shifts and increased insensible losses may lead to intravascular volume depletion and low CVP and PAOP. Early broad-spectrum antibiotic therapy and emergent surgical drainage or debridement (when indicated) are cornerstones of treatment. Early goal-directed therapy (EGDT) has been shown to reduce mortality in patients when intravascular volume, MAP and $ScvO_2$ goals are attained within 6 hours of presentation (see Chapter 5). Volume replacement should be guided by invasive monitoring, with CVP goals of 8–10 cm H_2O, MAP goals of >65 mmHg, and $ScvO_2$ goals >70%. If the CVP is not at goal, further fluid boluses are needed. After several boluses, if the MAP is not at goal, vasopressors should be initiated. Presently, norepinephrine is the vasoactive agent of choice. If the $ScvO_2$ is not at goal, blood transfusion to keep Hb >10 mg/dL, followed by CO augmentation with dobutamine should be initiated. Activated protein C complex (Xigris®) may improve survival in selected patients with severe sepsis. In addition, because the potential for adrenal insufficiency exists in septic patients, steroid replacement should be empirically started in patients with vasopressor-dependent septic shock.

Anaphylactic shock is accompanied by the massive release of cytokines in an inflammatory cascade with loss of vasomotor tone and increased vascular permeability. Epinephrine, steroids and antihistamines are initial therapies. Persistent hypotension requires infusion of an agent that supports vasomotor tone. In this situation, norepinephrine makes the most sense. In severe cases, an epinephrine drip may be required.

Neurogenic shock

Neurogenic shock, classified by some as a type of distributive shock, is a consequence of injury to the sympathetic ganglion chain. Neurogenic shock characteristically manifests as hypotension and bradycardia. Because acute spinal cord injury is most prevalent in younger patients, this shock state usually occurs in patients with normal cardiac function. Before diagnosing neurogenic shock, it is of the utmost importance to rule out occult hemorrhage, and to use signs of organ perfusion to guide the initiation of pharmacologic therapy. Many patients in neurogenic shock perfuse their organs well at below-normal MAP. If signs of hypoperfusion develop, selection of an agent that supports SVR (norepinephrine or phenylephrine) makes the most sense physiologically. Phenylephrine should be used with caution in the markedly bradycardic patient, as its use may cause a reflex bradycardia.

Obstructive shock

Two causes of obstructive shock, tension pneumothorax and cardiac tamponade, are reversible by surgical intervention. Support of these patients by volume loading is temporizing at best. In tension pneumothorax, needle decompression is both diagnostic and therapeutic. Ultrasound may show the loss of "lung slide," the loss of the "seashore sign," and a positive "bar code" sign in M-mode. In cardiac tamponade, ultrasound may show diastolic collapse of the right ventricle in the presence of a pericardial effusion. Pericardiocentesis is best done under ultrasound guidance.

Massive pulmonary embolus causes the release of vasoactive cytokines from the pulmonary vascular bed, obstruction of flow, and acute right ventricular dysfunction, collectively impairing left ventricular filling. Bedside ultrasound may reveal a dilated right ventricle and/or septal bulging into the left ventricle. Thrombolysis may be lifesaving in patient with massive pulmonary embolus. If lytic therapy fails, or if contraindications exist, thrombectomy may be used as a rescue procedure in centers with such expertise. Support of cardiac function with volume infusion and norepinephrine may be a bridge to these interventions.

Chronic pulmonary hypertension may limit flow through the pulmonary vascular bed; therefore, the onset of shock is commonly an end-stage, preterminal event. Treatment with potent pulmonary vasodilators is hazardous in this shock state since hypotension from peripheral arterial dilation is a frequent side effect, mandating use of a pulmonary artery catheter. Although rarely available in the ED, inhaled nitric oxide may have a role in patients with severe pulmonary hypertension.

General treatment principles

The treatment goal in a patient presenting with shock is optimization of oxygen delivery to end organs. Understanding the treatment for deficiencies in oxygenation (oxygen supplementation and possibly assisted ventilation), preload (volume intervention), afterload (vasopressors), contractility (inotropes), and hemoglobin (transfusion) are essential for the clinician.

Oxygenation

Whenever a shock state is present, O_2 supplementation is required. O_2 may be delivered via facial delivery devices, noninvasive mechanical ventilation, or conventional mechanical ventilation.

Simple means of delivering supplemental O_2 include the use of a nasal cannula, venturi mask, or O_2-reservoir non-rebreathing apparatus. O_2 delivered via nasal cannula is appropriate only when low O_2 flow is required. It is impossible to determine the fraction of inspired O_2 (FiO_2) delivered to any given patient because it varies with respiratory rate, the degree of nasal versus mouth breathing, and the O_2 flow rate. In general, if more than 5 L/min of O_2 flow is required with a nasal cannula, an alternative device should be employed.

A venturi mask uses various O_2 flow rates combined with various venturi apertures to produce increasing O_2 supplementation, generally higher than can be delivered by nasal cannula. Although each mask lists specific FiO_2 ratings from 0.28 to 0.50, these are rough estimates at best. If the listed flow rate with the smallest aperture does not provide enough supplemental O_2, then an alternative device is required.

A non-rebreathing apparatus combines a collapsible bag reservoir with high-flow O_2 and an exhalation valve so that high FiO_2 can be delivered. When used optimally, the FiO_2 range may approach 0.6–0.8.

The current literature supports the use of noninvasive positive pressure ventilation (NPPV) in patients without hemodynamic compromise, cardiac dysrhythmias, or altered mental status. Therefore, NPPV use in the management of shock should be limited to patients with respiratory failure without hemodynamic instability. This literature strongly supports the use of NPPV in patients with hypercapnic hypoxemic respiratory failure, such as those with exacerbation of chronic obstructive pulmonary disease (COPD). Data are available from descriptive studies regarding its use in selected cases of hypoxemic respiratory failure, such as acute respiratory distress syndrome (ARDS), but prospective randomized controlled trials are lacking. Prospective trials investigating noninvasive mechanical ventilation use in CHF with pulmonary edema suggest both continuous positive airway pressure (CPAP) and bilevel positive airway pressure (Bi-PAP) are beneficial. NPPV is especially beneficial and should be used if hypercapnia is present and contraindications are absent.

Early generations of noninvasive ventilators bled O_2 into the ventilator tubing, so FiO_2 was not tightly controlled. O_2 flow was increased until the patient's arterial O_2 saturation was optimized. In newer models, the FiO_2 can be more precisely set with a mixture valve, and adjusted as needed based on saturation monitoring.

Invasive mechanical ventilation should be considered for any patient who does not achieve adequate SaO_2 despite maximal noninvasive O_2 supplementation. Additionally, patients whose expected clinical course is projected to progressively deteriorate should be intubated early. Patients with a high work of breathing (WOB) should be intubated in order to reduce the amount of work the respiratory system must perform and support the metabolic needs of the patient. All patients who are placed on invasive ventilation should initially receive an FiO_2 of 1 because the switch from spontaneous breathing (negative pressure) to assisted ventilation (positive pressure) causes unpredictable alterations in pulmonary blood flow and ventilation–perfusion mismatch. FiO_2 can then be decreased as the patient's SaO_2 allows. Patients with pulmonary edema, particularly those with ARDS, may require the addition of positive end-expiratory pressure (PEEP) to optimize oxygenation. Although many factors must be considered in determining the optimal level of PEEP, most authors recommend starting at 3–5 cm H_2O. Thereafter, PEEP is incrementally increased by 2–3 cm H_2O, allowing 15–30 minutes after each increase for alveolar recruitment. PEEP is increased until SaO_2 reaches a minimum of 88–90%. Further increases in PEEP may then be required to allow the FiO_2 to be decreased. Increases in PEEP, however, result in increases in mean intrathoracic pressure. A critical point is reached when venous return to the heart is compromised due to increased intrathoracic pressure, impairing CO.

Volume intervention

Following initial assessment of preload, either fluid or diuretic therapy should be instituted. The size of an initial fluid bolus is a matter of clinical judgment. A previously healthy young adult with acute hemorrhage may safely receive rapid infusion of several liters of crystalloid solution. In contrast, a frail, elderly patient with a history of CHF may require (or tolerate) only a few hundred milliliters of crystalloid boluses at a time. The crucial step is continued reassessment after each intervention to decide whether further volume expansion is indicated.

A patient who is volume overloaded requires diuresis. Loop diuretics (e.g., furosemide, torsemide, bumetanide) are the most commonly used first-line agents. Frequent reassessment of urinary output is mandatory to guide subsequent therapy. Other interventions that may be used to reduce preload include the administration of nitrates (the most effective pharmacologic therapy), B-type natriuretic peptide (nesiritide), opiates, rotating tourniquets and dialysis. Opiates should be used with caution as they are associated with worse outcomes in acute CHF.

Vasoactive agent intervention

Treatment of abnormal contractility and afterload should follow preload correction, particularly in hypovolemic states. Vasoconstricting agents in the setting of volume depletion will further compromise organ perfusion, causing organ ischemia and infarction. Many vasoactive medications used to treat shock affect both myocardial contractility and SVR. A thorough knowledge of the action of adrenergic receptor physiology and the action of the vasoactive agents on these receptors is necessary to guide selection of a vasoactive agent.

Alpha-1 (α-1) receptors are found in arterial smooth muscle and in the conduction system of the heart. The physiologic effect of α-1 stimulation is increased cardiac excitation/conduction and arterial vasoconstriction (including coronary, cerebral, renal and splanchnic arterial beds). Beta-1 (β-1) receptors are found in the myocardium and the conduction system. β-1 stimulation results in increased contractility and cardiac excitation. Beta-2 (β-2) receptors are found in arterial and bronchial smooth muscle. β-2 stimulation results in arterial vasodilation.

Although many of the vasoactive medications affect both afterload (MAP and SVR) and contractility, it is helpful to remember which vasoactive medications function more as a vasopressor and which function more as an inotrope. The vasoactive agents are listed in Table 6.3, as first-and second-line agents.

Table 6.3 Vasoactive medications and initial dose

Vasopressors	Inotropes
First-line agents	**First-line agents**
Phenylephrine	Dobutamine
0.2 mcg/kg/min	3 mcg/kg/min
Norepinephrine	Dopamine
0.02 mcg/kg/min	3 mcg/kg/min
Dopamine	
3 mcg/kg/min	
Second-line agents	**Second-line agents**
Vasopressin	Epinephrine
0.04 Units/min	0.02 mcg/kg/min
Epinephrine	Milrinone
0.02 mcg/kg/min	0.25 mcg/kg/min

Vasopressors

Table 6.4 provides the relative affinity of first-line agents at the α and β receptors. Table 6.5 lists suggested dose range of vasopressors.

Phenylephrine is a pure α-1 agonist. It may be useful in the management of vasomotor collapse, as in distributive or neurogenic shock. This vasopressor is best used when cardiac contractility is good, heart rate is markedly elevated, and the goal is to raise SVR. Due to its pure α-1 activity, phenylephrine has been known to cause a reflex decrease in HR and should therefore be avoided in those with low initial HR.

Table 6.4 Receptor affinity and hemodynamic effects

	α-1[a]	β-1[b]	β-1[c]	β-2[d]
Dopamine				
Low dose	0	2+	2+	2+
High dose	3+	2+	2+	2+[e]
Dobutamine				
Low dose	0	4+	1+	1–2+
High dose	1–2+	4+	1+	1–2+
Norepinephrine	4+	2+	2+	0
Epinephrine	4+	4+	4+	3+
Phenylephrine	4+	0	0	0

[a]Vasoconstriction; [b]inotropic; [c]chronotropic; [d]vasodilation; [e]effect lost.

Modified from Khalaf S, DeBlieux PMC. Managing shock: The role of vasoactive agents, part I. *J Crit Illness* 2001;16(6)281–7, and Khalaf S, DeBlieux PMC. Managing shock: The role of vasoactive agents, part II. *J Crit Illness* 2001;16(7)334–42.

Table 6.5 Dose ranges of vasoactive agents in adults

Dopamine and dobutamine	
Low dose	0–5 mcg/kg/min
High dose	10–20 mcg/kg/min
Norepinephrine	0–3 mcg/kg/min
Phenylephrine	0–5 mcg/kg/min
Epinephrine	0–2 mcg/kg/min
Vasopressin	0.04 Units/min

Norepinephrine is predominately an α-1 agonist, although it has non-selective β activity as well. At low doses, it raises CO and SVR proportionately, but its potential to raise CO is limited. As the infusion rate increases, its effect is essentially limited to an increase in SVR and HR. The primary role of norepinephrine is in the treatment of shock with hypotension attributable to low SVR. A consensus committee has recommended norepinephrine as the agent of choice in cardiogenic shock with SBP <70 mmHg. It also appears to be the vasopressor of choice in septic shock.

Dopamine activates β receptors at moderate dose range (3–8 mcg/kg/minute) and both α and β receptors at higher infusion rates (>10 mcg/kg/minute). Clinically, SVR is decreased and CO is increased at low doses. At higher doses, SVR increases, blunting further rise in CO. Dopamine has been recommended as the agent of choice in patients with cardiogenic shock and SBP between 70 and 90 mmHg. Dopamine may cause pulmonary vasoconstriction, with resultant rise in PAOP, limiting its value as an index of left heart preload. Tachyphylaxis to dopamine infusion may occur.

Epinephrine is a potent α and β agonist, roughly 500 times more potent than dopamine or dobutamine. It is dysrhythmogenic, increases myocardial O_2

consumption and causes tachycardia. Its use is limited to cardiac arrest, refractory life-threatening bradycardia and anaphylactic shock.

Dopamine and epinephrine may be used primarily for their inotropic properties in selected patients.

Vasopressin is an endogenous peptide hormone that has vasoconstrictive and antidiuretic effects via receptors in the vascular smooth muscle and kidneys. It has undergone preliminary investigation as an agent for use in septic shock. Its main role has been as a second-line agent when norepinephrine has reached its maximum threshold.

Inotropes

Dobutamine activates β receptors throughout its dose range, and is a more potent cardiac stimulant than dopamine. It has weaker α receptor activity than dopamine. The balance of its effects of increased CO and decreased SVR can have a variable effect on MAP. Patients with large increases in contractility tend to experience a rise in MAP, whereas those with little increase in CO in response to dobutamine tend to have no change in or diminished MAP. It is impossible to predict which patients will respond to dobutamine with increased CO; however, younger patients tend to be more responsive than the elderly. In contrast to dopamine, dobutamine tends to cause pulmonary vasodilation.

Milrinone is not an adrenergic receptor agonist. Instead, it inhibits phosphodiesterase, producing an effect similar to β agonists. This is a second-line agent for the treatment of cardiogenic shock, and may be additive in effect to dobutamine. It can also be used in patients with hypotension associated with end-stage liver disease.

Blood transfusion intervention

The effect of raising the hemoglobin (Hb) on O_2 delivery is profound. The administration of 2 units of packed red blood cells (PRBCs) to increase the Hb by 25% (e.g., an increase of hematocrit from 20% to 25%) will also increase the calculated O_2 delivery by 25%. For this reason, administration of blood should be considered in patients with shock and anemia. Rapid estimation of Hb is available in most centers by commercially available analyzers, blood gas machines, or centrifuge techniques.

Until recently, the threshold for blood administration has been dictated by practice habit, not evidence in the medical literature. It is generally recommended that adult trauma victims unresponsive to initial volume expansion with 2 L of crystalloid receive blood transfusion. Patients in shock states with coronary artery disease or CHF should be transfused with a goal of keeping the hematocrit above 30%. Other patients may benefit from blood therapy if the hematocrit falls below 20–24%. Of note, blood therapy has not been demonstrated to improve survival, decrease the duration of mechanical ventilation, or decrease the need for vasopressors. Controversy also exists because transfused allogenic PRBCs may impair host immune response, and are less efficient at carrying O_2 than native RBCs.

Septic patients who present to the ED with shock benefit from transfusion of PRBCs if the Hb falls below 10 mg/dL and the $ScvO_2$ is less than 70% despite adequate volume resuscitation (CVP 8–10) and adequate perfusion pressures (MAP >65 mmHg).

Cardiac intervention

Pathologic rhythms may be a cause or consequence of a shock state. In either scenario, the goal of therapy should be conversion to a perfusing rhythm. Bradycardic rhythms should be sped up either pharmacologically or with electrical transthoracic or transvenous pacing. Atropine is considered the first-line agent in patients with a pulse. It should be considered a temporary measure, and preparation for pacing should be rapidly accomplished. In contrast, a bradycardic patient without a pulse should receive CPR and doses of epinephrine while preparing to initiate electrical pacing.

The principles for electrically pacing the heart are the same for transthoracic and transvenous techniques. In both modes, the initial HR is set between 80 and 100 beats per minute. In the pulseless patient, the output is set at maximum, and dialed downward after the heart demonstrates capture. In contrast, the output is set at a minimum in the patient with a pulse, and dialed upward until capture is achieved. This is due to discomfort that maximal output can cause an awake patient. In both scenarios, the final output should be set at 10–20% above the threshold for capture. Causes of failure to capture include malposition of the pacing leads, hypothermia, hypoglycemia, hypoxemia, acidosis and electrolyte disturbance.

Sinus tachycardia in the shock state is compensatory. Except in some types of intoxication (sympathomimetic or anticholinergic overdose), acute ischemic coronary syndromes and other unusual circumstances, measures directed at slowing the HR should be limited to correcting the underlying cause. All other tachycardias are pathologic, and may be the etiology for the shock state. These should be converted to a perfusing rhythm as rapidly as possible, usually with electrical cardioversion.

The exception to this rule is atrial fibrillation. Acute atrial fibrillation, defined as atrial fibrillation for less than 48 hours, may be treated with cardioversion. Patients with chronic atrial fibrillation, defined as atrial fibrillation with a duration greater than 48 hours, have an increased risk of systemic embolization of an atrial thrombus if cardioverted. Such patients, or those in whom the duration of atrial fibrillation is unknown, should receive anticoagulation or undergo transesophageal echocardiography before attempting cardioversion. There are occasions when a patient with chronic atrial fibrillation presents in shock from a rapid ventricular rate. Synchronized cardioversion may be required in this setting despite the risks of an embolic event. However, cardioversion should not be performed if a known ventricular thrombus is present or if the ventricular rate is less than 130 beats per minute. If possible, the decision to cardiovert such a patient should be made in consultation with a cardiologist.

Pearls, pitfalls and myths

The following constitute important pitfalls related to the management of the patient in shock:

- Failure to recognize and aggressively treat early signs of shock, before hypotension develops;
- Failure to provide early ventilatory support to the hemodynamically compromised patient;
- Inadequate fluid resuscitation of the volume-depleted patient before initiating vasoactive infusion;
- Delay in administration of empiric broad-spectrum antibiotics and source control in septic shock;
- Failure to continuously monitor hemodynamic parameters (Table 6.6) as a guide to fluid therapy and vasoactive infusion titration;
- Improper selection of vasoactive agents;
- Reliance on pulse oximetry as an index of SaO_2 during periods of hypoperfusion, severe hypoxemia, or the presence of a hemoglobinopathy.

Table 6.6 Normal hemodynamic parameters

CVP	2–6 cmH_2O
PAOP	8–12 cmH_2O
CO	3.8–7.5 L/min (approximate for normal size adult)
CI	2.4–4.0 L/min/m^2
SVR	800–1400 dyne/sec/cm^5 (approximate for normal size adult)
SVRI	1600–2400 dyne/sec/m^2/cm^5

CO: cardiac output; CI: cardiac index; CVP: central venous pressure; PAOP: pulmonary artery occlusion pressure; SVR: systemic vascular resistance; SVRI: systemic vascular resistance index.

Acknowledgment

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7 Traumatic injuries

David Manthey, MD and Kim Askew, MD

Scope of the problem

Traumatic injuries account for about 36% of emergency department (ED) visits. In 2006, US EDs evaluated and treated more than 42.5 million people for injuries. The most frequent mechanism of injury was unintentional falls (20.3%), followed by motor vehicle collisions (9.5%). More than 179,000 people died as a result of traumatic injuries. Of these deaths, approximately 43,000 were the result of motor vehicle crashes, 31,000 from firearms, and 21,000 from falls.

Patients with severe or life-threatening traumatic injuries may present to the ED at any time of day, either immediately following their injury or in a delayed fashion. They may arrive by ambulance, having benefited from prehospital care and advanced notification, or be “dropped off” by a friend or family member. Emergency physicians must be skilled at the initial evaluation and treatment of these patients.

Peaks of death

Death from traumatic injury tends to occur during one of three distinct time frames following the injury. The first “peak of death” occurs within seconds to minutes of the injury, typically resulting from devastating injuries to the central nervous system, heart, or major vessels. Very few of these patients can be saved.

The second peak of death occurs minutes to hours following the injury. Deaths during this period occur as a result of major head, chest, abdominal or pelvic injuries, as well as injuries associated with significant blood loss. During the “golden hour” of trauma care, the rapid transportation, identification and treatment of these injuries is essential to preserving life. These injuries require emergent stabilization and generally surgical intervention.

The third peak of death occurs days to weeks after the original injury. This is most often the result of sepsis or multiorgan failure.

Primary survey

Initial evaluation of the trauma patient begins with the primary survey:

- Airway with cervical spine control
- Breathing
- Circulation with hemorrhage control
- Disability
- Exposure and environmental control

This is a systematic approach to the assessment and simultaneous treatment of life-threatening traumatic injuries.

It is essential that traumatic life- or limb-threatening injuries are treated at the time they are identified, not after the entire examination is completed. Obtaining a detailed patient history and evaluation for secondary (non–life-threatening) injuries may be deferred until the secondary survey. This is often difficult because some secondary injuries are very dramatic, and human nature draws us to them.

Airway with cervical spine control

Assessment

The airway should be assessed immediately to make certain that it is both patent and protected. If there is a risk that the patient will not be able to maintain his or her airway, early intervention must be considered. Establishment of a secure airway takes precedence over the remainder of the trauma evaluation.

Have the patient speak to assess patency of the airway. Listen for stridor and/or dysphonia, as both serve as indicators of upper airway injury requiring rapid intervention.

Assess the patient for agitation, obtundation and cyanosis. These findings may be indirect signs that the patient is not adequately oxygenating or ventilating, resulting in hypoxia or hypercarbia.

Carefully remove the front of the cervical collar (while providing spinal stabilization) to look for evidence of penetrating injuries, subcutaneous emphysema, or an expanding hematoma of the anterior neck. Determine if the trachea is midline at the sternal notch, as deviation of the trachea may be associated with a local hematoma or tension pneumothorax.

Examine the patient for oropharyngeal bleeding and swelling. The gentle use of a tongue blade may facilitate this task. Ensure that the patient can swallow and handle secretions.

Some trauma patients arrive at the ED after intubation in the field. Do not assume that the airway is secure. Correct endotracheal tube (ETT) placement should be confirmed by a combination of the direct visualization of the ET tube passing through the vocal cords, the presence of normal oxygen saturation, and detection of end-tidal carbon dioxide (ETCO$_2$).

Assume injury to the cervical spine in any patient with the following findings: (1) multi-system or major trauma; (2) altered level of consciousness; (3) blunt injury above the clavicles; (4) concerning mechanism of injury; (5) neck

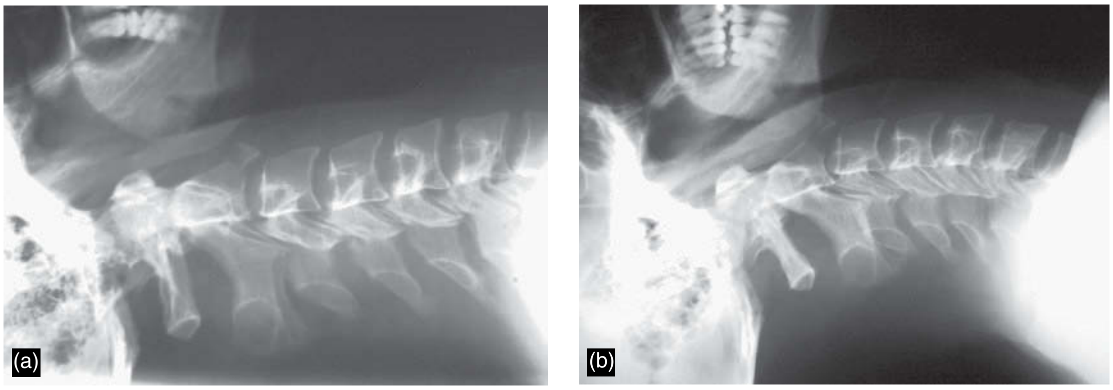

Figure 7.1
(a) A patient with an extension teardrop fracture of the vertebral body of C2. (b) Inadvertent hyperextension of the patient's neck could lead to subluxation of the vertebral bodies and injury to the spinal cord. Courtesy: Michael Zucker, MD.

pain, ecchymosis or deformity; and (6) neurologic deficits. A normal neurologic exam does not exclude cervical spine injury.

Treatment

The tongue remains the most common reason for airway obstruction in an unconscious patient. This obstruction may be treated by manual maneuvers such as the chin lift or jaw thrust, or with devices such as the nasopharyngeal (semiconscious patient) or oropharyngeal (unconscious patient) airway. If a cervical spine injury is suspected or the patient is unconscious, the neck should not be flexed, extended or rotated (Figure 7.1). The airway should be kept clear of debris and vomit by a suction device or a manual sweep.

A trauma patient should be intubated for any of the following reasons:

- Apnea or inadequate ventilation
- Protection from aspiration
- Impending or suspected airway compromise
- Hypoxia despite supplemental oxygen
- Paralysis required for safe evaluation of a combative patient
- Closed head injury with Glasgow Coma Scale (GCS) ≤ 8

A complete approach to controlling a patient's airway is described in Chapter 2. An organized approach in a stepwise pattern should utilize one or more of the following methods:

1. *Chin lift/jaw thrust/nasopharyngeal airway/oropharyngeal airway:* The use of adjunctive airways and simple maneuvers to lift the tongue out of the pharynx often allows ventilation of the patient until a definitive airway can be established.
2. *Bag-mask ventilation (BMV):* Every clinician should be skilled at BMV, which allows ventilation of an apneic patient or patient with respiratory distress until a definitive airway can be established. Providing a good mask seal and ensuring that the tongue does not obstruct the hypopharynx are essential components of effective BMV.
3. *Intubation:* ET intubation can be performed by direct or video laryngoscopy, over a bougie or endoscope, or through a laryngeal mask airway (LMA). Direct laryngoscopy is safe in the trauma patient when performed with in-line stabilization to protect the cervical spine. Rapid sequence intubation (RSI) may facilitate intubation of a patient without requiring BMV. However, prior to paralyzing the patient, it is important to assess for a difficult airway and ensure that the patient can be effectively bag-mask ventilated should the intubation prove difficult or impossible.
4. *Transtracheal jet ventilation:* When intubation fails (especially in children < 10–12 years of age), a large-bore cannula placed through the cricothyroid membrane will temporarily allow oxygenation or jet ventilation of the patient.
5. *Surgical cricothyroidotomy:* A surgical airway may be necessary when ET intubation either fails or is not feasible. This procedure involves incising the cricothyroid membrane to allow placement of an ET or tracheostomy tube directly into the trachea in the patient greater than 8 years of age (Figure 7.2).

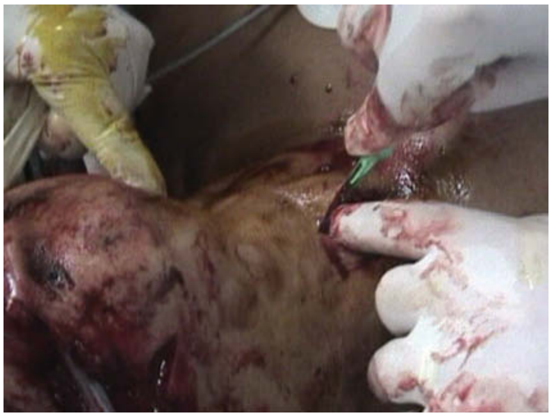

Figure 7.2
Surgical cricothyroidotomy. Courtesy: Mel Herbert, MD.

Breathing

Assessment

Evaluate the patient's breathing to determine how well the patient is oxygenating and ventilating. Employ a pulse oximeter to assess oxygenation and, if available, a quantitative end-tidal CO_2 monitor to assess ventilation. An arterial blood gas, drawn after the primary survey, will assess both oxygenation and ventilation, and provides the patient's acid–base status, which often reflects the adequacy of resuscitation.

Auscultate the lungs for bilateral symmetric breath sounds. The lack of breath sounds on one side may indicate a pneumothorax or hemothorax. However, the presence of normal breath sounds may miss a moderate (30%) pneumothorax and a large (800 mL) hemothorax. Percussion of the chest and ultrasound may help differentiate a pneumothorax from a hemothorax. The clinician should search for signs of tension pneumothorax, such as tracheal deviation away from the affected side, distended neck veins, decreased breath sounds on the affected side, and hypotension (Figure 7.3).

Observe the chest wall for symmetric rise as well as for paradoxical movement suggestive of flail chest (Figure 7.4). Flail chest is caused by the fracture of three or more ribs in two or more places, causing a free-floating segment that moves inward with inspiration and outward with expiration.

Palpate the entire thorax (anterior and posterior) for crepitus and rib tenderness. Crepitus suggests an underlying pneumothorax, whereas rib tenderness alerts the physician to a possible rib fracture and underlying pulmonary contusion.

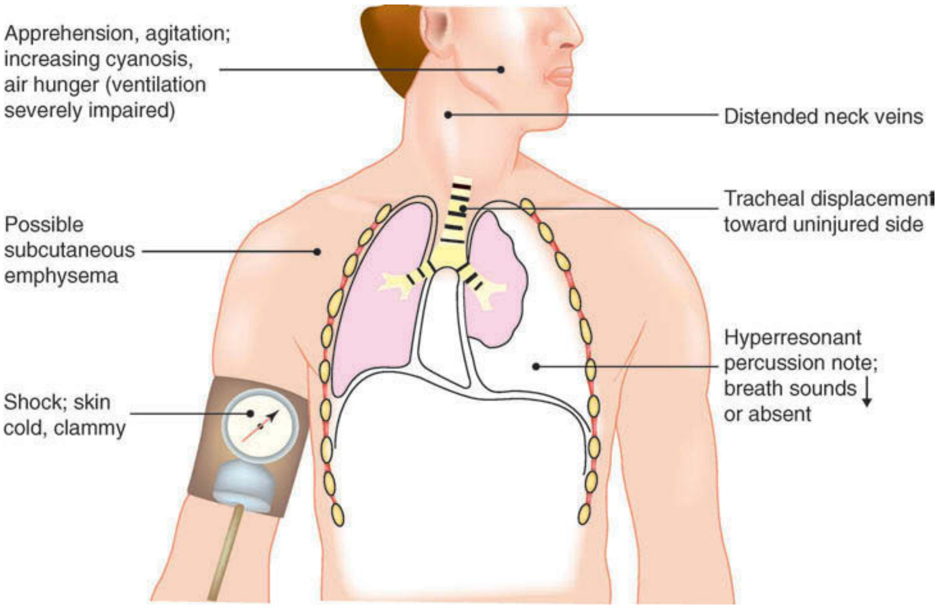
Apprehension, agitation; increasing cyanosis, air hunger (ventilation severely impaired)
Distended neck veins
Tracheal displacement toward uninjured side
Possible subcutaneous emphysema
Hyperresonant percussion note; breath sounds ↓ or absent
Shock; skin cold, clammy

Figure 7.3
Tension pneumothorax. From Campbell, John E., *Basic Trauma Life Support for Advanced Providers*, 5th ed., Copyright 2004. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

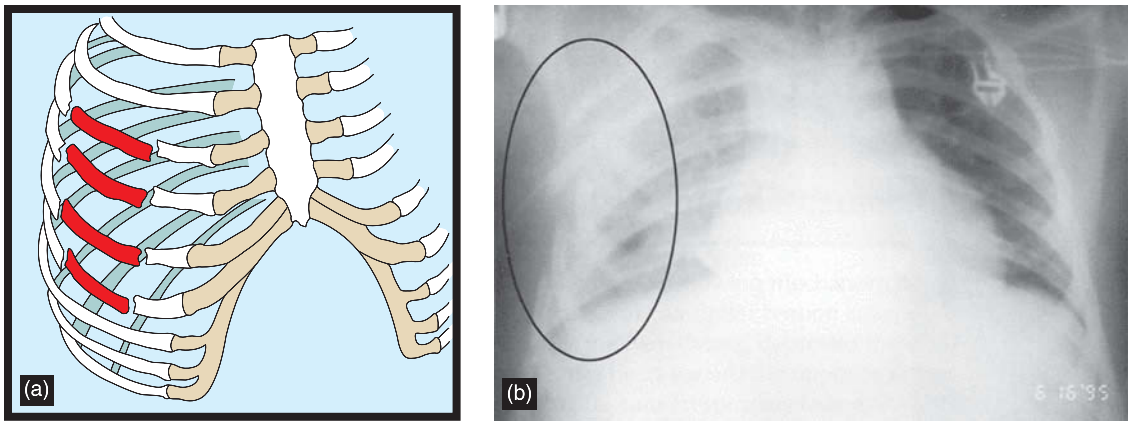

Figure 7.4
(a) Illustration of flail chest. (b) Chest X-ray showing flail chest with an underlying lung contusion. Reproduced from D. Mandavia, et al, *Color Atlas of Emergency Trauma*. Cambridge University Press, Cambridge, 2003.

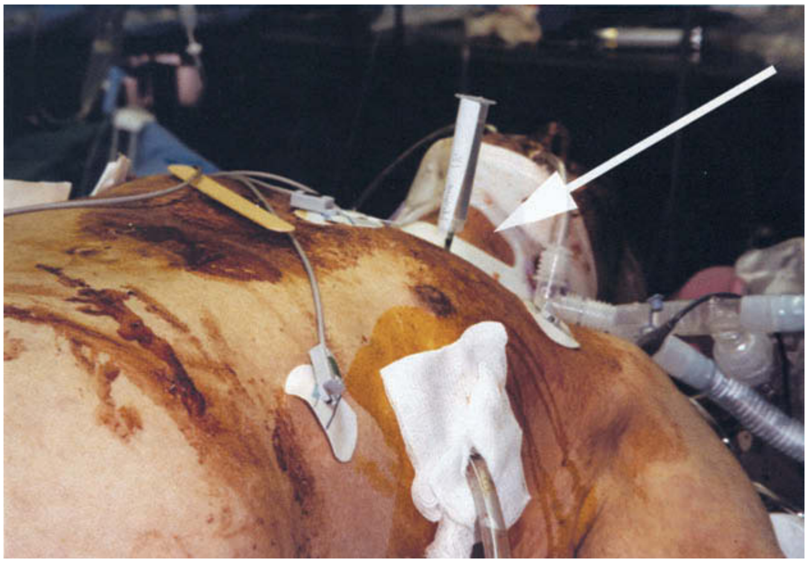

Figure 7.5
Needle thoracostomy (white arrow) for tension pneumothorax. Reproduced from D. Mandavia, et al, *Color Atlas of Emergency Trauma*. Cambridge University Press, Cambridge, 2003.

Look for an open (sucking) chest wound. If the chest wound is at least two-thirds the diameter of the patient's trachea, air can preferentially enter the pleural space through this chest wall defect instead of the trachea, resulting in no alveolar oxygen exchange.

Treatment

All trauma patients should receive supplemental high-concentration oxygen regardless of their oxygen saturation. Oxygen saturation may be monitored with a pulse oximeter if an appropriate waveform can be identified.

Life-threatening conditions affecting breathing include hypoxia, tension pneumothorax, open pneumothorax, massive hemothorax and flail segment.

Hypoxia should be treated with supplemental oxygen. Intubation should be performed if necessary to provide oxygen saturations above 90%. A diligent search for reversible causes of impaired oxygenation should occur.

Emergent treatment of a tension pneumothorax converts it to a simple pneumothorax. This can be accomplished by needle decompression (needle thoracostomy) using a 14-G catheter over needle (Figure 7.5). Insertion of the needle over the third rib (second intercostal space) in the mid-clavicular line results in a release of intrapleural air and the subsequent reversal of adverse hemodynamic effects. The catheter is left in place until a 36-French chest tube is promptly placed at the fourth intercostal space in the mid-axillary line (chest tube thoracostomy). The larger chest tube addresses the concomitant hemothorax that accompanies most traumatic pneumothoraces.

An open pneumothorax allows air to preferentially enter the thoracic cavity through the defect rather than the trachea. This results in significant hypoxia, increased work of breathing and hypercarbia. This wound should be treated with an air-occlusive dressing (such as a defibrillator pad or Vaseline gauze) leaving one corner untaped to produce a flutter valve (Figure 7.6). This type

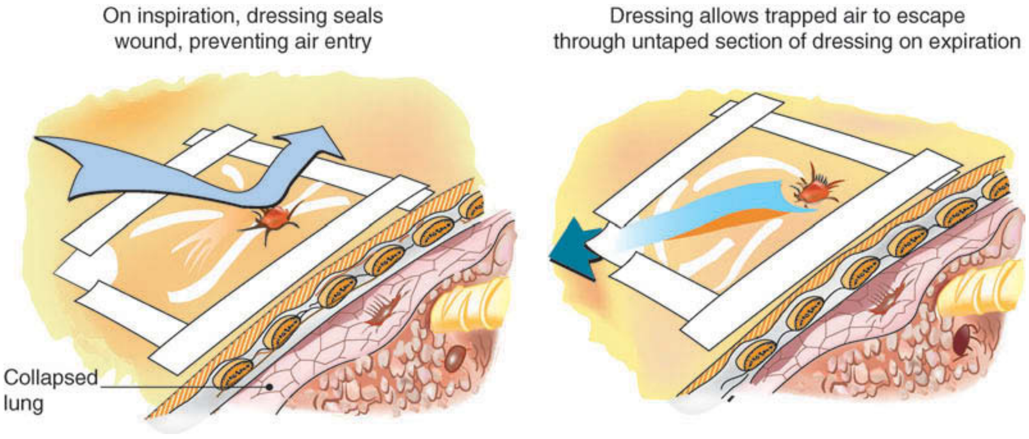
On inspiration, dressing seals wound, preventing air entry
Dressing allows trapped air to escape through untaped section of dressing on expiration
Collapsed lung

Figure 7.6
Treatment of an open pneumothorax. From Campbell, John E., *Basic Trauma Life Support for Advanced Providers*, 5th ed., Copyright 2004. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

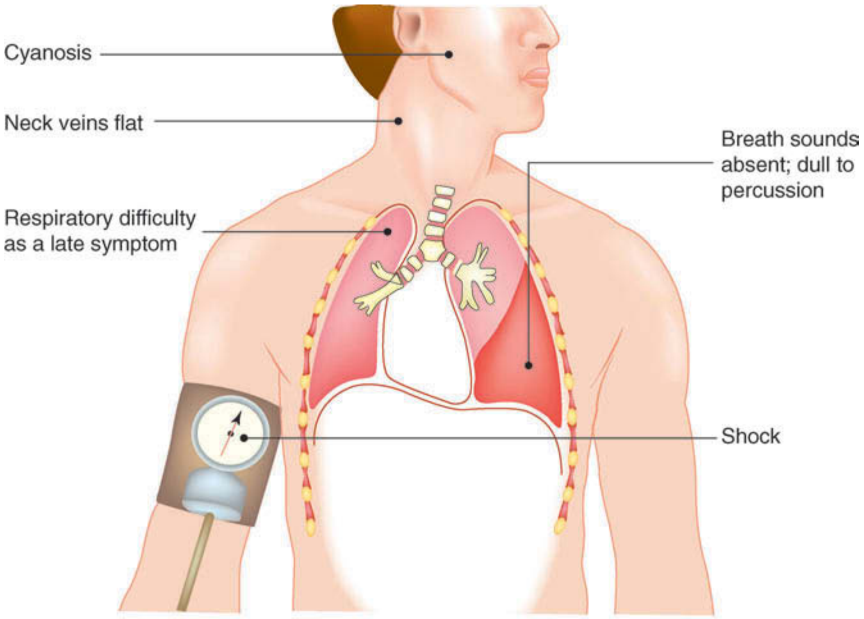
Cyanosis
Neck veins flat
Respiratory difficulty as a late symptom
Breath sounds absent; dull to percussion
Shock

Figure 7.7
Massive hemothorax. From Campbell, John E., *Basic Trauma Life Support for Advanced Providers*, 5th ed., Copyright 2004. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

of dressing will prevent the entrance of air into the pleural space during inhalation but allow the escape of intrapleural air during exhalation.

A massive hemothorax (Figure 7.7) is identified by more than 1,500 mL of blood (or greater than one-third the patient's blood volume) within the thoracic cavity. Initial treatment is evacuation of the blood by tube thoracostomy. An auto-transfuser will allow transfusion of the blood right back to the patient. Although continued bleeding (drainage of >200 mL/hr for 2–4 hours) is concerning, the patient's hemodynamic status and continued need for blood transfusions dictate the need for operative intervention (thoracotomy).

A flail segment occurs when three or more contiguous ribs are broken in two or more places. Paradoxical movement of this segment, restricted chest wall movement due to pain, and underlying pulmonary contusion may lead to hypoxia and ineffective ventilation. Prevention of over-hydration may avert additional alveolar capillary leakage within the injured lung. Intubation with positive pressure ventilation is often required to treat this injury. Pain control may be achieved by judicious use of narcotics, instillation of rib nerve blocks, and thoracic epidural anesthesia. Strapping, bulky dressings or ace bandaging are of no use and actually further restrict patient's breathing.

Circulation

Assessment

Shock is defined by inadequate organ perfusion and tissue oxygenation, not by a specific blood pressure measurement. A patient with a low blood pressure may continue to perfuse well, as evidenced by normal mentation, skin temperature and color. Alternatively, a "normal" blood pressure may be measured in a patient without adequate perfusion of their vital organs.

Hypovolemia, typically from hemorrhage, is the most common cause of shock in trauma victims. Most preventable trauma deaths result from the failure to recognize and adequately treat hemorrhagic shock. Always assume that hypovolemic shock is present, and treat until proven otherwise. Familiarity with the classes of hypovolemic shock is important, as they are based on volume of blood loss and help guide therapy (Table 7.1). Other causes of shock in the trauma patient include neurogenic shock (e.g., spinal cord injury) and obstructive shock (e.g., cardiac tamponade, tension pneumothorax). Cardiogenic shock may be the initial cause of a traumatic injury, but is rarely the result of one.

When assessing a trauma patient for the presence of shock, start with the patient's mental status. After addressing airway and breathing issues, signs such as confusion, restlessness, combativeness or unconsciousness strongly suggest shock. Other causes of altered mental status in the trauma patient include head injury or intoxication.

Continuously assess the patient's vital signs. The earliest manifestations of shock include tachycardia and cutaneous vasoconstriction. The patient's pulse may be elevated due to hypovolemia or secondary to pain and stress. The pulse may also be misleadingly normal secondary to age, medications (e.g., beta- or calcium channel blockers), neurogenic shock, or pacemaker.

Calculate the pulse pressure, which is the difference between the systolic and diastolic blood pressure. A narrowed pulse pressure reflects a fall in systolic pressure (due to a drop in stroke volume) and a rise in diastolic

Table 7.1 Estimated blood loss, signs and treatment for classes of shock

Class of shock	Blood loss	Signs	Treatment
Class I	0–750 mL (<15% of blood volume)	Tachycardia	IV crystalloid fluids
Class II	750–1,500 mL (15–30% of blood volume)	Tachycardia Tachypnea Pulse pressure narrows	IV crystalloid fluids
Class III	1,500–2,000 mL (30–40% of blood volume)	Tachycardia (HR >120 beats/min) Tachypnea (RR 30–40 breaths/min) Narrowed pulse pressure Decreased systolic blood pressure (<90 mmHg) Decreased urinary output Decreased mental status Decreased capillary refill	IV crystalloid fluids, packed RBCs
Class IV	2,000 mL (>40% of blood volume)	Tachycardia (HR >140 beats/min) Tachypnea (RR >35 breaths/min) Absent pulse pressure Markedly decreased systolic blood pressure No urinary output Confused to lethargic Markedly decreased capillary refill	IV crystalloid fluids simultaneously with packed RBCs

HR: heart rate; RBCs: red blood cells; RR: respiratory rate.

Adapted from the Committee on Trauma, American College of Surgeons. *Advanced Trauma Life Support Student Manual*, 8th ed. American College of Surgeons, Chicago, IL, 2008.

pressure (due peripheral vasoconstriction). Both are compensatory mechanisms to maintain perfusion.

The presence of hypotension suggests a significant shock state. Children and healthy adults can maintain their blood pressure even in the face of severe blood loss; however, other signs of shock are usually present.

Examine the patient's extremities for delayed capillary refill time (>2 sec), which may reflect decreased peripheral perfusion. Cool or pale extremities without associated extremity injury suggest shock.

Always compare peripheral and central pulses. If the central pulses are markedly stronger than the peripheral pulses, this may be a sign of peripheral vasoconstriction in order to preserve preload and maintain cardiac output.

Evaluate the patient's jugular veins. Flat jugular veins suggest hypovolemia. Full neck veins are normal in the recumbent patient. Distended jugular veins and impending shock suggest an obstructive process such as cardiac tamponade (Figure 7.8), tension pneumothorax, or cardiogenic shock.

Assess the patient's urinary output. It should be at least 0.5 mL/kg/hr in the adult patient, 1 mL/kg/hr in the pediatric patient and 2 mL/kg/hr in children <1 year of age. Decreased urine output may reflect poor renal perfusion secondary to continued hypovolemia and under-resuscitation.

When evidence of shock is present, extended-focused assessment with sonography for trauma (e-FAST) can identify hemoperitoneum (requiring an exploratory laparotomy), pericardial effusion (suggesting pericardial tamponade), or pneumo/hemothorax (see Appendix E).

The assessment of a patient's circulatory status is an ongoing process. When resuscitating the patient with crystalloid, it is important to determine how the patient responds to each fluid challenge.

Treatment

During assessment of circulation, one must stop all obvious external bleeding. Direct pressure or a compression dressing is sufficient in most instances. In some cases, placing a hemostatic figure-of-eight stitch over the bleeding area may be required. Blind probing or clamping deep within a wound should be avoided.

Venous access is essential for the administration of isotonic fluid and blood (if necessary). Two large-bore intravenous (IV) catheters (16 G or larger) are preferred. Short, large-caliber peripheral IVs allow the rapid infusion of large volumes of fluid. If the patient's condition prevents placement of peripheral IVs, a central venous catheter may be placed in the subclavian, internal jugular, or femoral vein. A peripheral venous cutdown may be performed on the saphenous vein. Intraosseous catheters provide rapid vascular access as an alternative approach if peripheral IV access fails.

Fluid resuscitation should be given rapidly, up to a predetermined amount (usually 2 L). A fluid challenge of 2 L of crystalloid replaces 3:1 the losses (750 mL) associated with class I shock. The patient's hemodynamic response should be evaluated after this initial bolus. Patients who respond quickly may not need further fluids or blood, as they may have limited initial blood loss without ongoing hemorrhage. Patients with more than 750 mL of initial blood loss, but no ongoing blood loss, may have an incomplete response to the fluid resuscitation. Patients who have ongoing blood loss will respond

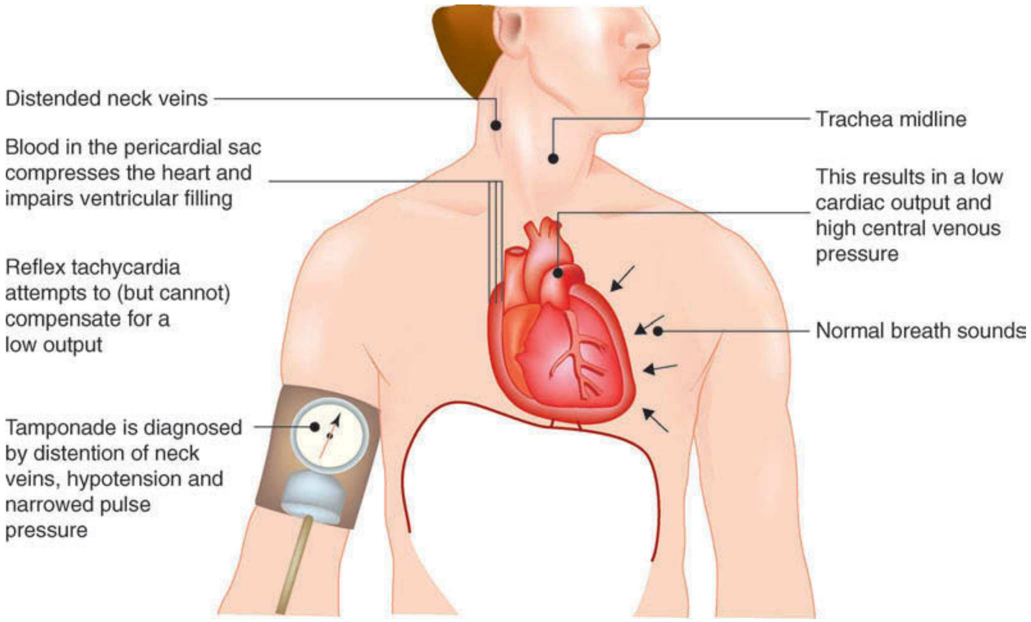
Distended neck veins
Blood in the pericardial sac compresses the heart and impairs ventricular filling
Reflex tachycardia attempts to (but cannot) compensate for a low output
Tamponade is diagnosed by distention of neck veins, hypotension and narrowed pulse pressure
Trachea midline
This results in a low cardiac output and high central venous pressure
Normal breath sounds

Figure 7.8
Cardiac tamponade. From Campbell, John E., *Basic Trauma Life Support for Advanced Providers*, 5th ed., Copyright 2004. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

only transiently, requiring further resuscitation with fluids and blood products. These patients require rapid identification of the source of their blood loss. Patients who do not respond at all to the initial bolus have either massive blood loss or marked continued bleeding and require additional resuscitation with blood and fluids. An emergent trip to the operating room (OR) may be required to diagnose and control the source of bleeding.

Blood products are reserved for patients who remain hemodynamically unstable or who have ongoing blood loss. When there is no time to type and screen a patient, type O blood should be utilized. For women of or entering childbearing age, use Rh-negative blood. When possible, administer ABO type-specific and Rh-compatible blood, which is usually available 15 minutes after the blood bank receives the type and screen specimen. Type and cross-matched blood is best for avoiding incompatibility reactions but usually requires over an hour to obtain.

Finally, consider other causes for hemodynamic compromise, such as neurogenic or obstructive shock, which require alternate therapeutic approaches. Depending on the etiology of the shock state, the physician may utilize other procedures, such as:

1. Needle decompression followed by tube thoracostomy for tension pneumothorax
2. Needle pericardiocentesis (under ultrasound guidance) for cardiac tamponade
3. Pressors and fluid resuscitation for neurogenic shock
4. Traction for femur fractures
5. Circumferential pelvic binding, external fixation, or pelvic angiography with embolization of bleeding vessels for the treatment of displaced pelvic fractures

An ED thoracotomy is indicated for penetrating chest trauma and loss of vital signs within a few minutes of arriving at or within the ED. This heroic procedure should only be performed if the hospital has the facilities and staff to address the injury. A thoracotomy allows for definitive treatment of pericardial tamponade, repair of a cardiac laceration, cross-clamping the aorta to prevent ongoing blood loss inferior to the diaphragm, and clamping the pulmonary arteries.

Often the patient may leave the ED for the OR during the circulation assessment portion of the primary resuscitation. This may be necessary to obtain control of active bleeding within the chest or abdominal cavity.

Disability

Assessment

Assessment of the patient's disability (i.e., brief, directed neurologic exam) during the primary survey should address the following three areas: level of consciousness, pupillary examination, and movement of extremities. It is important to assess neurologic function prior to pharmacologic paralysis of the patient as part of rapid sequence intubation (RSI).

Assess the level of consciousness with the AVPU approach or the Glasgow Coma Scale (GCS). AVPU relates to the patient's level of response: the patient may be Alert, respond to Voice or Pain, or remain Unresponsive.

The GCS (Table 7.2) is used to follow the patient's neurologic status, guide therapy and communicate with consultants. Scores range from a minimum of 3 to a maximum of 15, with a score of 8 or less indicating severe head injury. Any drop in the GCS should prompt re-evaluation, specifically for rising intracranial pressure.

Table 7.2 Glasgow Coma Scale

Eye opening		
Spontaneous	4	Reticular activating system intact (though patient may not be aware)
To verbal command	3	Opens eyes when told to do so
To pain	2	Opens eyes in response to pain
None	1	Does not open eyes to any stimuli
Verbal response		
Oriented – converses	5	Aware of self and environment; oriented to person, place and time
Disoriented – converses	4	Organized and well articulated, but disoriented to person, place, situation, or time or asks repetitive questions.
Inappropriate words	3	Random recognizable words
Incomprehensible	2	Moaning, vocalized sounds with no recognizable words
No response	1	No response (Use I for intubated)
Motor response (best response from any extremity)		
Obeys verbal commands	6	Readily moves limbs following instructions
Localizes to painful stimuli	5	Moves limb in an effort to remove painful stimulus
Flexion withdrawal	4	Pulls away from pain in flexion
Abnormal flexion	3	Decorticate rigidity
Extension	2	Decerebrate rigidity
No response	1	Hypotonic, flaccid; suggests loss of medullary function or concomitant spinal cord injury

Adapted from Marx JA (ed.). *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 7th ed. Mosby, St. Louis, MO, 2010.

The pupil examination should look for symmetry and reactivity to light. A dilated, unreactive ("blown") pupil in a comatose patient suggests transtentorial intracranial herniation leading to unilateral compression of the third cranial nerve. Disconjugate gaze may be associated with various etiologies of coma.

Assessment of movement in all extremities is a gross evaluation of spinal cord function, not peripheral nerve function. It is more important to judge symmetry and strength in all extremities than isolated peripheral nerve function.

Treatment

The two most dangerous insults to the traumatized brain, hypoxia and hypotension, should be addressed during the initial evaluation and resuscitation. Any patient with a GCS ≤ 8 should be intubated.

In cases of neurologic deterioration or lateralizing neurologic signs, mannitol and controlled hyperventilation to a partial pressure of carbon dioxide (PCO_2) of 35 mmHg may be employed as temporizing measures to reduce intracranial pressure. Other therapies to consider in the severely brain-injured patient include anticonvulsants, deep sedation, and elevating the head of the bed to 30°. Neurosurgical procedures such as operative craniotomy, skull trephination with burr hole placement (Figures 7.9a and b), or intraventricular pressure monitor placement are often required.

The cervical collar should be maintained until a cervical spine injury has been excluded. However, once the stability of the spine has been assessed, the patient may be carefully log rolled off the spine board to prevent skin breakdown and minimize patient discomfort.

For patients with acute spinal cord injuries, rapid IV administration of high-dose steroids has been recommended, although this treatment remains controversial. Early discussion with neurosurgical consultants is recommended.

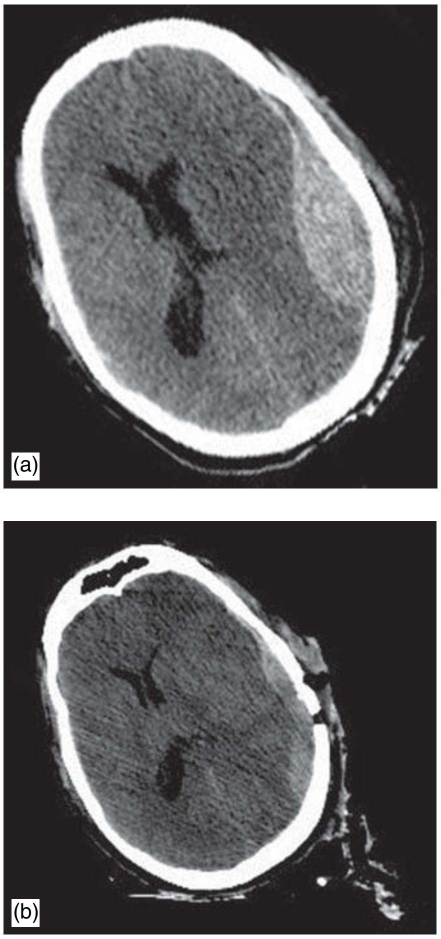

Figure 7.9
(a) Epidural hematoma. (b) Evacuation of the epidural hematoma following burr hole placement in the ED. Courtesy: Damon Kuehl, MD.

Exposure and environmental control

Assessment

Fully undress the victim from head to toe to allow a complete assessment. Look under collars and splints, in the axilla, and under skin folds; log roll the patient to examine the back and buttocks. Identify and treat any active sites of bleeding. Failure to completely expose the patient may result in missing a significant traumatic injury, such as a gunshot or stab wound (Figure 7.10).

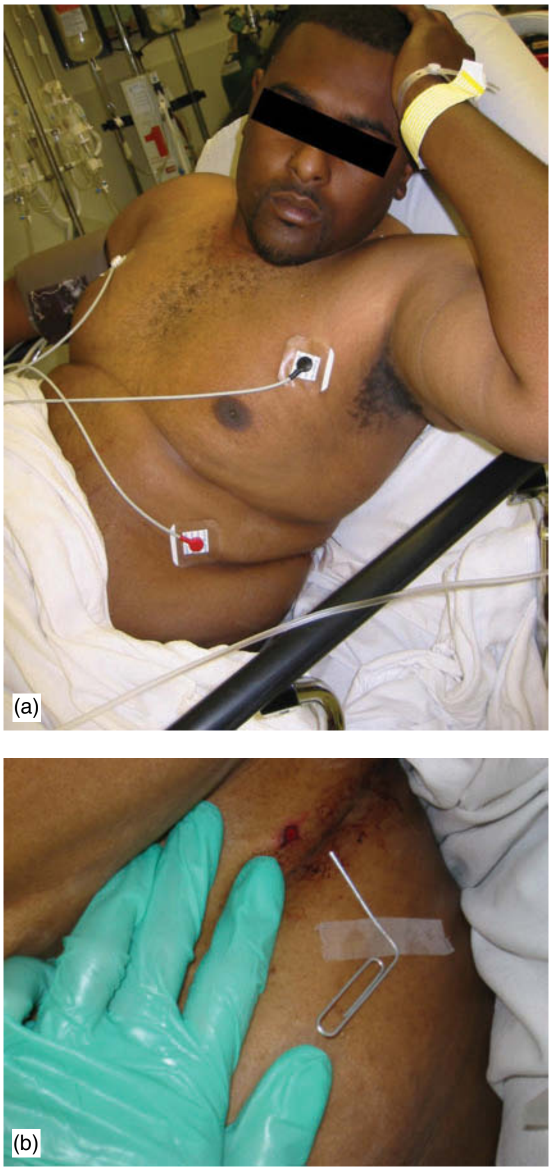

Figure 7.10
(a) A patient with a suspected gunshot wound. The initial physical examination did not reveal the injury, delaying definitive treatment. (b) The gunshot wound was later located under the patient's skin fold. Courtesy: Clement Yeh, MD.

Treatment

Remove all wet or contaminated clothing. If the patient has been in an industrial or chemical accident, decontamination is critical for patient care. It is also critical that the medical staff protect themselves from exposure, morbidity and incapacitation.

Keep the patient warm by raising the temperature of the resuscitation room, applying warm blankets, ventilating with warm humidified air, and administering warmed IV fluids. Hypothermia in trauma patients is associated with increased mortality, and should be prevented. The patient's chance of survival may drop with every degree drop in core temperature.

Secondary survey

This detailed head to toe examination is initiated only after life-threatening injuries have been evaluated and treated during the primary survey. At this time, multiple other evaluations may occur, including trauma radiographs and laboratory studies. Although there are a multitude of items to address in each anatomical area, what follows is a review of items specific to trauma.

1. Head, eyes, ears, nose and throat (HEENT)
   - **(a)** Assess for depressed skull fractures by careful palpation. Impaled foreign bodies and bone fragments should not be manipulated.
   - **(b)** Look for lacerations that will require repair. Unattended scalp lacerations can bleed vigorously.
   - **(c)** Assess for evidence of a basilar skull fracture by identifying the presence of Battle sign (ecchymosis over the mastoid) (Figure 7.11), Raccoon eyes (ecchymosis around the eyes) (Figure 7.12), or hemotympanum (blood behind the eardrum) (Figure 7.13). Look for a cerebrospinal fluid (CSF) leak manifested by rhinorrhea or otorrhea.

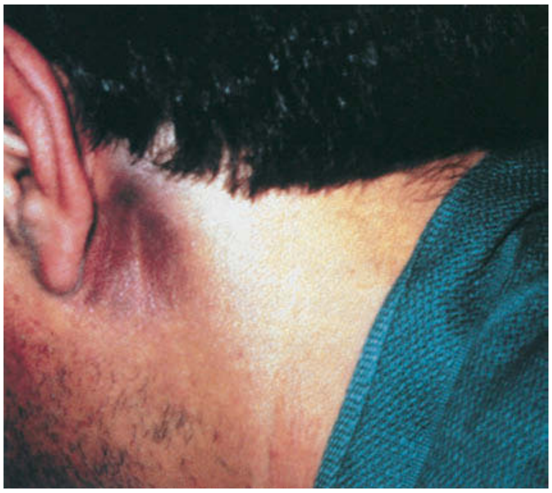

Figure 7.11
Battle sign. Reproduced from D. Mandavia et al, *Color Atlas of Emergency Trauma*. Cambridge University Press, Cambridge, 2003.

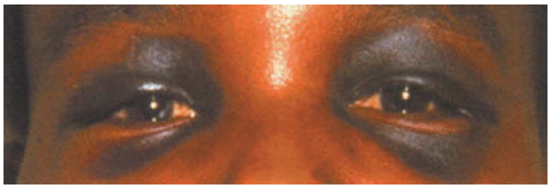

Figure 7.12
Raccoon eyes due to a frontobasilar skull fracture. Reproduced from D. Mandavia et al, *Color Atlas of Emergency Trauma*. Cambridge University Press, Cambridge, 2003.

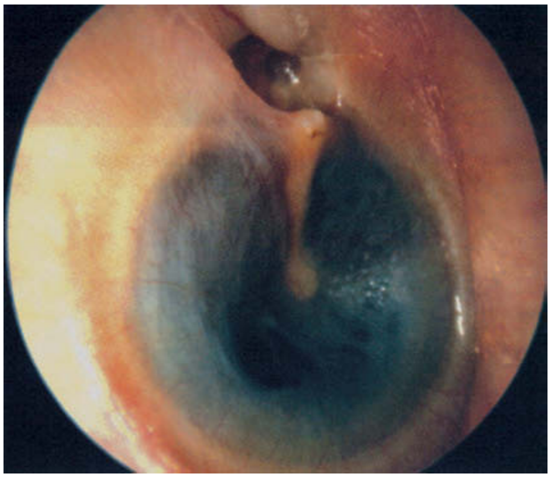

Figure 7.13
Hemotympanum. Reproduced from D. Mandavia et al, *Color Atlas of Emergency Trauma*. Cambridge University Press, Cambridge, 2003.

- **(d)** Assess for facial injury and stability by palpating and attempting movement of the facial bones. Severe facial fractures can lead to airway compromise and may alter the approach to the airway. Malocclusion of the teeth may indicate a mandible fracture (Figure 7.14). Dental trauma should also be identified (see Chapter 20).
- **(e)** Determine visual acuity and assess pupillary size and function. Assess the eye for abrasions, lacerations and signs of internal damage, such as hyphema.
- **(f)** Examine the nasal septum for a hematoma, which, if untreated, may lead to an abscess or nasal cartilage necrosis.

2. Cervical spine/neck
  - **(a)** Palpate the cervical spine and identify areas of tenderness, swelling or step-off deformity.
  - **(b)** Look for penetrating injuries within the three separate zones of the neck. Zone I lies inferior to the cricoid cartilage, zone II extends from the cricoid cartilage inferiorly to the angle of the jaw superiorly, and zone III lies superior to the angle of the jaw to the base of the skull.
  - **(c)** Evaluate for subcutaneous emphysema, which may be associated with laryngotracheal injury or pneumothorax.

3. Chest
  - **(a)** Palpate the sternum, clavicles and ribs for tenderness, deformity, or crepitus. The presence of subcutaneous emphysema suggests an underlying pneumothorax.
  - **(b)** Look for bruising or deformity to suggest an injury to the underlying lung.

4. Abdomen
  - **(a)** Assess for any distention, tenderness, rebound or guarding. Two common sources of blood loss in patients with abdominal trauma are injuries to the spleen and liver.
  - **(b)** Flank ecchymosis may suggest a retroperitoneal bleed.
  - **(c)** The presence of a “seat belt sign” is correlated with an eight-fold higher relative risk of intraperitoneal injury (Figure 7.15).
  - **(d)** Reliable assessment of the abdomen may be compromised by the presence of altered mental status, intoxication with alcohol or illicit drugs, or painful distracting injuries.

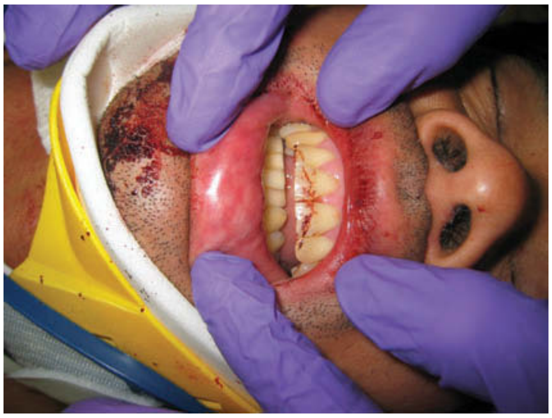

Figure 7.14
Malocclusion associated with a mandible fracture. Courtesy: S.V. Mahadevan, MD.

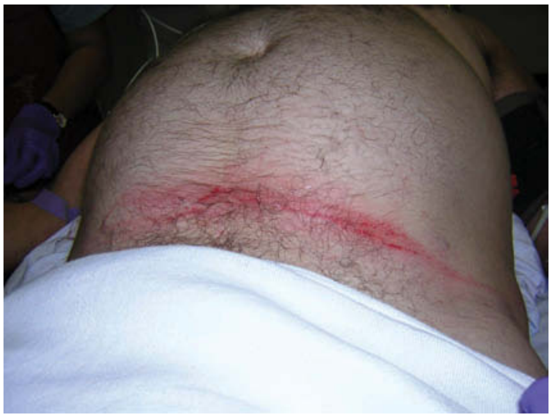

Figure 7.15
Seat belt sign. Courtesy: Jo Feldman, MD.

5. Back
   - **(a)** Log roll the patient with assistance while maintaining spinal alignment. Palpate the entire spine for any spinous process tenderness or deformity.
   - **(b)** Assess for hidden wounds in the axilla, under the cervical collar, and in the gluteal and perineal regions.
6. Pelvis
   - **(a)** In order to assess the stability of the pelvis, the physician may *gently* employ anterior-posterior compression of the anterior superior iliac spines, lateral compression of iliac crests, and cranial-caudal distraction of opposite iliac crests. Vigorous manipulation of the bony pelvis may exacerbate bleeding from a pelvic fracture or the venous plexus.
   - **(b)** Palpate the symphysis pubis for pain, crepitus, or widening.
   - **(c)** Pelvic fractures can be responsible for as much as 4–6 L of occult blood loss.
7. Perineum
   - **(a)** Evaluate the perineum for ecchymosis, suggestive of a pelvic fracture or urethral disruption.
8. Urethra
   - **(a)** Look for blood at the urethral meatus to assess for possible urethral disruption before placing a urinary catheter.
9. Rectum
   - **(a)** A rectal examination is required to assess sphincter tone as part of the neurologic assessment.
   - **(b)** A high-riding prostate suggests disruption of the membranous urethra. A urinary catheter should not be placed in this circumstance.
   - **(c)** A pelvic fracture may cause a rectal wall laceration and rectal bleeding.
   - **(d)** Gross blood on digital rectal examination suggests a bowel injury.
10. Vagina
    - **(a)** A vaginal examination should be performed in female patients to assess for palpable fractures, vaginal lacerations and blood within the vaginal vault.
11. Extremity examination
    - **(a)** Re-check the vascular status of each extremity, including pulses, color, capillary refill and temperature.
    - **(b)** Inspect every inch, palpate every bone, and check the range of motion of all joints. Assess for deformity, crepitus, tenderness, swelling and lacerations.
    - **(c)** Unstable fractures or those associated with neurovascular compromise should be reduced immediately. Splinting and/or traction of fractured bones can provide hemostasis, prevent further injury, and enhance patient comfort.
    - **(d)** Femur fractures can result in as much as 2 L of occult blood loss.
12. Neurologic
    - **(a)** At this time, a complete neurologic examination should be done. This includes a repeat GCS score, reevaluation of the pupils, a cranial nerve examination, a complete sensory and motor examination, testing of the deep tendon reflexes, and an assessment of the response to plantar stimulation.

History

Historical information should be obtained from all available sources, including the patient, bystanders, EMS and rescue personnel.

Where and how were you injured (shot, struck)? Where are you hurting?

An understanding of the mechanism of injury may provide clues to the type(s) of injuries seen in trauma patients (Table 7.3). Significant injuries may occur without obvious external evidence of trauma. The cervical spine is a classic example.

Table 7.3 Mechanisms of traumatic injury and associated injuries

Mechanism	Possible traumatic injury
Steering column damage	Myocardial or pulmonary contusion
Sudden deceleration (fall, MVC)	Traumatic aortic disruption, immobile C7-T1 junction injury
Windshield star	Subdural hematoma, epidural hematoma, cervical spine injury
Rear impact, head turned to side	Jumped cervical facet, vertebral artery dissection
Side impact	Fractured hip
Seat belt sign, stab wound below the nipple or scapular tip	Intra-abdominal injury
Fall, landing on heels	Tibial plateau fracture, lumbar spine fracture, calcaneal fracture
Direct blow to head	Coup and contre-coup brain injuries
Blast injury	Air-containing body cavities most vulnerable
High kinetic energy missile (bullet)	Injury extends beyond bullet wound

MVC: motor vehicle collision.

Did you lose consciousness?

Loss of consciousness should raise concern for an intracranial injury.

What amount of blood loss occurred at the scene/en route?

Quantifying this amount may be difficult, but recognizing that the patient has already lost a significant amount of blood will guide therapy.

What was the temperature at the scene?

Assess the potential for hypothermia or hyperthermia.

What was the direction of impact?

This allows clinicians to ascertain the forces imposed upon the body and identify associated injuries, such as a jumped facet in the cervical spine.

What was the appearance of the vehicle?

This includes damage to the steering wheel, starring of the windshield, and intrusion of the door into the passenger compartment. A verbal report or photo allows an estimation of the amount of kinetic energy delivered to the patient (Figure 7.16).

What was your position in the car?

Knowledge of both damage to the car and the patient's position in the car allows the clinician to better ascertain what the patient's injuries might be.

What was the speed of the vehicle (if isolated collision) or vehicles? What types of vehicle(s) were involved?

Remember that energy equals mass times velocity squared, so identifying the mass and velocity allows determination of the amount of force transmitted. Larger cars, sport utility vehicles (SUVs) and trucks that ride higher off the ground generally protect the passenger more than small, light vehicles.

Did you use restraining devices?

Ask about the use of seat belts (including shoulder and lap, or lap-only restraints) and the deployment of air bags.

Did your vehicle roll over or stop with sudden impact? Was there a fatality at the scene?

Affirmative answers to any of these questions raise the likelihood of serious injury.

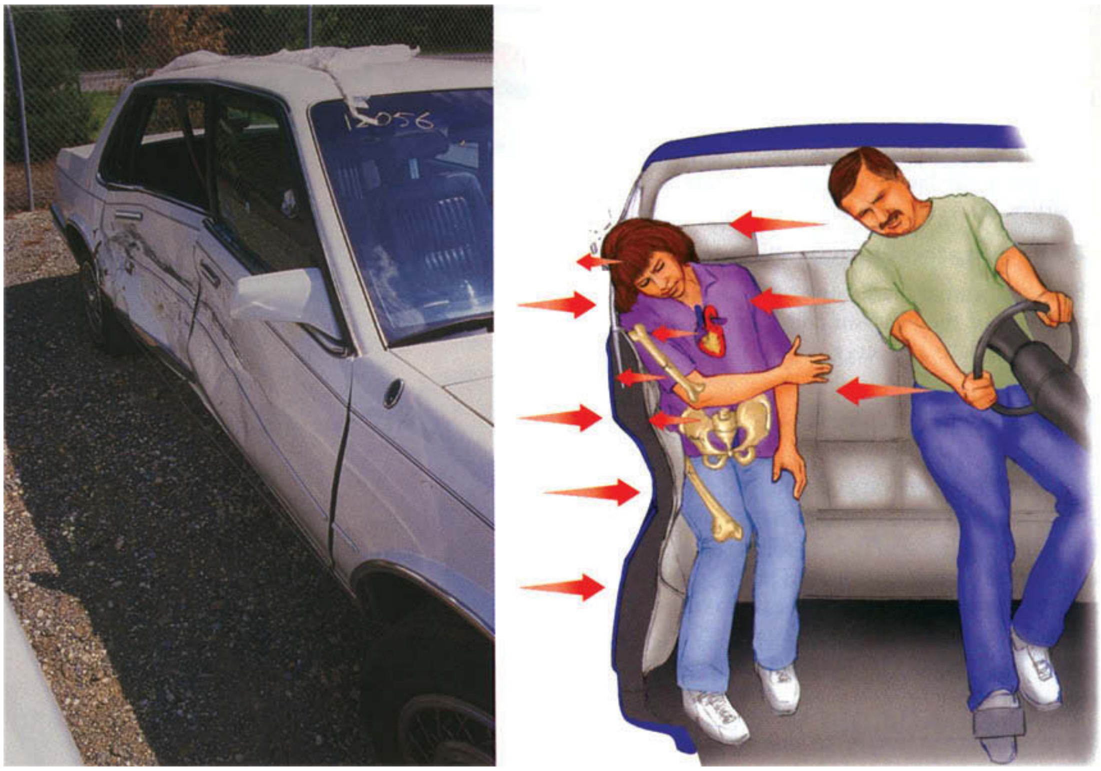

Figure 7.16
Side impact motor vehicle collision with passenger space intrusion. From Campbell, John E., *Basic Trauma Life Support for Advanced Providers*, 5th ed., Copyright 2004. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

Were you wearing a helmet?

For patients of motorcycle or bicycle crashes, this information is important given the amount of protection that helmets afford the brain. Evaluation of the helmet is also important to determine the amount of force distributed to the head.

How far did you fall and what did you land on?

Both the height of the fall and the hardness of the surface the patient struck are important in determining the likelihood of injury and the body parts injured.

What caused the fall or collision?

Did the patient have a seizure or syncope which caused the fall or collision? Was either preceded by chest pain or difficulty breathing? Was alcohol involved? Was this a suicide attempt?

Did an explosion occur?

What was the patient's distance from the blast? Blast injuries may occur from the primary blast force, secondary missiles, or due to tertiary impact against a hard surface (Figure 7.17).

How many shots were fired?

This question may help determine if you are missing an injury or if the wounds may represent two separate entrance wounds rather than a single wound (entrance and exit).

Do you know what type of weapon was used?

Although this is notoriously unreliable, knowledge of the type of weapon and/or bullet's velocity may help determine the injury pattern. The length and width of the blade in a stab wound may also assist with patient evaluation. Additionally, for stab wounds, the hand dominance and gender of the attacker may provide useful information if it is known.

What were you struck with?

Being struck with a bat or pipe versus a fist implies a greater magnitude of force applied to the tissue, suggesting the possibility of a larger amount of external and internal damage.

Was this a crush injury?

If so, ascertain the weight and force of the object that struck the patient.

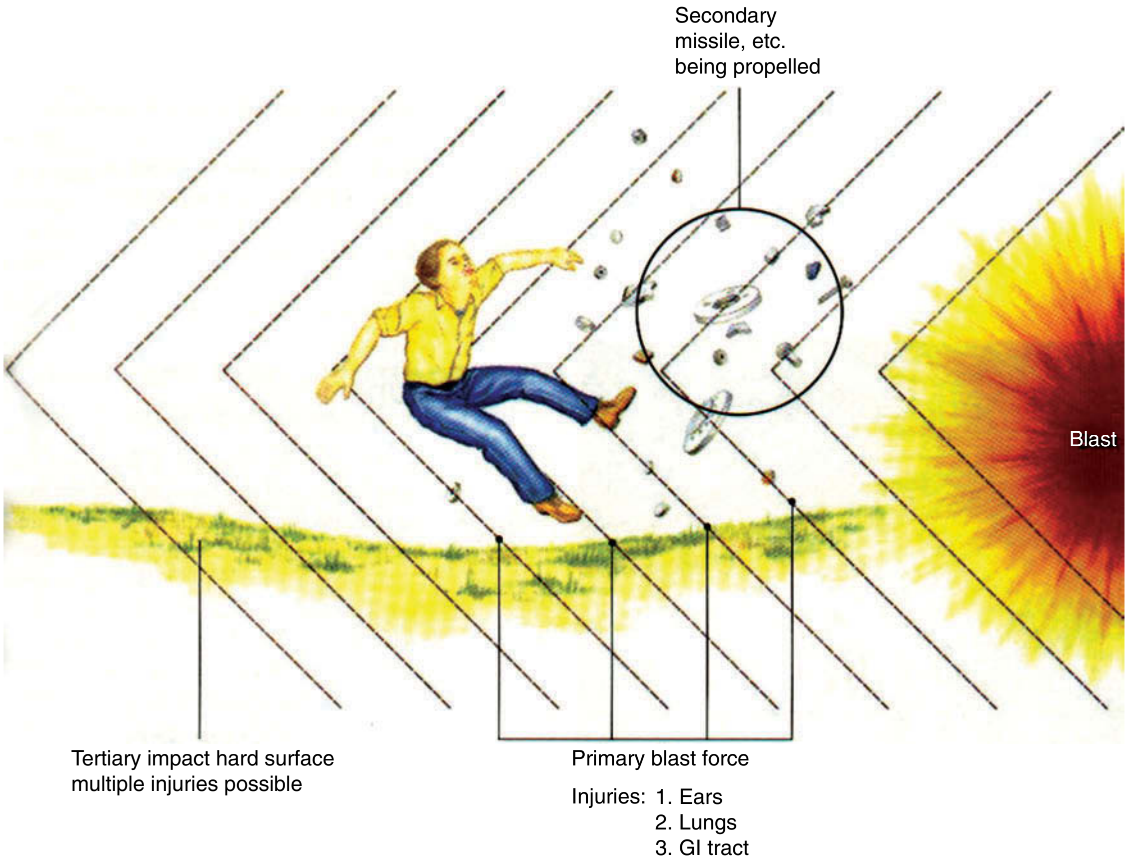

Figure 7.17
Blast injury. Explosions can cause injury with the initial blast, when the victim is struck by debris, or by the victim being thrown against the ground or other fixed objects by the blast. From Campbell, John E., *Basic Trauma Life Support for Advanced Providers*, 5th ed., Copyright 2004. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

Were there any drugs (including alcohol) at the scene?

Ask this question of emergency medical system (EMS) providers in addition to querying the patient about his or her use of drugs. This is important for the assessment of mental status and establishing the reliability of the patient's history and examination.

Associated symptoms

Did you experience any symptoms (chest pain, seizure, abdominal pain, headache, etc.) before the collision?

It is possible that the patient may have had a collision resulting from a medical problem. Always give consideration to these conditions as a possible cause of the incident. Furthermore, these conditions may be exacerbated by the stress of the incident.

Past medical

Can you tell me your AMPLE history?

Always take an "AMPLE" history from the patient or EMS providers. This information will help clinicians if the patient subsequently becomes noncommunicative:

A	Allergies
M	Medications
P	Past surgical and medical history
L	Last meal
E	Events surrounding trauma/environment

When was your last tetanus shot?

Many trauma patients have some degree of skin injury coupled with contamination (tetanus-prone wound); therefore, inquire about the patient's immunization status. Tetanus immunization should be updated according to accepted guidelines.

Do you have a bleeding disorder or are you taking anticoagulant medication?

Patients with bleeding disorders or taking anticoagulant medication (Coumadin) may bleed significantly following minor trauma; the threshold to search for occult bleeding is lower in these patients. It is important to get this information early on to allow timely reversal of their coagulopathy.

Are you taking any medication that would limit your cardiovascular response?

Patients taking certain medications (i.e., beta-blockers or calcium channel blockers) and patients with pacemakers may present with a "relative bradycardia" (a normal heart rate despite significant blood loss).

Differential diagnosis

Although not an exhaustive list, injuries that can be elusive or determined by physical examination have been included (Table 7.4).

Diagnostic testing

Laboratory studies

Type and crossmatch

A type and crossmatch should be obtained on all trauma patients at risk for significant hemorrhage. This is crucial for obtaining type-specific blood during the initial resuscitation.

Complete blood count

A complete blood count (CBC) may be misleading in trauma patients. White blood cell (WBC) counts are often elevated due to demargination of WBCs during the stress response, not infection. Although a hemoglobin concentration of less than 10 g/dL in a trauma patient indicates clinically significant anemia, a normal initial hemoglobin level does not exclude significant hemorrhage. It takes many minutes to hours before the hemoglobin value accurately reflects the degree of blood loss in trauma patients. Following the trend of serial hemoglobin measurements (every 1–2 hours) can provide useful information regarding ongoing blood loss.

Coagulation studies

Early identification and aggressive treatment of coagulopathy is important, especially in patients taking oral anticoagulants.

Electrolytes and renal function

Routine assessment of electrolyte status and kidney function may identify baseline renal impairment before patients receive IV contrast for imaging studies. Serious electrolyte imbalances should be treated depending on their role and risk to the patient.

Arterial blood gas

An arterial blood gas assesses both oxygenation (PaO_2 – partial pressure of oxygen in arterial blood) and ventilation ($PaCO_2$ – partial pressure of carbon dioxide in arterial blood). Many physicians utilize the *base deficit* to assess the patient's response to resuscitation efforts. The presence of an increased base deficit (≥6) or decreased serum bicarbonate may signify a metabolic acidosis resulting from acute blood loss and under-resuscitation.

Table 7.4 Traumatic injuries

Diagnosis	Symptoms	Signs	Work-up	Treatment
Airway obstruction/ esophageal intubation	Altered mental status, combativeness	Hypoxia, gastric breath sounds, abdominal distention, inability to BVM ventilate	Check ETT placement and oxygen supply.	Jaw thrust, chin lift, OPA, NPA; appropriately placed ETT
Cardiac tamponade	Shortness of breath, shock	Beck's triad: hypotension, muffled heart tones, JVD	Ultrasound (FAST)	Pericardiocentesis, pericardial window, thoracotomy
Flail chest	Shortness of breath, chest pain	Rib fractures, paradoxical movement of ribs, hypoxia	CXR may show pulmonary contusion as well as rib fractures.	Pain control, positive pressure ventilation, fluid restriction
Head injury (subdural hematoma, epidural hematoma, impending herniation)	Altered mental status, headache, combativeness	Focal neurologic examination, asymmetric pupils, Cushing's triad (HTN, bradycardia, irregular respirations)	Brain CT scan will define emergent intracranial injuries.	OR or ICU management, intracranial pressure monitor
Hemothorax	Shortness of breath, chest pain	Decreased breath sounds, percussion dullness	CXR may reveal opacification of the affected side due to supine position.	Tube thoracostomy, consider cell-saver device and auto-transfusion
Neurogenic shock	Paralysis, shock	Hypotension, relative bradycardia, paralysis, absence of sweating, wide pulse pressure	Clinical examination, CVP monitoring, exclude other causes.	Fluids, atropine, dopamine/norepinephrine, phenylephrine
Open Pneumothorax	Open defect in chest wall at least two-thirds the diameter of the trachea	"Sucking" chest wound	Detect on clinical examination, CXR.	Occlude wound on three sides to create one-way valve, tube thoracostomy
Pelvic fracture	Pelvic pain, suprapubic pain	Instability and/or pain with palpation or compression, ecchymosis, limited range of motion, cool or clammy skin, hypotension	Pelvic X-ray, CT scan, Hbg/Hct and crossmatch.	Pelvic binder, transfusion, interventional radiology for embolization, pelvic fixation (surgery)
Pneumothorax	Shortness of breath, chest pain	Decreased breath sounds, percussion tympany	CXR may demonstrate lung line; ultrasound.	Tube thoracostomy
Pulmonary contusion	Shortness of breath, chest pain	Decreased breath sounds	CXR, pulmonary infiltrates; ABG for A-a gradient, PaO_2.	Intubation if necessary and pain control
Solid organ injury	Abdominal and/or back pain, dizziness, confusion	Tenderness to palpation, ecchymosis, distention, cool or clammy skin, hypotension	FAST, CT abdomen, DPL/DPA, Hbg/Hct and crossmatch, chemistries.	Observation, transfusion, laparotomy, interventional radiology for embolization
Tension pneumothorax	Shortness of breath, shock	Hypotension, unilateral decreased breath sounds, tracheal deviation, JVD	Detect on clinical examination, not by CXR.	Needle decompression followed by tube thoracostomy
Traumatic aortic disruption	Chest pain radiating to back, between scapulae	Limited findings externally	CXR: widened mediastinum; CT angiography: periaortic hematoma, aortography.	Fluid and blood resuscitation, emergent operative repair

ABG: arterial blood gas; BVM: bag-valve mask; CT: computed tomography; CVP: central venous pressure; CXR: chest X-ray; DPL/DPA: diagnostic peritoneal lavage/aspiration; ETT: endotracheal tube; FAST: focused assessment with sonography for trauma; Hbg/Hct: hemoglobin/hematocrit; HTN: hypertension; ICU: intensive care unit; JVD: jugular venous distension; NPA: nasopharyngeal airway; OPA: oropharyngeal airway; OR: operating room.

Lactate

The body produces lactate during anaerobic glycolysis, which occurs during a shock state. A lactate may be followed to identify the adequacy of resuscitation.

Drug screen

Many institutions routinely obtain a urine drug screen on all trauma patients. This policy has limited utility, as most illicit drugs do not have specific antidotes (with the exception of opiates) and only require supportive care. Additionally, by the time the levels return, the condition as it relates to the traumatic injury should have already been identified and treated.

Pregnancy

The presence of a pregnancy less than 24 weeks gestational age does little to change the evaluation of a trauma patient. However, a positive pregnancy test may influence medication selection and non-emergent radiologic studies. Obvious or known pregnancy greater than 20–24 weeks should prompt inclusion of OB/GYN in the evaluation of the trauma patient and subsequent fetal monitoring.

Urinalysis

Gross hematuria or hypotension with microscopic hematuria requires an assessment of the renal system.

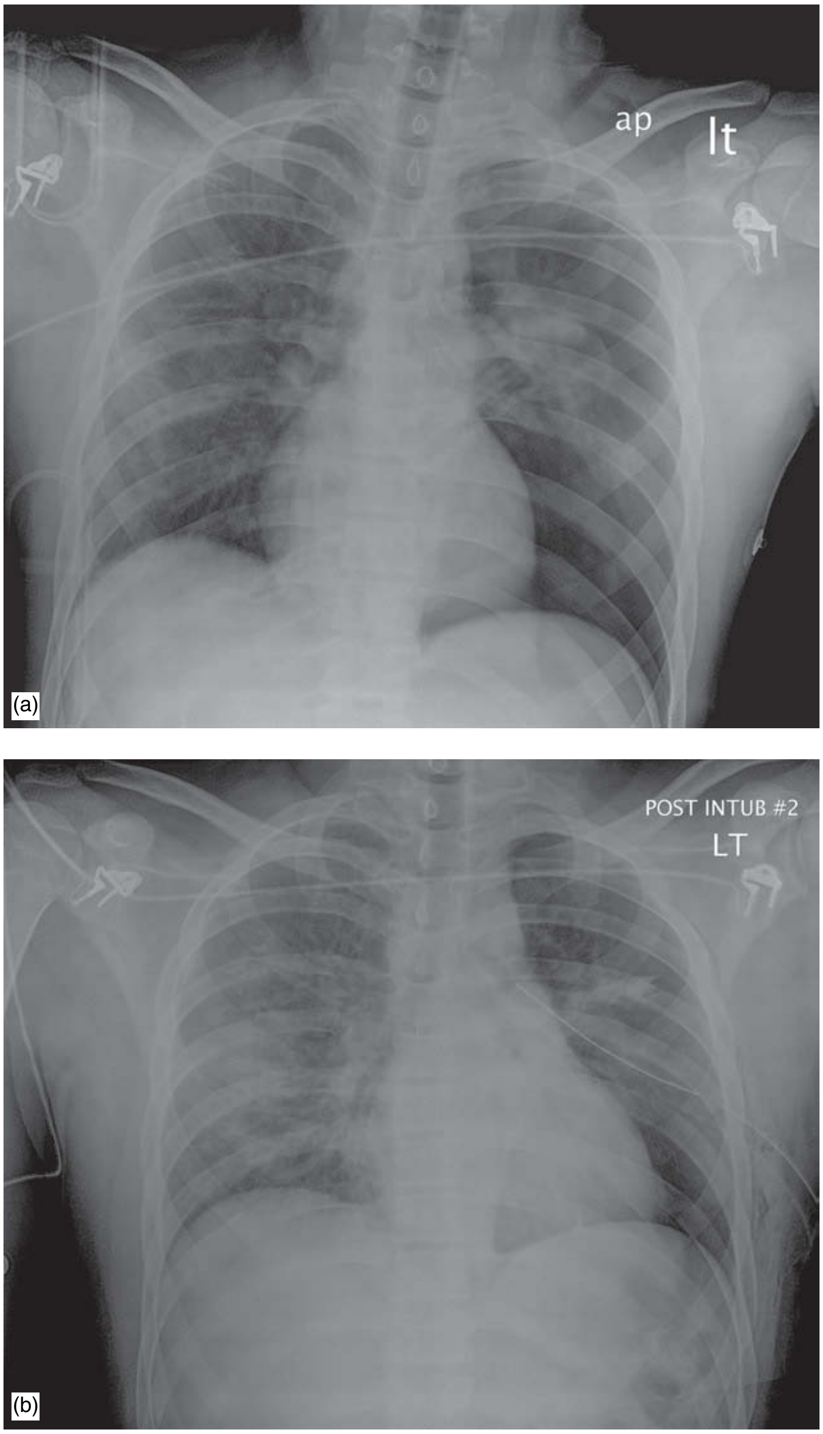

Figure 7.18
(a) Supine chest radiograph on a trauma board demonstrating a fracture of the left 7th rib posteriorly. A large left-sided pneumothorax is present, with deepening of the costophrenic sulcus, and partial collapse of the underlying lung. There are also fractures of the right posterior 5th, 6th, and 7th ribs, with no obvious pneumothorax on the right. (b) Anteroposterior (AP) chest radiograph following chest tube placement, with almost complete resolution of the pneumothorax and re-expansion of the left lung. Band atelectasis is present in the left mid-zone. Courtesy: S.V. Mahadevan, MD.

Electrocardiogram

The electrocardiogram (ECG) has limited utility in the trauma patient. An ECG should be obtained if a myocardial infarction is suspected or dysrhythmia is present, or as an aid to identifying the cause of trauma. An ECG and cardiac monitoring are recommended in cases of suspected traumatic cardiac injury, although the evaluation of this diagnostic entity remains controversial.

Radiologic studies

Trauma radiographs should include an anteroposterior (AP) chest and AP pelvis. The cervical spine may be evaluated by plain films or CT.

Chest X-ray

A chest radiograph is useful to assess for pneumothorax, hemothorax, pulmonary contusion and rib fractures (Figures 7.18a and b). It also allows for the early nonspecific assessment of an aortic injury by demonstrating a widened mediastinum or blurring of aortic knob (Figure 7.19).

Pelvis X-ray

An AP plain radiograph of the pelvis will identify the majority of pelvic fractures. It allows for early identification of serious pelvic injuries that may be a source of blood loss, and may also detect proximal femur fractures and hip dislocations.

Cervical spine X-ray

A three-view plain film series of the cervical spine is required to assess for fracture, subluxation and dislocation. Many trauma centers now utilize CT for assessment of the cervical spine due to its superior sensitivity for detecting injury.

The NEXUS cervical spine criteria identify low-risk trauma patients who do not require cervical spine radiography. Patients who meet all of the following five clinical criteria are at extremely low risk for cervical spine injury:

1. Normal level of consciousness
2. No painful distracting injuries
3. No evidence of intoxication
4. No posterior midline cervical tenderness
5. No focal neurologic deficits

Ultrasound

Focused assessment with sonography for trauma (FAST) has the advantages of being quick and noninvasive, and may be performed concurrently with the trauma resuscitation. The FAST examination can rapidly identify free fluid (presumably blood) in the peritoneal or pericardial cavity. Intra-abdominal fluid (presumably hemoperitoneum) in an unstable trauma patient calls for an emergent exploratory laparotomy without further CT imaging. The FAST examination may be repeated depending on the patient's ongoing hemodynamic status. A detailed description of the FAST examination can be found in Appendix E.

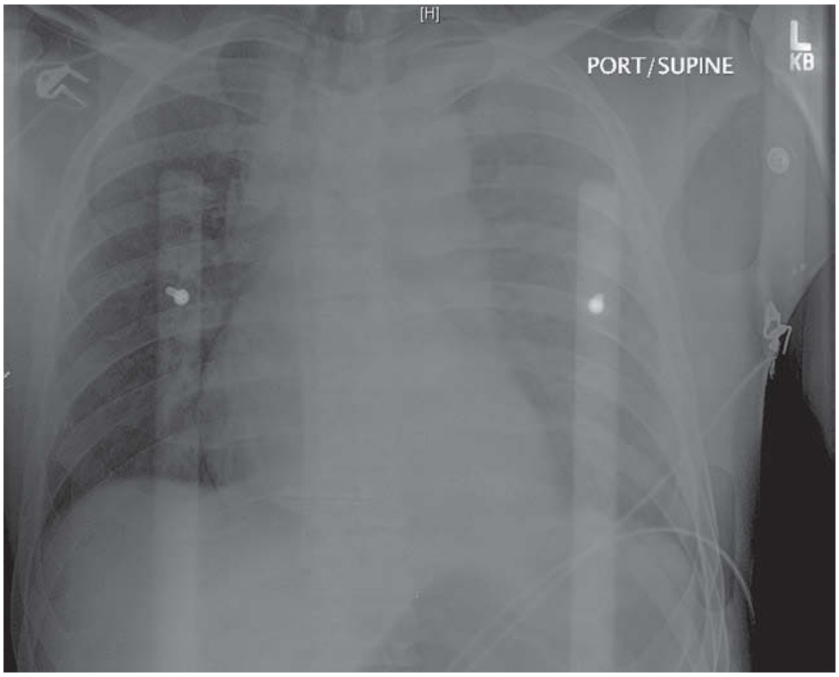

Figure 7.19
AP supine chest radiograph demonstrating widening of the superior mediastinum, with a poorly defined aortic contour and apical pleural capping suggestive of an underlying aortic injury. There is also a layering hemothorax on the left, with mediastinal shift to the right. Courtesy: S.V. Mahadevan, MD.

Computed tomography

Computed tomography (CT) is an essential diagnostic tool for the evaluation of hemodynamically stable trauma patients. CT scanning is commonly performed of the head, cervical spine, chest, abdomen and pelvis. The advent of helical (spiral) multi-detector CT scanners has improved the speed, accuracy and resolution of CT imaging. The use of reformatted CT images aids in the detection and characterization of subtle traumatic injuries. However, because most CT scanners are located outside of the ED, the decision to send a patient to the CT scanner should be made only after careful assessment of a patient's clinical condition (i.e., hemodynamic status) and the likelihood of hemodynamic decline.

Angiography

Many specialty centers have angiography suites designed for treatment of certain traumatic vascular injuries. In skilled hands, embolization of bleeding vessels can control or stop hemorrhage from pelvic fractures or other vascular sources with success rates approaching or surpassing those from surgery. It is important that the ED staff, trauma team and angiography staff work together regarding the management of these challenging patients.

Additional therapies

A nasogastric tube may decompress the stomach. However, nasogastric tube insertion is contraindicated in the presence of mid-face fractures. This may result in the inadvertent insertion of the nasogastric tube through a fractured cribriform plate into the cranial vault (Figure 7.20). An orogastric tube can still be carefully placed in this circumstance.

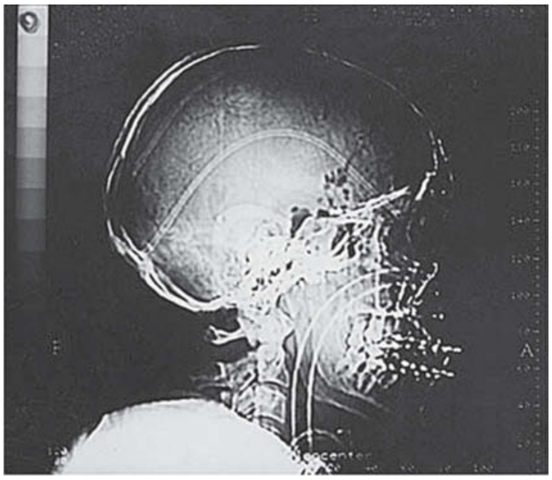

Figure 7.20
Computed tomography scout film reveals intracranial placement of a nasogastric tube in a patient with severe craniofacial trauma. Reprinted from Ferreras J, Junquera LM, Garcia-Consuegra L, Intracranial placement of a nasogastric tube after severe craniofacial trauma, in *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology*, Vol. 90, 564–566, © 2000 Mosby, with permission from Elsevier. Image courtesy of L.M. Junquera, Universidad de Oviedo, Spain.

A transurethral bladder (Foley) catheter monitors urine output, a sensitive indicator of renal perfusion and volume status. Special catheters have temperature probes. The Foley catheter is contraindicated if a urethral injury is suspected, as suggested by the following clinical findings:

1. Perineal ecchymoses
2. Blood at the urethral meatus
3. High-riding or non-palpable prostate
4. Scrotal hematoma

With any of these findings, a retrograde urethrogram may be necessary to exclude urethral injury prior to Foley catheter insertion.

Consider IV antibiotics and tetanus administration for traumatic wounds. In patients with open fractures, empiric prophylactic antibiotics should be administered as soon as possible after the injury.

Remember to treat pain early and adequately. It is important to frequently reassess the patient's need for additional pain medication.

Special patients

Pediatric

Although falls account for most pediatric injuries, motor vehicle–related injuries are the most common cause of death in all children. The most serious pediatric injury is blunt head injury. Accordingly, children are much more likely to suffer from apnea, hypoventilation and hypoxia than from hypovolemia and hypotension. Pediatric trauma patients require specialized management with respect to almost all portions of care. The use of a length-based resuscitation tape (e.g., Broselow tape) can assist with rapid determination of fluid volumes, drug doses and equipment size.

Airway management of children may be challenging due to anatomic differences from adults (e.g., large occiput in young children, sizable tongue, floppy epiglottis and anterior larynx). Because of the shorter trachea in young children, any movement of the head can lead to displacement of the ETT, including extubation or right mainstem intubation. When an intubated child deteriorates, consider the *DOPE* mnemonic (D for dislodgment, O for obstruction, P for pneumothorax, and E for equipment failure).

Children may maintain a normal sytolic blood pressure, even in the presence of shock and impending hemodynamic collapse. Hypotension in a child is a dangerous finding and represents decompensated shock. It is vital to screen for shock using other parameters, such as tachycardia and poor skin perfusion, and to initiate crystalloid fluid resuscitation early on. For children who fail to respond to three 20 mL/kg crystalloid boluses, strong consideration should be given to transfusing 10 mL/kg of packed red blood cells.

Peripheral venous access may be in difficult in children. An intraosseous infusion should be considered for children with circulatory collapse and impossible venous access, and after two failed attempts at peripheral venous cannulation.

Due to the increased body surface area-to-volume ratio, children lose heat and develop hypothermia more quickly than adults. Because children have a smaller body mass, blunt trauma delivers more force per unit of body area and results in multiple injuries.

As the ribs are more pliable in children, underlying organs may suffer significant damage without evidence of external injury. Identification of rib fractures in young children suggests a massive energy transfer and the possibility of severe underlying organ injury (e.g., pulmonary contusion). Certain abdominal visceral injuries (i.e., duodenal hematoma, blunt pancreatic injury, bladder rupture, small bowel perforation) are more common in children than adults.

The head is proportionately larger in young kids, leading to an increased incidence of blunt brain injuries. Head injuries in children are often devastating, as little room exists for brain swelling. Damage to the spinal cord and resulting neurologic injury may occur without evidence of spinal column fracture, a condition known as SCIWORA (Spinal Cord Injury Without Radiographic Abnormality). The GCS is modified for pediatrics by altering the pediatric verbal score for children younger than 4 years of age (Table 7.5).

Table 7.5 Modified Glasgow Coma Scale for preverbal children

Eye opening	
Spontaneous	4
To verbal command	3
To pain	2
None	1
Verbal response	
Coos, babbles	5
Cries, consolable	4
Persistently irritable (cries)	3
Restless, agitated (moans)	2
No response	1
Motor response	
Spontaneous	6
Withdraws to voice	5
Withdraws to pain	4
Abnormal flexion	3
Abnormal extension	2
No response	1

Any child who presents with a traumatic injury should be screened for physical abuse or nonaccidental trauma (Chapter 46). Child abuse accounts for the vast majority of homicides in infants.

Elderly

Elderly trauma patients most commonly die from falls, motor vehicle collisions and burns. Seemingly minor mechanisms of injury can result in serious injury or death in elderly patients. Older patients are more likely to die from their injuries due to limited physiologic reserves, comorbid conditions and medication complications.

The evaluation of elderly patients may be more challenging due to age-related changes to their anatomy and physiology. Early airway management should be considered for elderly patients presenting with shock, altered mental status, or chest wall injuries. Rib fractures and pulmonary contusions are poorly tolerated, and increased work of breathing and decreased pulmonary reserve may lead to respiratory failure. Degenerative changes in the cervical spine make endotracheal intubation more difficult and increase the risk of spinal cord injury from unintended spinal manipulation.

Elderly patients often lack the cardiovascular reserve necessary to respond to hypovolemia. They may not develop tachycardia due to age, medications, or heart block. A "normal" blood pressure and heart rate may not reflect euvolemia. Decreased sensitivity of abdominal exam due to aging and medical conditions (e.g., diabetes) may lead to missing catastrophic injuries. Close hemodynamic monitoring is essential to detect early signs of intravascular volume loss and shock.

As the elderly brain atrophies, stretching of parasagittal bridging veins makes them more prone to injury following impact. The additional intracranial space created by the loss of brain mass may mask clinical signs of intracranial bleeding despite significant hemorrhage. Head-injured patients taking anticoagulants have a much higher mortality rate. Elderly patients are susceptible to cervical spine and long bone fractures as a result of osteoporosis and osteoarthritis. The most common fracture locations are the proximal femur, hip, humerus and wrist.

All geriatric trauma patients should be screened for elder abuse, which is underrecognized and underreported (see Chapter 46).

Pregnant

Advanced Trauma Life Support (ATLS) suggests that a qualified trauma surgeon and obstetrician should be consulted early in the evaluation of the pregnant trauma patient. However, the initial evaluation and management priorities of a pregnant woman remain unchanged. The physician must keep in mind that there are two patients: mother and fetus. The best care for the fetus is optimal resuscitation of the mother and early assessment of the fetus. This includes adequate fluid resuscitation and prevention of maternal hypoxia or hypercarbia.

The primary cause of fetal demise is maternal shock and maternal death. Due to a rise in cardiac output and increased plasma volume, a pregnant trauma patient may lose a significant amount of blood (1,200–1,500 mL) before manifesting clinical signs of hemorrhage. Accordingly, the fetus may be in shock even though the mother appears stable.

Some pregnant women develop hypotension when placed in the supine position, as uterine compression of the inferior vena cava (IVC) impedes venous return to the heart, thereby reducing cardiac output. This *supine hypotension syndrome* may occur in a pregnant patient immobilized to a backboard. Elevating the right side of the spinal board or manually displacing the gravid uterus to the left may relieve pressure on the IVC.

As the uterus enlarges into the abdomen, the uterus and its contents become more vulnerable. The absence of a maternal abdominal injury does not exclude significant injury to the fetus, uterus, or placenta. Placental abruption is the second most common cause of fetal death. All pregnant patients should be assessed for vaginal bleeding, uterine irritability, abdominal tenderness, leakage of amniotic fluid, and changes or absence of fetal heart tones.

The fetus may be at risk, even with minor maternal injury. Therefore, cardiac tocodynamometry (to assess for uterine contractions and fetal cardiac activity) for a minimum of 4–6 hours is recommended for all pregnant trauma patients with an estimated gestational age greater than 20–24 weeks.

Almost all Rh-negative pregnant trauma patients should receive Rh immunoglobulin (RhIG) therapy. RhIG can effectively prevent Rh isoimmunization of an Rh-negative mother if administered within 72 hours of fetomaternal hemorrhage. A Kleihauer–Betke (KB) acid elution test can screen for large fetomaternal hemorrhage that exceeds the efficacy of the 300 mcg dose of RhIG. Remember to administer O negative blood if a type and crossmatch is not feasible.

All pregnant trauma victims should be screened for intimate partner violence regardless of ethnicity or socioeconomic status (see Chapter 46). Pregnant patients are commonly punched, kicked, or pushed during arguments, and yet still may not consider themselves victims of violence. These patients are often afraid to describe the details of their injury or their injuries are inconsistent with the stated history. Fears of being alone, intimidation, subsequent acts of violence, or losing financial support are only a few reasons pregnant women fail to report abuse.

Disposition

Although most EMS providers have established guidelines for transporting trauma patients to specific trauma centers, every ED should be prepared to handle patients who sustain traumatic injuries. Patients who require subspecialty care are typically transferred to trauma centers. The transfer process should be started as soon as a need for transfer is identified. Most trauma centers prefer to receive the patient earlier in the course of care (i.e., following initial stabilization). However, all life-threatening injuries should be addressed prior to transfer.

Be familiar with institution-specific criteria for notification of the trauma or surgical service. Most of the time, a trauma surgeon will be involved in the initial evaluation of the trauma patient, and will assist with admission and disposition decisions after the initial resuscitation. If a trauma surgeon is not present from the start, the EM physician should perform the necessary stabilization and treatment, including performance of life-saving procedures. While awaiting the arrival of the surgeon, emergency physicians have primary responsibility for trauma patients while they remain in the ED.

Disposition options for each trauma patient include discharge to home; admission to an observation unit, ward or intensive care unit (ICU); transfer to the OR or angiography suite; or transfer to another facility. Admission decisions should not be delayed until completion of an exhaustive evaluation. Rather, disposition options should be considered early and repeatedly throughout the evaluation.

Pearls, pitfalls and myths

- Always start with the ABCs. Attention should not be drawn to grotesque injuries, which may result in missing life-threatening ones. If the patient starts to deteriorate, return to the ABCs.
- Be suspicious of specific injuries based on the mechanism of injury. Maintain a high level of suspicion for injuries even if the patient looks well initially.
- The examination of the trauma patient should be quick, thorough and systematic. Managing the resuscitation of the trauma patient in the first (golden) hour often determines the patient's outcome. Being idle or not attending to detail can prove devastating for the patient.
- Use of the FAST exam has decreased the time to OR for unstable patients with hemoperitoneum.
- Keep all trauma patients warm. Exposure and IV fluids can cause hypothermia, which may lead to coagulopathy and worse outcomes.

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8 Emergency medical services

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History of emergency medical services

Historical accounts of emergency medical care being delivered by non-physicians outside of traditional health care settings date back well over a thousand years. Such accounts often reveal evidence of remarkable and inspiring advances. However, despite a long history of out-of-hospital medical care being delivered in civilian and battlefield situations, "modern" emergency medical services (EMS) has only truly emerged within the recent half-century.

Accidental Death and Disability: The Neglected Disease of Modern Society, released by the National Academy of Sciences' National Research Council in 1966, detailed America's serious deficiencies in out-of-hospital trauma management. Widely regarded as a sentinel call for improving out-of-hospital emergency medical care for all causes of illness, this "White Paper" stimulated state and federal lawmakers to enact needed standards for training, equipment and oversight in EMS systems. Essential development of EMS systems throughout the United States occurred through funding authorized in the Federal EMS Systems Act of 1973. As much as we wish to think that all landmark improvements in EMS resulted from rigorous scientific research and advances in care developed by emergency clinicians, the development of EMS systems has been significantly influenced by popular culture and politics.

Many of today's experienced emergency medical technicians (EMTs) trace their initial interest to a television show running from 1972–1977 titled "Emergency!" Millions of Americans gathered in the comfort of their homes and accompanied firefighter–paramedics Johnny Gage and Roy DeSoto as they saved lives, often in daring situations. In addition to a cadre of future emergency professionals, local politicians and influential citizens also watched the show every Saturday night, becoming energized to advocate at subsequent City Council meetings that similar lifesaving forces should be established in their hometowns. While relatively few Americans could expect to receive paramedic help within 10 minutes of placing a call to summon emergency medical assistance in the early 1970s, within a decade following the premiere of "Emergency!," such services were available to a majority of Americans.

Although "Emergency!" highlighted the development of EMS systems in Southern California, several prominent EMS systems were simultaneously evolving, led by visionary physician directors in Seattle, New York City, Columbus, and Miami. These early EMS systems produced evidence demonstrating that improved outcomes were possible when medical care previously reserved for hospital-based nurses and doctors was placed in the trained hands of EMTs and paramedics.

In the past decade, EMS has moved beyond defining its care boundaries with care "hand-me-downs" from the emergency department (ED). In fact, EMS since the year 2000 has served notice to its more established disciplines within the houses of medicine and public safety that advances in the emergency medicine standards of care can also come *from* EMS research. Two examples include: (1) use of noninvasive positive pressure ventilation (NIPPV) for respiratory distress stemming from congestive heart failure and/or chronic obstructive pulmonary disease; and (2) use of continuous waveform capnography for airway placement confirmation, ventilation status assessment, and active ventilation status management.

Unquestionably, continuing advances in research, technology and education will empower EMS and administrative leaders to sustain claims of positively impacting a community's standards of health. Understanding the basic structure and capabilities of typical EMS systems is a critical component in the practice of effective emergency medicine.

Emergency medical services system design

Each municipality or government-established EMS service area may utilize one or more designs to deliver timely and effective medical care. Government-operated EMS organizations are typically organized within existing municipal departments (fire departments) or as "third service" public safety agencies (i.e., agencies distinct from the fire and police departments). A variant of the third service is the public utility model (PUM). PUMs utilize an administrative authority (much like a water "authority") to oversee the system that contracts with a labor organization for EMTs and paramedics.

Alternatively, EMS systems may utilize ambulance services administered by a health organization, such as a single hospital, a consortium of hospitals, or the local department of health. In addition, privately owned ambulance services responding to municipal emergency calls are still prevalent in many regions of the United States.

Regardless of organizational type, emergency response vehicles are staffed by either paid (sometimes referred to as "career") or volunteer professionals. In a "fixed location" deployment model, personnel may be based at ambulance or fire stations. System status management models utilize historical demand for service data to forecast optimal ambulance deployment locations at flexible "posts," and preassign ambulances near locations of predicted imminent emergency medical incidents. Depending on system demands and staffing realities, some small-volume systems may use personnel responding from their non-EMS workplace or home.

While factoring a multitude of geographical, population-based and financial constraints, each EMS system must ultimately choose a design that reliably and effectively provides needed clinical care. Regionalization of resources often proves helpful when promoting rapid response times with high standards of care across large land regions. As service areas grow, aeromedical EMS agencies can provide additional value in EMS system design. These agencies enable rapid access to specialized care for patients with particularly time-sensitive conditions, and augment prehospital and hospital care.

EMS personnel and qualifications

There are generally four broad categories of EMS personnel: emergency medical responders (EMRs, until recently referred to as first responders), emergency medical technicians (EMTs, until recently referred to as EMT-Basics), advanced EMTs, and paramedics (EMT-Ps).

EMRs are trained in initial scene and patient assessments and in basic first aid measures such as bandaging, splinting, hemorrhage control and cardiopulmonary resuscitation (CPR). A typical course to obtain EMR certification involves 48–60 classroom hours. Generally, EMRs are police officers, firefighters and trained volunteers who may be the first to arrive to render medical assistance. EMRs usually do not transport patients.

EMTs are trained to assess signs and symptoms of illness and injury; safely extricate, immobilize and transport patients; and administer several noninvasive therapies such as oxygen. Though not trained in cardiac rhythm interpretation, EMTs can defibrillate through the use of automatic external defibrillators (AEDs). Care by EMTs is termed basic life support (BLS). Most EMT courses consist of 150–190 hours of clinical instruction, including clinical rotations in EDs and on ambulances.

Advanced EMTs acquire additional training in airway management and intravenous (IV) access for fluid and limited medication administration. Care by Advanced EMTs is termed advanced life support (ALS). Advanced EMTs provide critical increases in EMS system capabilities in many rural areas that cannot afford or recruit full paramedic coverage. The advanced EMT curriculum typically involves an additional 150–250 hours of training, including clinical rotations in EDs, preoperative areas, and on ambulances.

Paramedics are trained in additional advanced airway management, including endotracheal intubation and cricothyrotomy. A significant portion of the paramedic curriculum focuses on cardiac rhythm interpretation, cardiovascular medication administration and electrical therapy, including manual defibrillation and external cardiac pacing. Paramedic ambulances are widely termed ALS units (although some regions refer to them as mobile intensive care units). Given the substance of paramedic education, the curriculum spans several months of dedicated study encompassing at least 1,000–1,300 hours, including significant clinical time in EDs, preoperative areas, surgical suites, intensive care units, obstetric suites, and on ambulances.

With increasing regionalization of specialized medical and surgical services, critical care transport (by air or ground) is increasingly recognized as a unique area of expertise. Many states now allow paramedics with specialized additional training and supervision to function in critical transport programs at a level beyond standard protocols. Additional personnel beyond EMT-Ps are also frequently used in air and ground-based critical care transport. Physicians are standard members of the air medical crew in most non-US settings, but in the United States, non-physicians staff over 90% of crews. One exception in the United States is where emergency medicine residencies provide physician coverage for EMS. Nurses staffing critical care transport units generally have experience in both emergency and critical care settings, with additional experience (pediatric or obstetric) depending on the characteristics of the program's patient population. Other crew members, such as neonatal nurse practitioners or balloon pump specialists, may be used depending on local preferences and specific patient needs. At this time, consistent national standards for critical care transport do not exist, but at least one non-governmental accreditation agency (the Commission on Accreditation of Medical Transport Systems) has recognized that the level of care, rather than the transport vehicle, should be a prime focus for the evaluation of critical care transport services.

EMS response

911 system

Approximately 96% of the US population currently has access to emergency care via the 911 telephone system. Enhanced 911 systems use computer databases to display the caller's address, activating the 911 system in the event the caller is unable to speak. Although many mobile phones cannot be precisely located, there are emerging technologies that may help dispatchers approximate the location of a cellular phone. Global positioning system (GPS) software is now available in mobile phones so that callers may be located if they are lost or unable to speak. Many EMS systems train their dispatchers to follow a careful script of questions when they receive a call for help in a medical emergency. The answers to these questions determine the priority of the call and allow for the nearest available ambulance to be dispatched to the patient with the highest priority complaint.

Arrival on scene

The first priority of rescuers in any emergency is to ensure scene safety. Rescuers should not become injured or victims themselves. Violent crimes often occur in scenes that remain unsafe after initial injury. Despite the usual temptation to aid a victim as soon as possible, rescuers must not enter a violent scene until the police have first secured it. Rescuers are also at risk at the scene of motor vehicle collisions. Rescuers must survey the scene for potential hazards such as passing traffic, hazardous materials, or electrical wires. In accidents involving hazardous materials, rescuers must position themselves at a safe distance uphill and upwind, and the materials should be identified and the scene assured to be safe before entering. Specialized teams may need to be activated before a patient is reached. In all cases, victims must be adequately decontaminated before arrival at the hospital to avoid further spread of toxins to other patients and health care providers. Other circumstances require mobilization of additional resources before a patient may be safely reached. These include water rescue, trench and confined space rescue, and high-angle (high-elevation) rescue. These situations may be beyond the training of local authorities, requiring additional personnel and resources summoned from larger community units in the state or surrounding area.

Extrication

Extrication is the technique of safely removing the patient from his or her environment to reach the transport vehicle. This may be especially difficult with tight spaces, obese patients, rough terrain and trauma. Extrication in trauma may involve displacing debris that entraps a patient. Because significant force using hydraulic or air pressure often must be employed to manipulate the debris, specially trained rescuers are critical to minimize the risk of further injury to the patient from the debris or from unnecessary movement. While certain therapies (e.g., oxygen administration, IV insertion and fluid administration, parenteral analgesia, needle decompression of the thorax, and occasionally definitive airway management) may be started before the patient is free of the entrapment, a lengthy extrication may delay treatment and subsequent transport to an ED, leading to worse outcomes.

Clinical capabilities of EMS

Airway management

There are multiple devices designed to assist in the prehospital management of a patient's airway and breathing. Rescuers at all levels are trained in the use of bag-valve-mask (BVM) devices, the nasopharyngeal airway (NPA) and the oropharyngeal airway (OPA). The NPA and OPA are used to maintain airway patency.

Rescuers not trained in endotracheal intubation (ETI) or unable to achieve tracheal intubation with direct laryngoscopy may use an alternative airway, such as the Combitube (Figure 8.1) for a patient with airway compromise. The Combitube is designed for blind insertion into the patient's airway. The laryngeal mask airway (LMA, Figure 8.2) was first introduced in the operative setting in the late 1980s as an alternative to ETI for select patients, but has increasingly gained favor as an alternative when ETI cannot be achieved. The device consists of an inflatable V-shaped diaphragm at the end of a large-bore tube that is placed blindly into the larynx. It is relatively easy to use and minimizes the risk of gastric insufflation during assisted ventilation. It does not, however, protect the trachea from aspiration of blood or vomitus.

ETI remains the gold standard for definitive airway protection, though this technique depends on operator skill and patient factors. Many factors commonly encountered in prehospital care can make oral ETI difficult or impossible: operator inexperience, inadequate patient sedation or relaxation, blood or vomitus in the airway, and anatomic variables such as an anterior larynx or expanding neck hematoma. Outside of

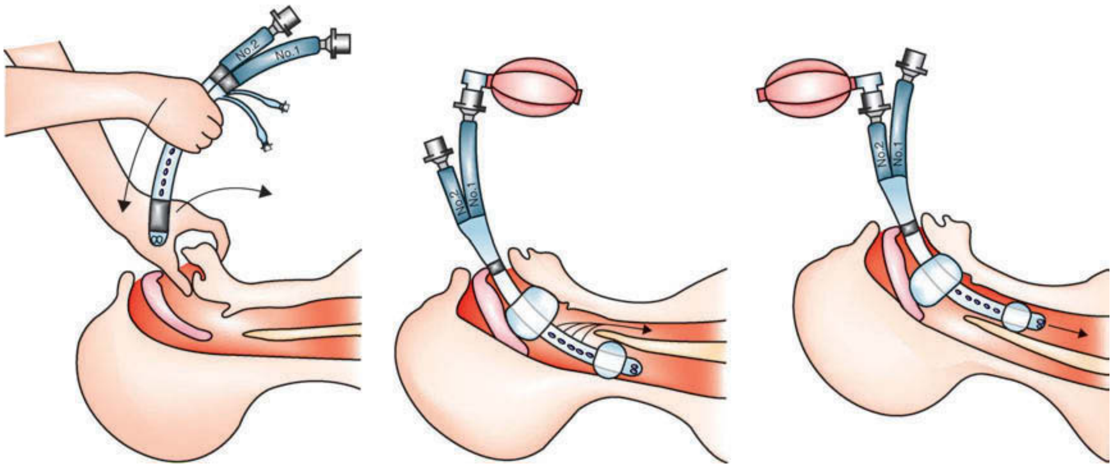

Figure 8.1.
The Combitube. Reproduced from D. Skinner et al, *Cambridge Textbook of Accident and Emergency Medicine*, Cambridge, Cambridge University Press, 1997.

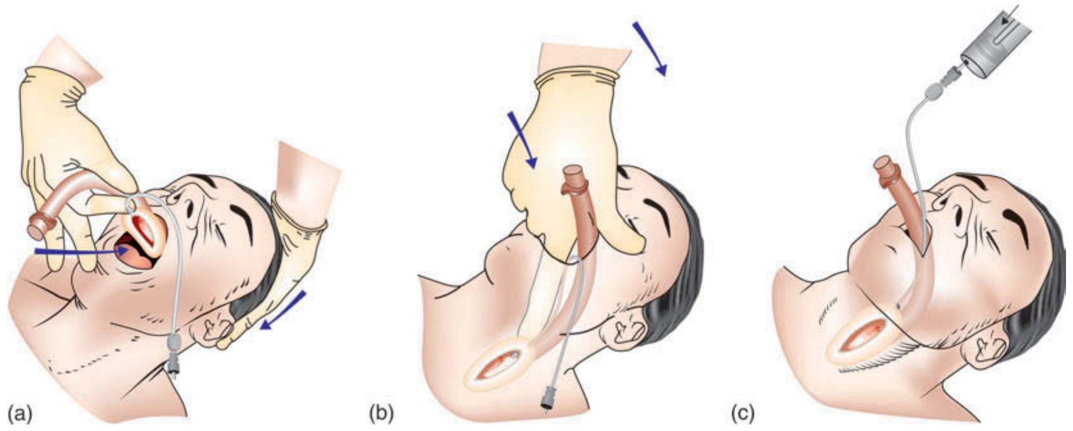

Figure 8.2
Laryngeal mask airway. (a) LMA placement into the pharynx. (b) LMA placement using the index finger as a guide. (c) LMA in place with cuff overlying larynx. Reprinted from J.R. Roberts and J.R. Hedges (eds), *Clinical Procedures in Emergency Medicine*, 4th ed., page 62, Copyright 2003, with permission from Elsevier.

investigational protocols, oral ETI is attempted in the prehospital setting only by rescuers with advanced EMT training or above.

Blind nasotracheal intubation (NTI) may be attempted for patients who still have spontaneous respiratory effort but need definitive airway control. This technique is of greatest use when orotracheal intubation is not possible or unlikely to succeed due to anatomic or traumatic reasons. The blind NTI technique, uncommon at receiving trauma centers, is employed more frequently in the prehospital setting when neuromuscular blockade is not available and jaw clenching prevents oral intubation. Blind NTI is contraindicated in patients with significant facial trauma.

Although neuromuscular blocking agents (paralytics) are an integral component of rapid sequence intubation (RSI) in the ED, historical issues raise concern about their use by prehospital personnel. Prehospital providers typically intubate infrequently and, if unable to intubate or ventilate a previously spontaneously breathing patient, have limited access to rescue techniques. Many helicopter transport services have reported high rates of successful intubations (>96%) using paralytics, but in general employ a very select and experienced group of practitioners. Some ground transport services have also reported high success rates of intubation (>94%) using paralytics, although this occurs most frequently in high-volume urban areas under close medical direction. At the current time, paralytics (and induction agents) to facilitate intubation should only be allowed in systems with highly trained and experienced providers operating under tight medical control, utilizing robust quality assurance and improvement programs; sufficient backup and rescue techniques must be available in the event of failed ETI.

Approximately 70% of US ground paramedics and all aeromedical paramedics are allowed to perform some form of emergency surgical airway if needed. Skills range from needle cricothyrotomy with jet ventilation, to the use of percutaneous kits employing the Seldinger technique (such as the Melker kit), to open cricothyrotomy. The need for cricothyrotomy in the field is fortunately infrequent. Surprisingly, given the lack of experience, success rates in the field are reported as high as 82–100%. All systems employing the use of paralytics must equip and train providers to perform a surgical airway in the event of failed intubation and ventilation.

Vascular access and fluid administration

Advanced EMTs and paramedics attempt IV access on all unstable or potentially unstable patients in the field. Many lifesaving medications are most effective, or only available, when administered IV. Though controversy exists in the resuscitation of trauma patients, crystalloid infusion remains the cornerstone of management of field hypotension. However, because cannulation attempts have been reported to add as much as 12 minutes to on-scene times, rescuers must not delay transport if adequate access can not been obtained. For most patients, the appropriate rule of thumb is two attempts per provider, ideally during transport of the patient. Intraosseous (IO) access, using the "non-collapsable vein" theory regarding the vascularity of bone marrow, has become common within paramedic EMS systems. In some systems, IO access is the primary vascular access route for patients in cardiopulmonary arrest. Regardless of device used (EZ-IO, Bone Injection Gun, or FAST), vascular access can typically be achieved within 30 seconds using the IO route. Typical sites of IO catheter placement include the proximal tibia, humeral head, and the sternum by default if using the FAST device.

In 1994, an important Houston study (Bickell, et al.) reported that aggressive prehospital fluid resuscitation of hypotensive victims of penetrating torso trauma did not improve survival and actually increased total blood loss compared with delayed resuscitation in the hospital. The results of this study and its applicability to other settings are still debated. However, prehospital fluid administration remains an area of ongoing research interest, and prehospital providers and physicians developing EMS protocols should be cognizant of over-resuscitation (with concomitant risk of increased hemorrhage) and under-resuscitation (with attendant risk of hypoperfusion).

Transport time and time to definitive control of suspected hemorrhage are important factors to consider when choosing whether to begin prehospital fluid resuscitation. Not all parameters will consistently be improved by fluid administration (e.g., altered mental status in a head-injured patient), and EMS providers must exercise judgment regarding the adequacy and appropriateness of their resuscitation.

Cardiac monitoring and defibrillation

Timely defibrillation is critically important for patients with ventricular fibrillation or non-perfusing ventricular tachycardia, since survival for these patients rapidly decreases with time. With the advent of AEDs, most EMRs and EMTs who arrive on scene before paramedics can defibrillate these patients. Rhythm interpretation and synchronized cardioversion of borderline perfusing rhythms are within the paramedic scope of practice. Paramedics are trained to perform and interpret 12-lead electrocardiograms (ECG). In many areas of the United States, paramedics directly activate the cardiac catheterization laboratory for patients exhibiting ST-segment elevation myocardial infarction (STEMI), saving valuable time and heart muscle from permanent damage. Several studies have demonstrated that well-trained paramedics have excellent accuracy for both rhythm recognition and detection of STEMI.

Medication administration

Many states allow EMTs to administer selected lifesaving medications such as oral glucose, auto-injected epinephrine, and nebulized albuterol. The bulk of medication administration, however, remains with paramedics. Paramedics are typically equipped with medications to treat pain, certain overdoses, hypoglycemia, bronchospasm, allergic reactions, hypotension and cardiac ischemia as specified in system-specific treatment protocols. Certain ALS systems may carry paralytic agents to facilitate intubation at the discretion of state and local medical directors. Field medication use, especially with controlled substances and with potentially proarrhythmic agents, must be tightly monitored and subject to regular quality assurance and improvement by the medical director.

Needle decompression

Most paramedics are permitted to perform chest decompression in a patient with suspected tension pneumothorax. Signs suggestive of tension pneumothorax include tachypnea, hypoxia, unilateral decreased or hyperresonant breath sounds, jugular venous distention, and deviation of the trachea away from the affected side. Needle decompression is indicated for a patient in severe distress with the above signs or in cardiac arrest following trauma.

Immobilization

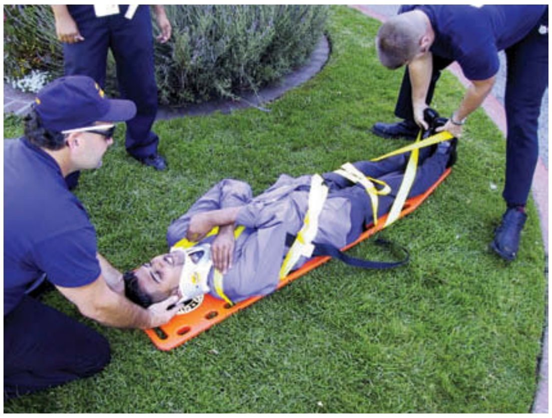

Figure 8.3
Spinal immobilization. Courtesy: S.V. Mahadevan, MD.

All EMS personnel are trained in the proper technique for spinal immobilization of patients (Figure 8.3). An appropriately sized rigid cervical collar should be placed on every victim of trauma with potential for spinal injury, including patients with pain, tenderness, or a suspicious mechanism of injury. However, because the cervical collar alone does not provide adequate immobilization for transport, patients should also be stabilized with a rigid backboard and some form of lateral stabilization (such as foam blocks) securing the head with straps or tape. Special steps, such as the use of a towel roll under the shoulders, may need to be taken to optimize head position (i.e., prevent flexion) in pediatric patients. Pregnant patients should have the right side of the backboard elevated 30° to keep the uterus off the inferior vena cava. This is done to avoid supine hypotension and fetal hypoperfusion. Placement of a patient on a backboard is not innocuous; studies have shown that pressure-mediated skin damage can begin after as little as 30 minutes on a backboard. Accordingly, most EMS systems now utilize selective spinal immobilization protocols.

The Kendrick extrication device (KED) is made up of a series of parallel splints longitudinally bound together in a vest-like device that provides assistance with spinal stabilization during the extrication of a trauma patient from an enclosed space, such as a motor vehicle. It does not provide full spinal immobilization, and therefore cannot be used in lieu of a backboard for adults. Due to its wrap-around nature, however, it may be useful for pediatric patients who cannot or will not lie still on a standard backboard.

Patients with unstable vital signs require immobilization of only those injured extremities that have the potential to cause further hemorrhage if moved (i.e., pelvis and long bones, especially suspected femur fractures). Angulated extremity fractures should be carefully evaluated for distal neurovascular status. Currently, most prehospital jurisdictions call for traction splinting of suspected femur fractures, but this is subject to debate. These devices require time for application and are of debatable benefit in the field, some with contraindications that may not be apparent. Any patient with an angulated fracture of any extremity resulting in absent distal pulses should have in-line traction applied in an attempt to regain pulses, followed by splinting. All other suspected fractures should be immobilized in the position of greatest comfort for transport.

Pneumatic anti-shock garment/military anti-shock trousers

Developed during the Vietnam War to treat soldiers exsanguinating in the field, the pneumatic anti-shock garment (PASG) was a mainstay of prehospital trauma care for nearly 20 years until its use was called into question by two outcome studies in the 1990s. Formerly known as the military anti-shock trousers (MAST), this device consists of a set of nylon pants with separately inflatable leg and abdominal sections that attach to a manual pump with a pressure gauge. Literature no longer supports the use of the PASG in penetrating or blunt trauma with hypotension. The PASG may be a useful immobilization device for pelvic fractures and/or femur fractures. PASG use is contraindicated in patients who are pregnant or who have pulmonary edema, evisceration of abdominal organs, cardiac tamponade, or cardiogenic shock.

Wound care

All EMS providers are trained to control external hemorrhage with direct pressure and elevation of the injury above the heart. Bandages that become soaked with blood should not be removed, but rather reinforced with further gauze. Tourniquets should be applied in cases of life-threatening limb hemorrhage. EMS providers should not remove tourniquets applied in the field for hemorrhage control.

Pediatrics

Although EMS personnel at all levels are trained to evaluate, treat and transport pediatric patients, many prehospital providers are uncomfortable when caring for acutely ill children. Such patients are relatively rare, and most cases evoke much greater stress for those involved. In general, the most significant differences between acutely ill adult and pediatric patients are as follows:

1. Vital sign abnormalities indicating significant injury or illness may be delayed compared with adult patients.
2. The age-specific nature of normal pediatric vital signs may lead practitioners to misinterpret absolute vital signs and potential for hemodynamic collapse.
3. Procedures, including IV access and intubation, are technically more challenging in children.
4. Children may be unable to give adequate histories or cooperate with procedures such as immobilization, and may require additional restraint for safe transport.

Recent data demonstrate that paramedics can deliver high-quality care to both adult and pediatric patients in nearly all arenas, but such care requires intensive education and regular skills review. One important exception to this rule is that pediatric patients should rarely be intubated in the field, even in cases of respiratory failure. Published data show that, in contrast with adults, morbidity and mortality are increased when prehospital care providers attempt to intubate apneic or hypoventilating pediatric patients. In general, prehospital pediatric intubation should only be attempted in EMS systems with particularly stringent intubation confirmation protocols to ensure that unrecognized esophageal intubations do not occur. For most EMS systems with short scene and transport times, current literature suggests the safest "advanced" airway management in pediatric patients is assisted bag-mask ventilation.

Mass casualty incidents/disaster

A mass casualty incident (MCI) is any event that produces multiple casualties (injuries or illness). A disaster is any event that overwhelms the capabilities of the local emergency response system and facilities. Although the two concepts are different, the principles of triage and care often overlap. Rescuers must be able to perform a brief (< 60 seconds) evaluation of each patient in an MCI, focusing on ventilation, perfusion and mental status, and triage each patient according to severity of injury. In large MCIs, a color-coded tag is attached to each victim to aid in efficient triage and transport. A sample medical emergency triage tag (METTAG) system is shown in Table 8.1 and Figure 8.4.

Incident command

Incident command is the system used for overall management of the disaster event. It is generally the responsibility of the ranking fire service officer on scene. EMS officials and occasionally an on-site physician experienced in disaster management are responsible for coordinating the medical activities and care with the incident commander.

Community-wide disaster systems

Planning and preparation prior to a disaster or MCI is critical for a successful response. Preparation should include plans for field response, hazardous materials, staging and transportation, documentation of available local hospital resources, communication plans and backup systems, documentation of victims and care provided, and debriefing and counseling after the events and recovery. Regular practice and drills are vital to train rescuers and test the system.

Medical direction

All care delivered by EMS personnel is provided under protocols and authority given to them by a physician medical director. The responsibility a physician assumes for the care delivered in an EMS system is called medical direction, medical control, or medical oversight. Most of the real-time medical care delivered by prehospital providers is done following prewritten standing orders ("off-line" medical control). This does not require direct

Table 8.1 Medical emergency triage tag system of field triage in a mass casualty incident

A suggested approach to treatment prioritization of victims is that found in the medical emergency triage tag system. The treatment priorities are defined as:

Zero priority (black):	Deceased or live patients with obvious fatal or non-resuscitatable injuries
First priority (red):	Severely injured patients requiring immediate care and transport (e.g., respiratory distress, thoracoabdominal injury, severe head or maxillofacial injuries, shock, severe bleeding, and severe burns)
Second priority (yellow):	Patients with injuries determined not to be immediately life-threatening (e.g., abdominal injury without shock, thoracic injury without respiratory compromise, major fractures without shock, head/cervical spine injury, and minor burns)
Third priority (green):	Patients with minor injuries that do not require immediate stabilization (e.g., soft tissue injuries, extremity fractures and dislocations, maxillofacial injuries without airway compromise, and psychological emergencies)

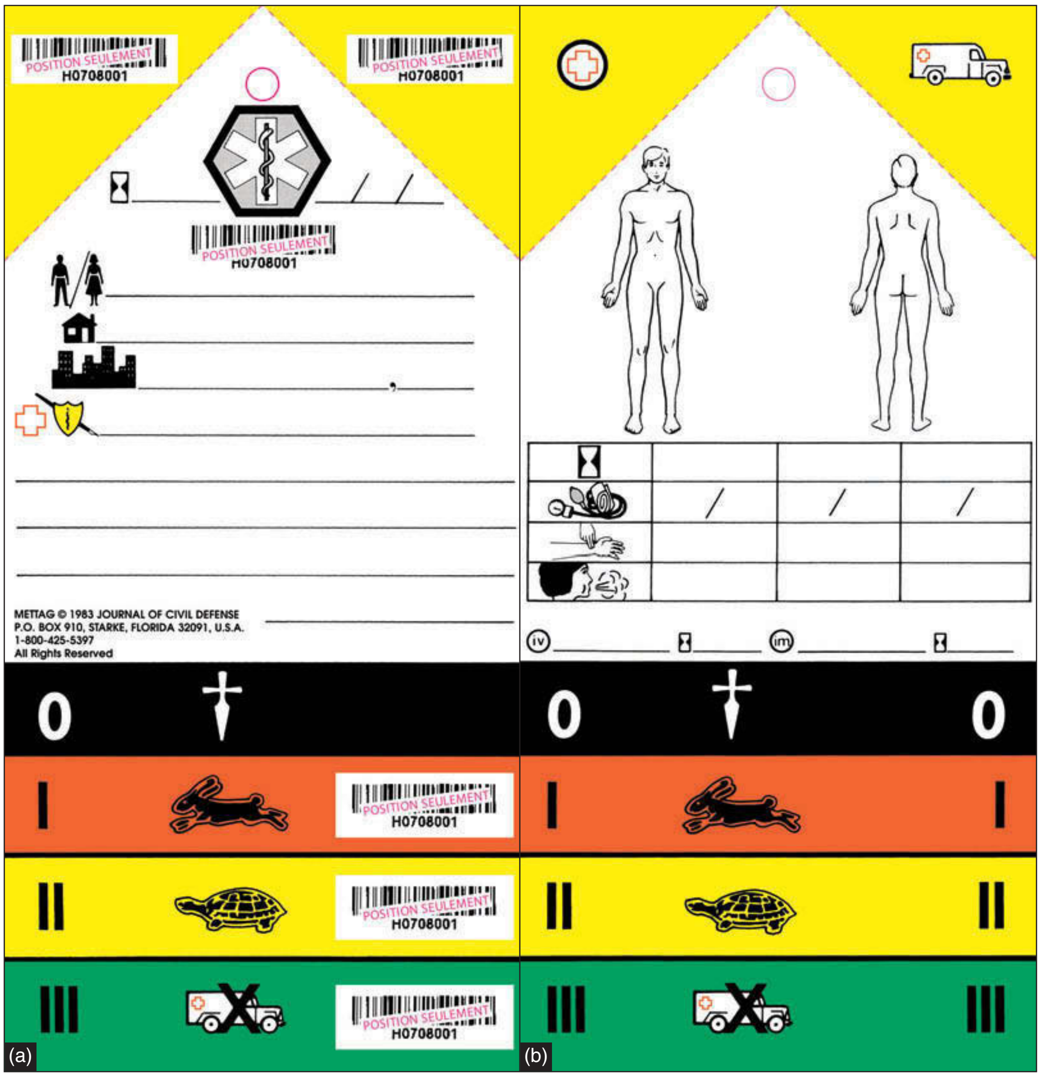
POSITION SEULEMENT
H0708001
METTAG © 1983 JOURNAL OF CIVIL DEFENSE
P.O. BOX 910, STARKE, FLORIDA 32091, U.S.A.
1-800-425-5397
All Rights Reserved
iv
im
0
I
II
III
(a)
(b)

Figure 8.4.
METTAG: Medical Emergency Triage Tag.

communication with a physician during the patient encounter. In these cases, patient care is reviewed retrospectively through standard processes, known as continuous quality improvement (CQI). This off-line component of education, training and continuing care review is the largest and the most important part of medical direction in EMS. In certain instances, however, such as the administration of IV opiates in some jurisdictions, paramedics must contact a physician directly by radio or phone for "on-line" medical control. In those cases, the orders given by the physician must still conform to state protocols and not exceed the paramedic's scope of practice. Rescuers may also use the on-line system to obtain a "field consultation" from a physician when necessary, as in cases of a patient's refusal of transport or other questions.

Patient transport

Vehicles

Standard ambulances come in various types, characterized by different vehicle designs. Type I ambulances are conventional box-type vehicles that lack a passageway between the driver and patient care compartments. Type II vehicles are van-type trucks. Type III vehicles are larger units with a forward cab and a walk-through passageway to the patient care area. Some units may require special equipment in order to provide electrical power to medical devices.

Many types of helicopters are used for patient transport. Depending on the resources and needs of a particular geographic area, helicopters of particular sizes, speeds, costs and physical characteristics may be selected. Most helicopters in use in the United States are twin-engine models, which have improved safety margins due to the redundancy afforded by the extra engine. Helicopter transports usually involve one patient only. For less acute patients, two-patient transports can be performed (if the helicopter allows). There is great variation between helicopter models with respect to size and speed; slower aircraft travel approximately 100–110 miles per hour, whereas other helicopters cruise nearly twice as fast.

Fixed-wing aircraft (airplanes) vary just as helicopters do, with a myriad of propeller- and jet-powered vehicles in use. In general, jet aircraft provide a smoother ride, faster speed, and are more likely to be able to pressurize to sea level, especially when flying at higher altitudes. Due to the relative isolation of patient care in a fixed-wing aircraft, patients should be reasonably stable before fixed-wing transport occurs.

Emergency warning devices

While the use of warning lights and siren (L&S) is standard among emergency vehicles, it is not without risk and controversy. Each year, rescuers, patients and bystanders are injured or killed in collisions during the use of L&S. In general, when operating with L&S, rescuers must exercise "due regard" for other vehicles; in all cases, the use of L&S must be based on standardized protocols that account for the severity of the complaint and the acuity of illness.

Patient transfer

It is not uncommon for IV catheters or endotracheal tubes (ETTs) to become dislodged during patient transport. Every possible precaution should be taken to secure medical access devices following their placement, and transfer patients deliberately. Optimally, one prehospital provider should have as his or her sole responsibility the assurance of maintaining ETT position during patient transfers. Additionally, re-confirmation of ETT position is warranted each time an intubated patient is moved from one surface to another. Utilization of continuous waveform capnography is an appropriate patient safety practice when intubated patients are in transit and any time movement can occur.

Communication

Communication between prehospital providers and hospital personnel most commonly occurs via simplex (one-way) radio systems using either ultra high frequency (UHF) or very high frequency (VHF). Advancing technology increasingly allows EMS providers to receive dispatch and scene information by computer and converse with dispatch or hospital personnel in a duplex (two-way) fashion, either with paired radio frequencies or cellular phones. Whenever possible, prehospital personnel should have backup systems to their primary means of communication.

Destination criteria

Severely injured victims of trauma should be transported directly to a designated Level I or II trauma center, bypassing smaller hospitals or non-trauma centers when transport times are not excessive. One study revealed that patients who must be transferred a second time from a local hospital to a trauma center had a 30% increased risk of mortality compared with those who were transported directly from the scene to the trauma center. Furthermore, for similarly injured patients, the risk of dying in a Level I trauma center was 54% lower than in Level II centers and 75% lower than in hospitals that were not trauma centers. EMS personnel should follow state protocols regarding indications for transport directly to a trauma center, but most protocols are similar to the American College of Surgeons' Field Triage Algorithm (Figure 8.5). The patient in cardiac arrest with continuing presence of shockable rhythms (e.g., ventricular fibrillation) should be transported to the nearest available ED, even in cases of trauma. Victims of trauma who arrest in the field have a dismal prognosis but may warrant the immediate application of hospital resources to treat potentially reversible causes of death.

As with victims of major trauma, significantly burned patients meeting appropriate triage criteria should be transported directly to a designated burn center when possible (Table 8.2).

Currently, nationally recognized point-of-entry (POE) criteria that allow EMS personnel to bypass a nearby hospital for one with specialty services farther away only exist for the transport of patients with severe trauma or burns. Expanded POE criteria are being instituted in several communities for selected disease processes, such as STEMI or acute stroke, and are becoming standard of care in many communities.

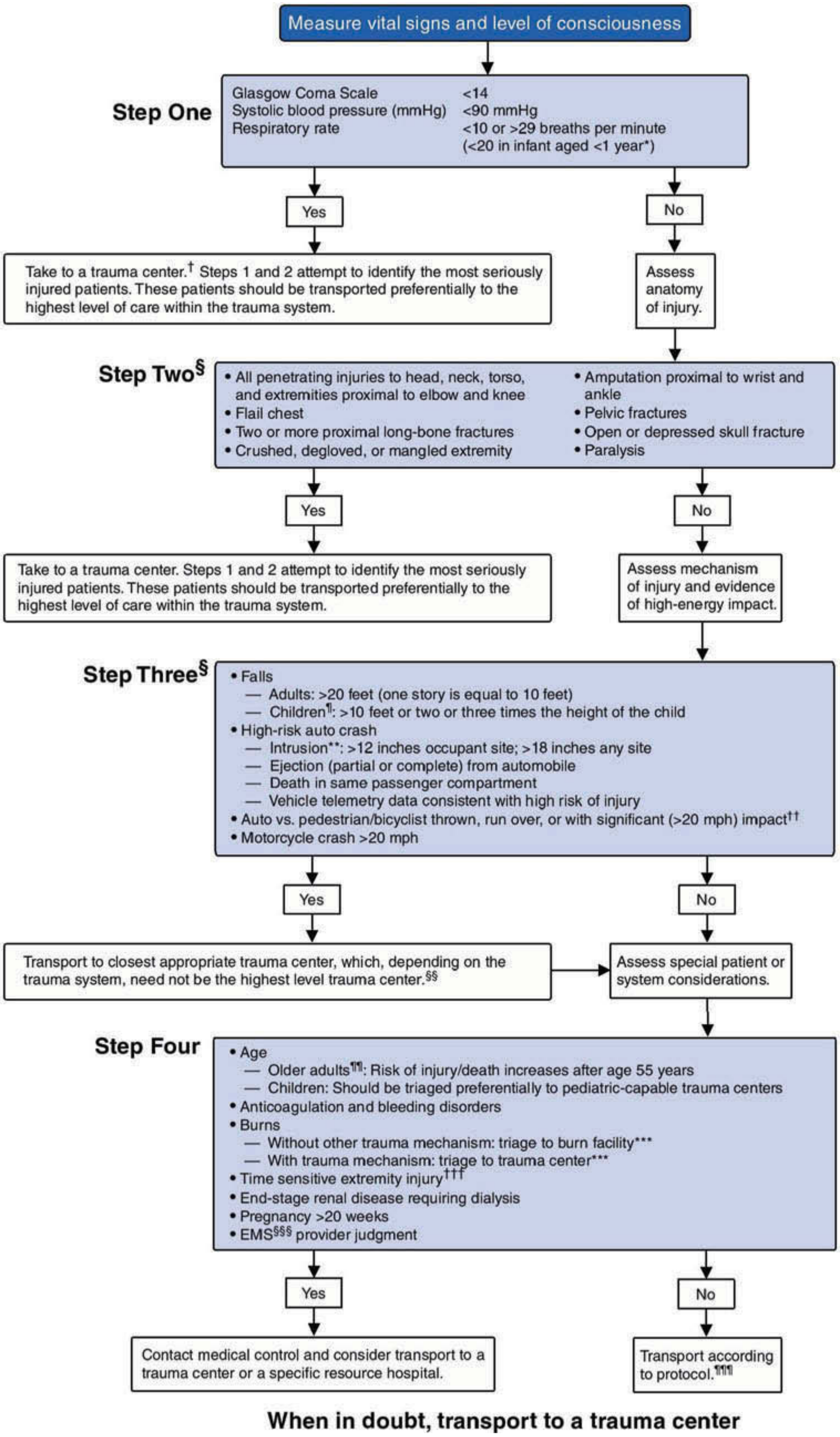
Measure vital signs and level of consciousness
Step One
Glasgow Coma Scale <14
Systolic blood pressure (mmHg) <90 mmHg
Respiratory rate <10 or >29 breaths per minute (<20 in infant aged <1 year*)
Yes
No
Take to a trauma center.† Steps 1 and 2 attempt to identify the most seriously injured patients. These patients should be transported preferentially to the highest level of care within the trauma system.
Assess anatomy of injury.
Step Two§
• All penetrating injuries to head, neck, torso, and extremities proximal to elbow and knee
• Flail chest
• Two or more proximal long-bone fractures
• Crushed, degloved, or mangled extremity
• Amputation proximal to wrist and ankle
• Pelvic fractures
• Open or depressed skull fracture
• Paralysis
Yes
No
Take to a trauma center. Steps 1 and 2 attempt to identify the most seriously injured patients. These patients should be transported preferentially to the highest level of care within the trauma system.
Assess mechanism of injury and evidence of high-energy impact.
Step Three§
• Falls
— Adults: >20 feet (one story is equal to 10 feet)
— Children¶: >10 feet or two or three times the height of the child
• High-risk auto crash
— Intrusion**: >12 inches occupant site; >18 inches any site
— Ejection (partial or complete) from automobile
— Death in same passenger compartment
— Vehicle telemetry data consistent with high risk of injury
• Auto vs. pedestrian/bicyclist thrown, run over, or with significant (>20 mph) impact††
• Motorcycle crash >20 mph
Yes
No
Transport to closest appropriate trauma center, which, depending on the trauma system, need not be the highest level trauma center.§§
Assess special patient or system considerations.
Step Four
• Age
— Older adults¶¶: Risk of injury/death increases after age 55 years
— Children: Should be triaged preferentially to pediatric-capable trauma centers
• Anticoagulation and bleeding disorders
• Burns
— Without other trauma mechanism: triage to burn facility***
— With trauma mechanism: triage to trauma center***
• Time sensitive extremity injury†††
• End-stage renal disease requiring dialysis
• Pregnancy >20 weeks
• EMS§§§ provider judgment
Yes
No
Contact medical control and consider transport to a trauma center or a specific resource hospital.
Transport according to protocol.¶¶¶
When in doubt, transport to a trauma center

Figure 8.5
American College of Surgeons' Field Triage Algorithm.

(*continued*)

Figure 8.5
American College of Surgeons' Field Triage Algorithm (*cont.*)

SOURCE: Adapted from American College of Surgeons. Resources for the optimal care of the injured patient. Chicago, IL: American College of Surgeons; 2006. Footnotes have been added to enhance understanding of field triage by persons outside the acute injury care field.

* The upper limit of respiratory rate in infants is >29 breaths per minute to maintain a higher level of overtriage for infants
† Trauma centers are designated Level I–IV, with Level I representing the highest level of trauma care available.
§ Any injury noted in Steps 2 and 3 triggers a "yes" response.
¶ Age <15 years.
** Intrusion refers to interior compartment intrusion, as opposed to deformation which refers to exterior damage.
†† Includes pedestrians or bicyclists thrown or run over by a motor vehicle or those with estimated impact >20 mph with a motor vehicle.
§§ Local or regional protocols should be used to determine the most appropriate level of trauma center; appropriate center need not be Level I.
¶¶ Age >55 years.
*** Patients with both burns and concomitant trauma for whom the burn injury poses the greatest risk for morbidity and mortality should be transferred to a burn center. If the nonburn trauma presents a greater immediate risk, the patient may be stabilized in a trauma center and then transferred to a burn center.
††† Injuries such as an open fracture or fracture with neurovascular compromise.
§§§ Emergency medical services.
¶¶¶ Patients who do not meet any of the triage criteria in Steps 1–4 should be transported to the most appropriate medical facility as outlined in local EMS protocols.

Table 8.2 Criteria for transport directly to a designated burn center

1. Partial thickness burns > 10% (total body surface area)
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints
3. Third degree burns in any age group
4. Electrical burns, including lightning injury
5. Chemical burns
6. Inhalation injury
7. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality. Burns in any patients with concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses a greater immediate risk than the burns, it may be necessary to stabilize the patient in a trauma center before being transferred to a burn unit. Physician judgment is necessary in such situations and should be in concert with the regional medical control plan and triage protocols
8. Burns in children being cared for in hospitals without qualified personnel or equipment for the care of children
9. Burn injury in patients who will require special social, emotional, or long-term rehabilitative intervention

Special considerations in air transport

The decision of when a helicopter should respond to the scene of injury or illness remains an inexact science. The best sources acknowledge that judgment of the prehospital personnel at the scene is of primary importance. The decision to use helicopter transport can be bolstered by criteria listed below and in Table 8.3:

1. Mechanism of injury
2. Physiologic variables
3. Anatomic variables
4. Time and logistics

Space constraints are the major issue in providing care in any aircraft. Both the actual space (cubic feet) and the arrangement of the space (cabin configuration) can have profound effects on the ability of the air medical crew to perform interventions. This translates into the need for the air medical crew to sometimes adjust the care provided accordingly. One example would be intubating a patient prior to flight if there is a significant chance of airway deterioration while en route. Crew members should be cross-trained to allow either crew member to provide indicated medical interventions during flight. Some interventions, such as chest compressions, are extremely difficult to provide effectively in an aeromedical setting.

Noise is of a sufficient degree to preclude reliable auscultation and monitoring of aural alarms (e.g., a ventilator). The flight crew must learn to use other means of patient assessment and equipment monitoring. Vibration is a theoretical problem for the patient, and high-frequency vibrations have been shown to induce fatigue in caregivers. In general, however, the ride in a helicopter or fixed-wing aircraft is often much smoother than a ride in a ground ambulance.

Lighting in an aircraft, and to a lesser extent in a ground ambulance, differs from that which is normally available in a hospital resuscitation area. Some helicopters, for instance, have patient care cabins that are contiguous with the pilot seat; in such situations, the medical crew must work in red, blue, or dimmed lighting at night.

Altitude issues relate to hypoxemia, pressure–volume changes, temperature and humidity. Altitude-related hypoxemia is not usually an issue because patients receive oxygen therapy and the altitude is usually not sufficiently high for the crew to require supplemental oxygen. Exceptions to this general rule occur, however, with both patients (e.g., premature neonates with narrow therapeutic windows for oxygen administration) and crew (e.g., crew in programs based at higher altitudes wear oxygen masks for prevention of hypoxemic symptoms). Boyle's law describes the inverse relationship between ambient pressure and gas volume. This is a factor with respect to both equipment (e.g., ventilator, intra-aortic balloon pump, Minnesota tubes for upper gastrointestinal hemorrhage tamponade) and patients (e.g., need for pre-flight placement of a gastric tube to prevent vomiting in unconscious patients). High altitude is associated with decreased ambient temperature. Especially in colder climates where a patient may be hypothermic before being loaded on the aircraft, and in aircraft with suboptimal heating systems, such as helicopters, guarding against

Table 8.3 National Association of Emergency Medical Service Physicians guidelines for dispatching a helicopter to an emergency scene

Clinical

1. General
    (a) Trauma victims need to be delivered as soon as possible to a regional trauma center
    (b) Stable patients who are accessible to ground vehicles probably are best transported by ground

2. Specific
    Patients with critical injuries resulting in unstable vital signs require the fastest and most direct route of transport to a regional trauma center in a vehicle staffed with a team capable of offering critical care en route. Often this is the case in the following situations:
    (a) Trauma score <12
    (b) Glasgow coma scale <10
    (c) Penetrating trauma to the abdomen, pelvis, chest, neck, or head
    (d) Spinal cord or spinal column injury, or any injury producing paralysis of any extremity if any lateralizing signs
    (e) Partial of total amputation of an extremity (excluding digits)
    (f) Two or more long bone fractures, or a major pelvic fracture
    (g) Crushing injuries to the abdomen, chest, or head
    (h) Major burns of the body surface area, or burns involving the face, hands, feet or perineum, or burns with significant respiratory involvement, or major electrical or chemical burns
    (i) Patients involved in a serious traumatic event who are <12 or >55 years of age
    (j) Patients with near drowning injuries, with or without existing hypothermia
    (k) Adult trauma patients with any of the following vital sign abnormalities:
        (i) systolic blood pressure <90 mmHg
        (ii) respiratory rate <10 or >35 breaths/min
        (iii) heart rate <60 or >120 beats/min
        (iv) unresponsive to verbal stimuli

Operational situations in which helicopter use should be considered:

1. Mechanism of injury:
    (a) Vehicle roll-over with unbelted passengers
    (b) Vehicle striking pedestrian at >10 miles per hour
    (c) Falls from >15 feet
    (d) Motorcycle victim ejected at >20 miles per hour
    (e) Multiple victims
2. Difficult access situations:
    (a) Wilderness rescue
    (b) Ambulance egress or access impeded at the scene by road conditions, weather, or traffic
3. Time/distance factors:
    (a) Transportation time to the trauma center >15 minutes by ground ambulance
    (b) Transport time to local hospital by ground greater than transport time to trauma center by helicopter
    (c) Patient extrication time >20 minutes
    (d) Utilization of local ground ambulance leaves local community without ground ambulance coverage

hypothermia is important. Higher altitude and lower temperature are associated with decreased humidity. This can result in hardening of secretions, which the air medical crew should monitor (and suction) as indicated. Helicopters generally transport patients at altitudes of 500–2,000 feet above ground level. Therefore, unless transports occur at geographic locations where ground level is significantly elevated, altitude issues are of relatively minor concern for the majority of helicopter transports. On the other hand, fixed-wing transports occur at much higher altitudes, which brings into play issues of cabin pressurization and risks of sudden decompression.

Safety is the paramount consideration for any air transport service. At any time, in any mission, the pilot or medical crew should be empowered to halt the transport if safety considerations become a concern. Direct comparison between air and ground vehicle safety is difficult, since crashes involving medical helicopters (or less commonly, fixed-wing aircraft) are more reliably tracked and more widely publicized than crashes of ground vehicles. Sometimes, considerable judgment must be exercised in determining whether to perform a critical procedure (e.g., intubation) before or after transport commences. Except in cases in which a fixed-wing aircraft is used solely because critical patients cannot be evacuated by air (e.g., fog precludes helicopter operations but a fixed-wing aircraft can safely operate in a remote area), patients transported by airplane typically have lesser acuity and greater stability than those transported by ground.

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9 Pain management

Eustacia (Jo) Su, MD

Scope of the problem

Acute pain is the most common complaint of patients presenting to the emergency department (ED), comprising 60% of presenting complaints in one study. In the United States, the National Hospital Ambulatory Medical Care Survey: 2006 Emergency Department Summary reported that patients' presenting level of pain was severe 20.4% and moderate 25% of the time. Recognition and acknowledgment of a patient's pain, assessment, adequate treatment, and timely reassessment are essential to acute pain management in the ED. Unfortunately, it has been demonstrated that many physicians fail to treat pain promptly or adequately in both inpatient and outpatient settings. Physicians are often reluctant to treat pain when they do not yet know the diagnosis, but pain is often worsened by the manipulations required for diagnosis (e.g., radiographs for long bone fractures). Adequate analgesia can improve results from diagnostic testing (e.g., better quality of the radiographs obtained if the patient is able to tolerate correct positioning).

Pain

Pain is defined by the patient: there are no objective signs that reliably help the physician determine whether a patient is in pain or how severe the patient's pain is at that moment. Some patients are extremely stoic, others very demonstrative. The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage," or described in terms of such damage as "always subjective" and "learned through experiences related to injury in early life." Pain can be classified as nociceptive or neuropathic. Nociceptive pain is a response to damaged tissue and may present as either somatic (localized and reproducible to palpation) or visceral pain. Neuropathic pain is generally associated with nerve injury. It is typically persistent, and described as burning or shock-like. Nociceptive pain usually responds better to opioids than neuropathic pain, which may require adjunctive, nonopioid therapies.

Pain includes behavioral and physical indicators in addition to self-report. Thus, preverbal, nonverbal, or cognitively impaired individuals who are experiencing pain can benefit from careful pain assessment; input from caregivers is essential. Fear and anxiety increase the perception of physical pain – the unfamiliar and frequently unfriendly ED environment does little to ameliorate a patient's pain.

Acute pain is a symptom of injury or illness that serves the biologic purpose of warning an individual of a problem and limiting activities that might exacerbate it. Acute pain is usually associated with identifiable pathology and causes anxiety. By convention, it is present for less than 6 months.

Chronic, malignant pain is associated with a terminal disease, such as cancer or acquired immune deficiency syndrome (AIDS). These patients are usually under the care of a multidisciplinary team that directs their analgesia regimen.

Chronic, nonmalignant pain is a complex problem, defined as pain being present for greater than 6 months. In general, it is not associated with a readily treatable, or sometimes even identifiable cause. It is generally associated with depression rather than anxiety. Patients may have a well-defined cause (e.g., tic douloureux) or no objectively confirmed cause (e.g., reflex sympathetic dystrophy). These patients frequently create animosity among ED staff because they can be demanding, often have personality disorders, and can be manipulative. The staff often senses that acute interventions will generally fail to help these patients.

There are patients who feign pain to acquire opioids, either for their own use or to sell. These individuals may be difficult to distinguish from the groups previously defined.

Analgesia

Analgesia is the "loss of sensitivity to pain." In the ED, this means the reduction of pain through therapy. The therapy is not solely pharmacologic in nature – psychologic and social support, as well as physical positioning for maximum comfort, help reduce perceived pain. These interventions reassure the patient that the provider is aware of his or her pain and is making attempts to relieve it. Child life therapists, when available, provide psychologic support to children as well as distraction from painful procedures, such as drawing blood or starting an intravenous (IV) line.

Oligoanalgesia

Acute pain that is inadequately or poorly treated may result in negative physiologic outcomes, may exacerbate the underlying pathophysiology of many illnesses and injuries, and may lead to the development of chronic pain.

The failure of physicians to treat pain has been documented in the ED as well as in the inpatient setting. Children receive fewer doses of analgesia, in general, and opiates, in particular, than adults with equivalent diagnoses or undergoing equally painful procedures.

In 1989, Wilson and Pendleton reported that 56% of patients presenting with painful conditions received no analgesics in one academic ED. Furthermore, only 14% received any analgesia within the first hour of their ED stay. In this study, meperidine was the medication most commonly administered. Findings included inadequate doses 55% of the time, and agents administered by intramuscular (IM) injection 60% of the time, despite the known disadvantages of this route. In this study, only 31% of patients with an acute myocardial infarction and persistent chest pain received IV opioids. Lewis and Sasater reported in 1994 that only 30% of patients with acute fractures in eight EDs received opioids while in the ED.

Assessment and measurement of pain

Goals and challenges

It is imperative for physicians to detect and measure pain rapidly so that prompt treatment can be instituted and its effect assessed. Even though a patient may not appear to be in pain, he or she may actually be in severe pain. Careful listening, observation and repeated solicitation may be necessary to fully elicit an admission of pain. Assessment must be both *qualitative* (is pain present?) and *quantitative* (how much does it hurt?). "Has the pain improved following treatment?" is an important reassessment question. Early reassessment must follow the initial treatment not only to ensure its adequacy, but also to provide repeat doses promptly in order to prevent pain recurrence.

There are no reliable objective or physiologic signs of pain. Normal vital signs may persist despite severe pain. Medication, a personal or cultural tendency to stoicism, or adaptive mechanisms such as joking may mask the presentation of pain. Language and cultural barriers also interfere with the patient's ability to communicate his or her pain to the physician and health care team. Preverbal children, especially toddlers, may only be able to express an "owie." Neonates and young infants cannot verbalize at all; interpreting their cries requires time, experience and motivation to recognize and treat their pain.

Treatment of pain should closely follow pain assessment. In one study, mandating a pain assessment in the electronic medical record did not result in more patients receiving analgesics or in more rapid delivery of analgesics.

Some EDs have implemented policies requiring triage personnel to notify physicians about patients who appear to be in pain, so that these patients might receive analgesic medications earlier in their ED course. These programs have reported some reduction in the amount of time prior to delivery of the first dose of analgesic medication. This depends on the ability of the nurse at triage to detect patients in pain, although studies have shown that emergency nurses frequently underestimate patients' pain intensity. Overcrowding also slows the ability of the ED staff to assess and treat patients in pain.

Many EMS systems now have protocols that allow paramedics to administer pain medication to patients during transport. These protocols vary as to whether this requires a real-time, on-line medical control order.

Self-report assessment

The most reliable approach to assessing pain severity is patient self-report. Self-report tools are the mainstay of pain management research, but require that patients have cognitive and communication skills. The ideal self-report tool should be easy to use and applicable across language, cultural, age and gender differences. It should also be valid and reliable between observers. Table 9.1 describes several commonly used tools for pain assessment in the ED.

Most of these tools are numerical. The Adjectival Rating Scale features six phrases describing pain intensity in ascending order, arrayed on a 10-cm baseline. It offers the same information as numerical tools without using numbers, an advantage for patients who cannot describe their pain numerically.

The Numerical Rating Scale is the most commonly used pain scale. It involves asking the patient to rate his or her pain on a scale from 0 to 10. In this scale, 0 is equivalent to

Table 9.1 Self-report assessments for pain

Adjectival	None	Mild	Moderate	Severe	Very severe	Worst possible	Comments
Numerical	0		5			10	Routine bedside evaluation
Visual analog scale (VAS) (10-cm baseline)	None					Worst imaginable	When hard copy needed
Hurt thermometer	White		Blue			Red	Bedside
Pictorial (FACES)							>6 years old
Pieces of Hurt (poker chips)	0	1	2		3	4	>3 years old
Thumb-to-index finger distance							Some toddlers

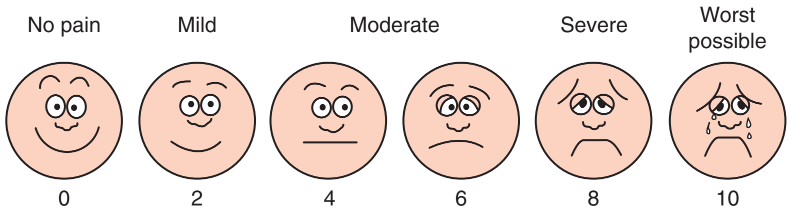

Figure 9.1
Wong-Baker FACES Pain Rating Scale. Instructions: Point to each face, using the words to describe the pain intensity. Ask the child to choose the face that best describes his or her own pain, and record the appropriate number. This rating scale is recommended for persons age 3 years and older. From Hockenberry MJ, Wilson D, Winkelstein ML, *Wong's Essentials of Pediatric Nursing*, 7th ed, St. Louis, 2005, p. 1259. Used with permission. Copyright Mosby.

no pain, 1 is equivalent to barely perceptible pain, and 10 represents the greatest pain that the patient has ever experienced or could imagine. However, even adults who are native speakers of the same language as the care providers have difficulty with this concept. Adults conversing in their second or non-native language may not be able to understand this scale or be able to express their pain adequately. Most children do not understand this at all: "big hurt," as opposed to "little hurt" may be the most that they can manage verbally.

The Visual Analog Scale (VAS) is the most widely used scale for clinical research. This is a 100-mm scale that has "no pain" on the left end and "maximum possible pain" on the right. Patients indicate a point on the scale to correspond to their level of pain. Visual, manual and some conceptual skills are required for patients to be able to do this. Patients seem able to reliably indicate a point to describe the level of their pain, and to shift this point in an expected direction after therapy. The major limitation of the VAS is that the distance that constitutes a significant clinical change has not been validated. Most studies indicate that a change of 13 mm constitutes a statistically significant change, but this does not necessarily correlate with clinical significance.

The Faces Scale (Figure 9.1) seems to work well for younger school-age children. The scale is self-explanatory and has strong agreement among children about the severity of pain reflected in the faces. The scale has also demonstrated adequate test–retest reliability.

The Hurt Thermometer Scale has faces superimposed on a scale on which the left end is white and represents no pain. From left to right, the color progresses from blue to red, with the bright-red end at the right representing maximal pain. This probably has no advantage in the assessment of pain in children, but may help assess pain in patients whose primary language differs from that of health care team members.

The Poker Chip Tool or Pieces of Hurt Scale works well for preschool children. The child gives between one and four poker chips to the care provider to indicate the "size" of pain the child is currently experiencing. For even younger children, the thumb-to-index-finger measurement offers another modality of pain communication. The child indicates the severity of his or her pain by spreading the thumb from the index finger. Children seem able to grasp subunit quantity when expressed as a change in the relationship of body parts at a much younger age than they can with objects such as building blocks.

Non–self-report assessment

Infants, toddlers, cognitively impaired patients, and those who do not speak the language of the health care team cannot effectively communicate their pain by the usual self-report scales. The physician must therefore carefully search for cues that suggest the presence of pain. Soliciting comments from caregivers may help providers assess their pain and the treatment's effectiveness.

Neonates have a limited repertoire of expression, and their ability to show body posturing is even further limited by wrapping or swaddling. Evaluation of neonatal facial expressions provides the best estimate for level of pain, even when their face is partially obscured by a nipple or pacifier. Of 10 possible facial actions in neonates, three provide the most reliable indicators of pain: the furrowed brow, the forehead bulge (just above the eyebrows), and squeezing of the eyes. Other facial actions include the nasolabial furrow (which can be obscured by a pacifier), open lips, horizontal and vertical mouth stretch, taut tongue, chin quiver, lip purse and tongue protrusion. The cry in response to pain tends to be more high-pitched and drawn out than the usual cry for food or diaper changing. Caregivers are often able to describe how the current cry differs from the usual cry, and whether or not the baby is more difficult to console. Moaning or whimpering is not normal for a neonate.

The FLACC scale (face, legs, activity, cry and consolability) is sometimes useful in infants (Table 9.2). Facial distortions due to pain are described above. The limbs are assessed for rigidity and muscle tone. In infants with severe cerebral palsy or known spasticity, this scale may not prove helpful in the assessment of pain. Crying and consolability are assessed with the help of the caregivers.

Assessment of pain in patients with limited communication skills is extremely challenging. Patients with developmental disabilities or cognitive impairment are often unable to express pain. It is unclear whether their neurologic impairment means that these patients do not actually experience pain or if the pain experience is diminished for them. Valid and reliable tools for assessment of pain in patients with significant neurologic impairment do not exist. As much as possible, health care providers

Table 9.2 FLACC pain scale (each category is scored from 0 to 2, totals up to 10)

Categories	0	1	2
Face	Smile or no expression	Occasional grimace or frown; withdrawn	Quivering chin, clenched jaw
Legs	Normal position or relaxed	Uneasy, tense, restless	Kicking, legs drawn up
Activity	Lying quietly, moves easily	Squirming, tense shifting back and forth	Arched, rigid, jerking
Cry	No cry	Moans or whimpers; occasional complaints	Crying, screaming, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touch, hug, or talk; distractible	Difficult to console or comfort

should keep caregivers at hand to assist with communication and management, maintain typical means of communication (e.g., patient's laptop), maintain typical means of comfort and mobility (e.g., wheelchair, form board), and remember that improved function may not mean that the pain has completely abated.

Treatment of pain

Expediting relief

Patients generally wait too long for their pain to be treated. Untreated pain has physiologic consequences and must be mitigated as soon as possible. Treatment should begin *even before* a definitive diagnosis has been established. Multiple studies have shown that patients with undifferentiated abdominal pain can receive analgesics safely without worse outcomes. This is true even in children.

The need for future informed consent is often given as a reason for withholding pain therapy. The concern that analgesics may compromise a patient's decision-making capacity (ability to understand and sign an informed consent form for a procedure) is unfounded. In fact, it may be considered coercive to withhold pain medication in order to gain consent. Pain itself can alter mood and thought. Analgesia can be titrated so that the patient's consciousness is not clouded. If the patient is overly sedated, as a last resort, an opioid antagonist can be administered.

Safety, speed of onset and ease of pain medication administration are key elements to pain relief in the ED setting. First, the patient must be monitored, and safety measures (such as putting up the gurney's side rails) must be instituted. The agent and route of administration must ensure rapid onset of adequate analgesia. In general, IV or inhalational routes ensure the fastest onset of action. Sophisticated techniques exist for delivering analgesia to specific sites. Many of these, such as intrathecal opioids, are too complicated and cumbersome for use in the ED. Sometimes, establishing an IV can be extremely difficult, especially in toddlers or chronically ill patients with friable or scarred veins. Transmucosal absorption of drugs (fentanyl) may provide relief of pain and may help improve cooperation during attempts at IV access. Intranasal administration of ketamine, midazolam or sufentanil offer another alternative. Orally administered analgesics may also provide relief in a more timely fashion if IV administration is likely to be delayed more than 20 minutes.

Nonpharmacologic modalities

Physical and psychologic comfort measures set the tone for an ED visit and help relieve pain and anxiety while preparations are underway for providing pharmacologic analgesia. Physical comfort measures include positioning the patient to minimize discomfort (e.g., in patients with musculoskeletal back pain), adjusting the lighting of the room (e.g., for patients with photophobia from migraine headache), ensuring that the patient is warm enough by providing blankets, placing ice packs on the site of injury, and immobilizing, elevating and supporting injured extremities.

Fear and anxiety exacerbate a patient's pain and suffering. The ED is unfamiliar to most patients, and the patients feel dependent on strangers for help. Patients may also fear that their injury may result in permanent disability, or that their pain may be due to cancer. Young children often fear that their pain is punishment for perceived misdeeds, and often believe that the body part that hurts will be amputated. Anxiety and anger on the part of family members may also heighten a patient's pain. Early reassurance that the patient and his or her family and friends will be treated with respect and compassion helps decrease suffering and ameliorate pain. Offering choices whenever possible (e.g., where the IV will be placed) lessens the feeling of loss of control. Letting a patient know approximately how long it will take to obtain medication, before the medication begins working, and whether to expect relief to be partial or full are also important.

Music, storytelling, blowing bubbles, and other verbal or imagery techniques can distract children from a painful procedure as well as reduce anxiety (Table 9.3). Child Life Department personnel, if available, can be invaluable by providing positive interactions with children and caregivers.

For neonates and young infants undergoing painful procedures (e.g., bladder catheterization, IV placement), oral sucrose solution administered by placing a saturated cotton ball in a nipple has been shown to significantly decrease the measured pain response.

Table 9.3 Analgesic modalities and their mechanisms of action

Distraction	Cognitive focus away from pain
Music	Cognitive focus away from pain and decreased anxiety
Hypnosis	Cognitive reinterpretation of painful stimuli
Biofeedback	Decreases muscle tension
Placebo	Activates descending pain inhibitory pathways; may involve endorphins
TENS	Interferes with transmission in dorsal horn ganglia; possibly stimulates endorphins
Acupuncture	Probably similar to TENS
Local anesthesia	Blocks transmission of afferent nerve impulses
NSAIDs	Block production of prostaglandins
Opioids	Bind to opiate receptors in CNS and possibly in peripheral nerves
Nitrous oxide	Blunts emotional reaction to pain; possible role of endogenous opioids

CNS: central nervous system; NSAIDs: nonsteroidal antiinflammatory drugs; TENS: transcutaneous electrical nerve stimulation.

Pharmacologic therapy

Pharmacologic therapy can be either curative or palliative. Relief of cardiac chest pain by the vasodilatory effect of nitroglycerin is an example of curative therapy. This chapter deals primarily with palliative therapy; once a diagnosis has been established, curative therapy is preferred over palliation alone if possible.

Non-opioid agents

Non-opioid agents are listed in Table 9.4.

Acetaminophen
Acetaminophen is an effective analgesic for mild to moderate pain. Its mechanism of action is unclear, yet it seems to act centrally. Acetaminophen has little anti-inflammatory effect and few gastrointestinal side effects. It does not affect platelet aggregation. Significant hepatotoxicity is known to occur with large overdoses.

Nonsteroidal antiinflammatory drugs (NSAIDs)
The mechanism of action of NSAIDs is thought to be due to inhibition of prostaglandin, and possibly leukotriene production. Alone, prostaglandins do not cause pain, but sensitize nerve endings to perceive an ordinary, non-painful stimulus as painful. NSAIDs are widely used for their antipyretic and anti-inflammatory properties, in addition to their analgesic properties. They are effective for mild to moderate pain, and their lack of respiratory depression and abuse potential makes them an attractive choice. A "ceiling effect" exists beyond which no further analgesia can be produced, even when a different NSAID is added. Their major side effects include gastrointestinal bleeding, renal failure, anaphylaxis, and platelet dysfunction. The same analgesics that are effective in adults can be safely administered to children greater than 2 months of age. In children, the margin of safety of these drugs approximately equals that in adults.

Aspirin
Aspirin may cause Reye's syndrome in children who contract influenza or chickenpox. Aspirin is now seldom used in children for its analgesic or antipyretic properties, except to treat autoimmune diseases such as juvenile rheumatoid arthritis.

Ketorolac tromethamine
Ketorolac is the first non-opioid analgesic agent available for parenteral use in the United States. For acute musculoskeletal pain, 60 mg of ketorolac administered IM has been shown to be approximately equivalent in analgesic efficacy to 800 mg of oral ibuprofen. Ketorolac inhibits prostaglandin synthesis, so its onset is no faster than that of an equivalent agent given orally. Ketorolac is considered to be most useful in the context of renal colic because decreased prostaglandin synthesis results in decreased ureteral peristalsis. In theory, opioids increase smooth muscle spasm and peristalsis; nonetheless, opioids have proven to be effective analgesics in renal colic and should be considered standard therapy.

Cyclooxygenase-2 specific inhibitors
Cyclooxygenase-1 (COX-1) serves as a "clean-up" or reparative agent and is not inducible with stimulation from inflammation or injury. COX-2 is present in lower levels and is inducible, showing increases that are closely related to the inflammatory response to injury or inflammation. Most traditional NSAIDs block both COX-1 and COX-2. The selective COX-2 inhibitors rofecoxib and celecoxib provide anti-inflammatory effects and moderate analgesia with a lower incidence of gastrointestinal side effects. Both are eliminated by the liver, and share similar drug interactions with standard NSAIDs. They may precipitate anaphylaxis in patients with aspirin allergy. Celecoxib is metabolized by the cytochrome P450 system and may cross-react in patients who have a sulfonamide allergy. Rofecoxib (Vioxx) has been taken off the market because of its association with an increased incidence of myocardial infarction.

General guidelines for choosing non-opioid analgesic agents

1. Use cautiously in the elderly, who are at greater risk of developing gastrointestinal bleeding, renal toxicity and renal failure.
2. Patients who are dehydrated or hypovolemic are at high risk of acute renal impairment.

Table 9.4 Non-opioid analgesics

Generic (proprietary)	Dose	Pediatric dose	Mechanisms of toxicity	Maximum dose
Acetaminophen (APAP)	650–1,000 mg PO Q 4–6 hrs 1 g PR Q 6 hrs 1–2 g PR Q 12 hrs	10–20 mg/kg PO 20–40 mg/kg PR Q 4 hrs	Not an NSAID. Exact mechanism unknown. Liver toxicity possible when above 150 mg/kg is taken in 24 hrs.	100 mg/kg/day (4000 mg/day)
Aspirin (ASA)	650–975 mg PO Q 4 hrs	10–15 mg/kg PO	Reye's syndrome in children who subsequently get flu or chickenpox. Tinnitus; toxic dose >150 mg/kg.	60 mg/kg/day
Ibuprofen (IB)	600 mg PO Q 6–8 hrs	10 mg/kg PO Q 6–8 hrs	GI irritation Platelet dysfunction Renal dysfunction Bronchospasm	40 mg/kg/day
Naproxen	250 mg PO Q 6–8 hrs 500–100 PR Q 12 hrs	5–7 mg/kg PO Q 12 hrs	Interacts with protein-bound drugs	20 mg/kg/day
Indomethacin	25–50 mg PO Q 12 hrs 100 mg PR Q 24 hrs	N/A	As for naproxen	3 mg/kg/day
Ketorolac	60 mg IM/dose 30 mg IV/dose	0.5 mg/kg IV Q 6 hrs	Same as IB. Decrease dose by one-half in elderly or if impaired renal function.	120 mg/day (adult), up to 72 hrs (pediatric use controversial)
Celecoxib	200 PO BID	Not approved	Not available as liquid; contraindicated in sulfa allergy. May increase risk of serious cardiovascular events.	
Valdecoxib	10 mg PO QD	Not approved	No renal elimination; should not be given to sulfa-allergic patients. May increase risk of serious cardiovascular events.	
Tramadol	50–100 mg PO	Not approved	May precipitate serotonin syndrome in SSRI patients (no actual pediatric indications, but studies support safety and efficacy in children)	

GI: gastrointestinal; BID: twice a day; IB: ibuprofen; IM: intramuscular; IV: intravenous; NSAID: nonsteroidal antiinflammatory drug; PO: per os; PR: per rectum; Q: every; QD: once daily; SSRI: selective serotonin reuptake inhibitors.

3. All have the potential for gastrointestinal side effects.
4. All may interfere with the effects of many antihypertensives.
5. There is little clinical evidence of individual superiority of one particular agent over another.

Opioid analgesic agents

Opioid analgesics are the mainstay of pharmacologic management of acute, moderate to severe pain (Table 9.5). The beneficial physiologic and psychologic effects of opium have been well documented for centuries; so have its toxicity and potential for abuse. Fear of inducing addiction has led many physicians to underuse opioids. However, many studies have demonstrated that short-term use of opioid analgesics for acute pain syndromes is not associated with future dependence.

There are multiple opioid receptors, each affected by opioids in different ways. The most commonly used opioids are μ-agonists: morphine, meperidine, methadone, codeine, oxycodone and the fentanyls. An agonist acts as a neurotransmitter – when the receptor recognizes the agonist, it causes alterations within the cell. An antagonist blocks the receptor by occupying it without initiating transduction. Partial agonists produce a partial response with decreased intrinsic activity. By binding the receptor site, they also block access of full agonists and function as partial antagonists.

Table 9.5 Opioid analgesics

Generic (proprietary)	Oral equipotent dose, Adult (Pediatric)	Parenteral dose, Adult (Pediatric)	Duration (in hours)	Comments	Precautions
Morphine sulfate	30–60 mg (0.5 mg/kg)	10 mg (0.1 mg/kg)	3–5	Standard for comparison	Respiratory depression Hypotension Sedation Histamine release
Codeine	30–100 mg (2 mg/kg)	30–100 mg (0.5 mg/kg)	4	Poor analgesic Good cough suppressant	Constipation Nausea and vomiting Abuse potential
Hydromorphone (Dilaudid)	2–6 mg (0.02–0.1 mg/kg)	1–2 mg (0.015 mg/kg)	2–4	Available as suppository	Euphoria
Hydrocodone (Vicodin, Lortab)	5–10 mg	N/A	3–4	Good cough suppressant. Fewer side effects than codeine and greater potency	Greater abuse potential
Oxycodone (Percocet, Tylox both with APAP)	(0.05–0.15 mg/kg)	N/A	3	Parenteral form not available in the US. Very effective analgesic.	Euphoria Abuse potential
Meperidine (Demerol)	250–300 mg (1.5–2 mg/kg)	75–125 mg (1 mg/kg)	2–3	Toxicity from metabolite normeperidine	Avoid with MAOI. Caution in renal or hepatic failure.
Fentanyl	N/A	0.05–0.2 mg (0.001 mg/kg)	1–2	No histamine release. Transcutaneous and transmucosal absorption.	For IV administration, push and flush slowly to avoid "rigid chest" syndrome
Alfentanil	N/A	1 mg/kg (0.01 mg/kg)	1.5	Shortest half-life, minimal cardiovascular side effects	Muscular rigidity if administered too quickly; expensive

APAP: acetaminophen; IV: intravenous; MAOI: monoamine oxidase inhibitor.

Morphine

Morphine is the gold standard opioid agent. In standard dosage, it produces analgesia without loss of consciousness. Relief of tension, anxiety and pain results in drowsiness and sleep. Nausea, vomiting, pruritus and miosis are the most common side effects. Vasodilatation and venous pooling from morphine do not cause significant hemodynamic effects in normovolemic patients, but can cause significant hypotension in hypovolemic patients. Morphine causes dose-dependent depression of ventilation, reducing the respiratory rate and then tidal volume. Morphine can increase sphincter tone at the pylorus, ileocecal junction and sphincter of Oddi, and decreases peristalsis, resulting in constipation.

Fentanyl

Fentanyl's advantages over morphine include a rapid onset (<1 minute) and brief duration of action (30–45 minutes). It is 50–100 times more potent than morphine and has little hypnotic or sedative effect. Fentanyl's main disadvantage is the glottic and chest wall rigidity that may develop after rapid infusion of higher doses (>5 mcg/kg). The mechanism of the "rigid chest" syndrome is unclear but can be life-threatening, since assisted ventilation may be impossible without muscle relaxants.

Hydromorphone

Hydromorphone is a derivative of morphine, and has greater selectivity for μ-opioid receptors. It has a rapid onset of action and lasts 4–6 hours. Hydromorphone is five times more potent and 10 times more lipid soluble than morphine, yet less sedating. It can be given IM, and also produces less nausea.

Methadone

Methadone does not follow first-order kinetics and should not be initiated or titrated in the ED. It is the only opioid that has a nonlinear relationship to standard opioids, with decreased equipotency for patients receiving <1,000 mg/day of morphine. Emergency physicians should consult with pain or palliative care consultants prior to adjusting any dosage.

Meperidine

Meperidine's principal metabolite, normeperidine, has no analgesic properties and causes significant toxicity (tremulousness, seizures) as it accumulates. Normeperidine's half-life is 6 hours, compared with the 3-hour half-life of meperidine; it accumulates quickly with repeated dosing. Because of its euphoria-producing effect, extreme addiction and dependence can result from this drug. Given this and its toxicity, many institutions have removed meperidine from their formularies.

Opioid drug selection

The idea that some opioids are weak and ineffective in severe pain is outdated. In equipotent doses, opioid agents can achieve the same effect as other opioids, but differ in their side effects and half-life. Factors affecting drug selection include the intensity of the pain, coexisting disease, potential drug interactions, treatment history, physician preference, patient preference and proposed route of administration.

Choice of route of administration

Injectable

Once an IV has been established, this route results in the shortest time to onset of pain relief. There is no "maximal" dose of opioid, and induction of undesired side effects usually signals the limit of the patient's ability to tolerate the drug.

The IM route has multiple disadvantages. Among them, the pain of injection limits the physician's ability to titrate the drug effect. Furthermore, drug uptake is variable, depending on the patient's peripheral circulation and body composition.

Patient-controlled analgesia (PCA) is commonly used in the inpatient setting for severe pain expected to last hours or days. In general, PCA does not have a role in the ED setting, but may be beneficial to patients in observation units or for those whose ED stay is prolonged due to lack of inpatient beds.

Oral

First-pass hepatic metabolism may inactivate as much as 80% of an oral opioid dose. Patients who will require general anesthesia cannot take anything by mouth. Patients who are vomiting may not be able to hold down the drug long enough for absorption to occur. Time to onset of analgesia is much longer and titration is more difficult. Outpatient pain control after discharge is the main reason to use oral opioids.

Rectal

The rectal route has the advantages of transmucosal absorption without the first-pass effect. Additionally, it does not rely on gastric motility. Absorption, however, is variable. Patients may object to this route of administration. Hydromorphone is the only opioid available as a suppository; the IV form can also be given rectally.

Transmucosal

Fentanyl lollipops are the most common form of opioid using the transmucosal route. This is especially helpful in children, but requires patient cooperation. Atomized fentanyl can be delivered nasally and is efficacious in treating pain.

Combination therapy

The combination of non-opioid and opioid agents produces significantly greater pain relief than either agent alone.

Use of adjuvant agents

Adjuvant agents are used in combination with opioids for various reasons: to provide synergy, to decrease side effects, to decrease anxiety, and to relax muscles, especially in acute musculoskeletal pain. The phenothiazines and hydroxyzine are most commonly used. There is no evidence, however, for analgesic synergy with these agents. Phenothiazines do not potentiate analgesia, as previously believed, and may actually diminish the analgesic effect of the simultaneously administered opioid. Hydroxyzine not only requires an additional injection, but also increases respiratory depression. In severe musculoskeletal pain associated with muscle spasm, the addition of a muscle relaxant may provide more relief than an opioid alone. In this scenario, respiratory status must be monitored closely. In general, adjuvant agents provide little additional analgesia and may potentiate or add side effects to the clinical picture.

Special patients

Intubated and paralyzed patient

A patient who has just undergone rapid sequence intubation is unable to move or communicate. It is possible to forget that he or she is still conscious once the induction agent has worn off. Bonomo et al. identified that well over half of intubated patients who remained in the ED for more than 30 minutes received no or inadequate analgesia or anxiolysis.

Undifferentiated abdominal pain

Fear of masking the clinical findings and missing the diagnosis has long prevented physicians from giving opioids to patients with undifferentiated abdominal pain, leaving them to suffer for hours during attempts to establish a diagnosis and definitive treatment. This practice was first described by Cope in his 1921 *The Early Diagnosis of Abdominal Pain*. "If morphine be administered, it is possible to die happy in the belief that he is on the road to recovery, and in some cases, the medical attendant may for a time be induced to share the same delusive hope." Newer diagnostic techniques, better monitoring and more accurate opioid titration have made his dire warning obsolete. In fact, the most recent edition of Cope's textbook retracts this myth. Several studies have documented that early pain relief in patients with acute abdominal pain is safe and does not result in diagnostic errors, delay in surgery, or worse outcomes, even in children.

Migraine headaches

There have been many studies comparing the effectiveness of non-opioid agents to opioids in the management of migraine headaches. The phenothiazines have shown success rates as high as 95%. Sumatriptan and dihydroergotamine have been associated with recurrence rates as high as 50%, especially in patients with persistent headache at the time of discharge from the ED. Many other drugs (metoclopramide, haloperidol, droperidol, NSAIDs, valproate and narcotics) have been studied. Intranasal lidocaine and trigger point injections have also been rigorously studied, with variable rates of success. The relative benefit of any of these drugs or any combinations has yet been satisfactorily established. Opioids have not been shown to perform better in clinical trials and have the potential to be associated with subsequent drug-seeking behavior.

Chronic pain

A patient with a terminal illness and chronic pain should receive generous amounts of opioids while the physician searches for a new process responsible for increased pain. These patients will have great tolerance for the analgesic effects of opioids, but not necessarily for their side effects. Patients who have chronic pain with a non-terminal illness should be under the care of a primary care provider who has a plan for managing this pain. Close consultation with that primary care provider or the pain management team, if applicable, will optimize the patient's care and reduce dependency and abuse.

Suspected drug seeker

Some patients will feign pain or claim pain syndrome diagnoses in order to receive opioids, either for their own use or to sell. Suspected drug-seeking behavior should be documented and becomes evident as the number of ED visits increase. In general, it is better to err on the side of humane treatment than to deprive a patient of needed pain relief. Diligence in checking the history and physical examination for inconsistencies, communicating with the patient's primary care provider, and checking the medical and pharmacy records will help identify drug seekers and drug-seeking behavior. Many patients with suspected drug-seeking behavior "lose" their prescriptions, travel without their pain medication, or "run out" of their medication, often on the weekend or when their primary care provider is unavailable. Non-narcotic medications should be substituted when possible. Prescriptions should be written for small amounts of medication without refills, with numbers written numerically and spelled out. Communication between the primary care provider and ED personnel serves not only to confirm the physician's suspicions, but can also provide the basis for a consistent care plan for future visits. Documentation of findings and discussions are necessary parts of the medical record.

Pearls, pitfalls and myths

Pearls

- Treat pain early and often; anticipate pain prior to its recurrence.
- Reassess patients frequently; remember that paralyzed patients still feel pain.
- Use enough agent to achieve the desired effect of pain relief, or until an undesirable side effect occurs. Switch to a different agent if side effects occur and pain persists, or if the initial agent is not effective.
- Select the route of administration that allows the fastest relief for the patient but neither delays definitive care nor causes unnecessary, additional discomfort.

Pitfalls

- *Wrong agent*: Most opioids can achieve the desired degree of analgesia. A major exception is oral codeine. Codeine is a weak agonist with a high incidence of nausea, vomiting and constipation; it has not been shown to be more effective than ibuprofen or acetaminophen alone.
- *Wrong dosage:* Titrate the dosage to achieve the desired degree of analgesia. There is no "maximal" dose of any opioid.
- *Wrong route*: The IM route has several disadvantages: pain, delayed onset of action, unpredictable uptake, difficult and painful titration, and complications such as hematoma formation or damage to underlying structures.
- *Wrong frequency*: Preventing pain from recurring by earlier readministration of opioid analgesia will result in less opioid use overall than retreating pain that has had time to reestablish itself.
- *Incorrect use of adjuvant agents*: Adjuvant agents do not reduce the dosage of opioid needed. Antiemetics may be used if nausea and vomiting persist after adequate analgesia has been achieved. The sedation or respiratory depression that occurs with most of the commonly used adjuvant agents is undesirable.

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Section 2

Primary Complaints

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10 Abdominal pain

S.V. Mahadevan, MD

Scope of the problem

Evaluation of the patient with acute abdominal pain is one of the most challenging aspects of emergency medicine. Seven percent of emergency department (ED) patients may present with abdominal pain. Diagnostic possibilities range from immediately life-threatening (e.g., ruptured abdominal aortic aneurysm [AAA]) to self-limiting (e.g., abdominal wall strain), and from common (e.g., gastroenteritis) to unusual (e.g., black widow spider bite). Benign-appearing presentations may progress to life-threatening conditions. Though the etiology of pain remains undiagnosed in as many as 40% of patients, recognition of surgical or life-threatening causes is more important than establishing a firm diagnosis.

Anatomic essentials

Abdominal pain is typically derived from one or more of three distinct pain pathways: visceral, parietal (somatic) and referred.

Visceral abdominal pain

Visceral abdominal pain is most commonly caused by distention, contraction or stretching of hollow and solid organs, and is often the earliest manifestation of a particular disease process. It may vary from a steady ache or vague discomfort to excruciating or colicky pain. For organs affected by peristalsis, the pain is often described as intermittent, crampy, or colicky in nature.

Because visceral pain fibers are bilateral, unmyelinated, and enter the spinal cord at multiple levels, visceral abdominal pain is usually dull, midline and poorly localized. Visceral pain is perceived as epigastric, periumbilical, or infraumbilical depending on the involved organ's embryonic origin. Foregut structures, such as the stomach, duodenum, liver, biliary tract and pancreas, produce upper (epigastric) abdominal pain. Midgut structures, such as the small bowel, appendix and proximal colon, cause periumbilical pain. Hindgut structures, such as the distal colon and genitourinary system, cause lower (infraumbilical) abdominal pain.

Parietal (somatic) abdominal pain

Parietal or somatic abdominal pain results from ischemia, inflammation or stretching of the parietal peritoneum. Myelinated afferent fibers transmit the painful stimulus to specific dorsal root ganglia on the same side and dermatomal level as the origin of the pain. For this reason, parietal pain, in contrast to visceral pain, often can be localized to the region of the painful stimulus. This pain is typically sharp, knife-like and constant; coughing and moving are likely to aggravate it. Conditions resulting in parietal pain often account for physical examination findings of tenderness to palpation, guarding, rebound and rigidity.

Abdominal conditions often begin with visceral pain and progress to somatic pain. The pain of early appendicitis is often periumbilical (visceral) but localizes to the right lower quadrant (parietal) as the inflammation extends to the peritoneum. Similarly, cholecystitis may begin as epigastric (visceral) pain and progress to right upper quadrant (somatic) pain and/or right shoulder (referred) pain.

Referred pain

Referred pain is defined as pain felt at a distance from the diseased organ. It results from shared central pathways for afferent neurons from different locations. For instance, a patient with pneumonia may present with abdominal pain because the T9 distribution of neurons is shared by the lung and abdomen. Other examples of referred pain include epigastric pain associated with myocardial infarction (MI), shoulder pain associated with diaphragmatic irritation (e.g., ruptured spleen), right infrascapular pain associated with biliary disease, and testicular pain associated with acute ureteral obstruction.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 10.1).

History

In patients with abdominal pain, a careful and focused history is the key to uncovering the etiology of most cases.

Where is your pain? Has it migrated?

The location of abdominal pain often corresponds to specific disease entities and is very important for

Table 10.1 Abdominal pain red flags

History	Concerning diagnosis
Sudden onset	AAA, aortic dissection, ectopic pregnancy, mesenteric ischemia, viscus perforation
Pain out of proportion to exam	Mesenteric ischemia
Pregnant	Ectopic pregnancy
Pain with walking or hitting bumps in the road on the car ride	Appendicitis, peritonitis
Elderly	AAA, mesenteric ischemia, MI, perforated ulcer, bowel obstruction, appendicitis, cholecystitis
Trauma	Intra-abdominal injury (e.g., spleen, liver)
Syncope	Ectopic pregnancy, AAA
Tearing pain or history of vascular disease	AAA, aortic dissection
History of or risk factors for coronary artery disease	MI, mesenteric ischemia
History of abdominal surgery or bowel obstruction	Bowel obstruction
Examination finding	**Concerning diagnosis**
Hypotension	AAA, ectopic pregnancy
Peritoneal signs or rigidity	Acute surgical emergency
Abdominal distention or bilious emesis	Bowel obstruction, volvulus, malrotation
Pulsatile mass	AAA
Fever, RUQ pain, jaundice	Cholangitis
Ascites	SBP
Blood in stool	Colitis, intussusception, inflammatory bowel disease, cancer
Benign abdominal exam (but severe pain)	Mesenteric ischemia, MI

AAA: abdominal aortic aneurysm; MI: myocardial infarction; RUQ: right upper quadrant; SBP: spontaneous bacterial peritonitis.

the development of an initial differential diagnosis (Figure 10.1). Keep in mind that the location of abdominal pain may vary with time, especially as the underlying disease evolves and the pain progresses from visceral to somatic. Periumbilical pain that migrates to the right lower quadrant (RLQ) is very specific for appendicitis, whereas epigastric pain that localizes to the right upper quadrant (RUQ) is classic for biliary disease.

Does the pain radiate anywhere?

The pain of biliary colic may radiate to the right infrascapular region; the pain of pancreatitis to the midback. Pain that radiates to the flank or genitals may represent a kidney stone or ruptured AAA.

How did the pain begin (sudden vs. gradual onset)? How long have you had the pain?

Sudden or abrupt onset of abdominal pain often indicates a serious underlying disorder, such as mesenteric ischemia and intestinal perforation. Fainting or collapsing with such pain is worrisome for conditions such as a ruptured AAA, perforated ulcer or ectopic pregnancy. Inflammatory or infectious causes of pain (e.g., cholecystitis, appendicitis, diverticulitis) tend to develop over hours to days and are generally less severe at onset. Pain for greater than 6 hours or less than 48 hours duration, or pain that is steadily increasing in intensity is more likely to require surgical intervention.

What were you doing when the pain began?

Severe pain that awakens a patient from sleep is concerning and may represent perforation or ischemia. Pain related to eating may be from peptic ulcer disease, biliary disease or mesenteric ischemia (intestinal angina). A history of abdominal pain following trauma raises concern for an intra-abdominal injury.

What does the pain feel like?

The significance of the patient's characterization of pain (visceral, somatic, referred) is described in detail earlier in this chapter. Classic descriptions of pain include the burning or gnawing pain of peptic ulcer disease, the colicky pain of biliary colic, the penetrating pain of pancreatitis, the tearing pain of an aortic dissection, and the crampy, intermittent pain of intestinal obstruction.

On a scale of 0–10, how severe is the pain?

Unfortunately, the patient's quantification of pain severity is often inconsistent and generally unreliable in determining the specific cause of pain. Studies have shown that elderly patients tend to have a higher pain threshold than younger patients. In general, nonsurgical causes of pain tend to be less painful than surgical etiologies. Although acute nephrolithiasis (kidney stone) may present with severe, incapacitating pain, the majority of patients will spontaneously pass their stone without surgical intervention. The finding of

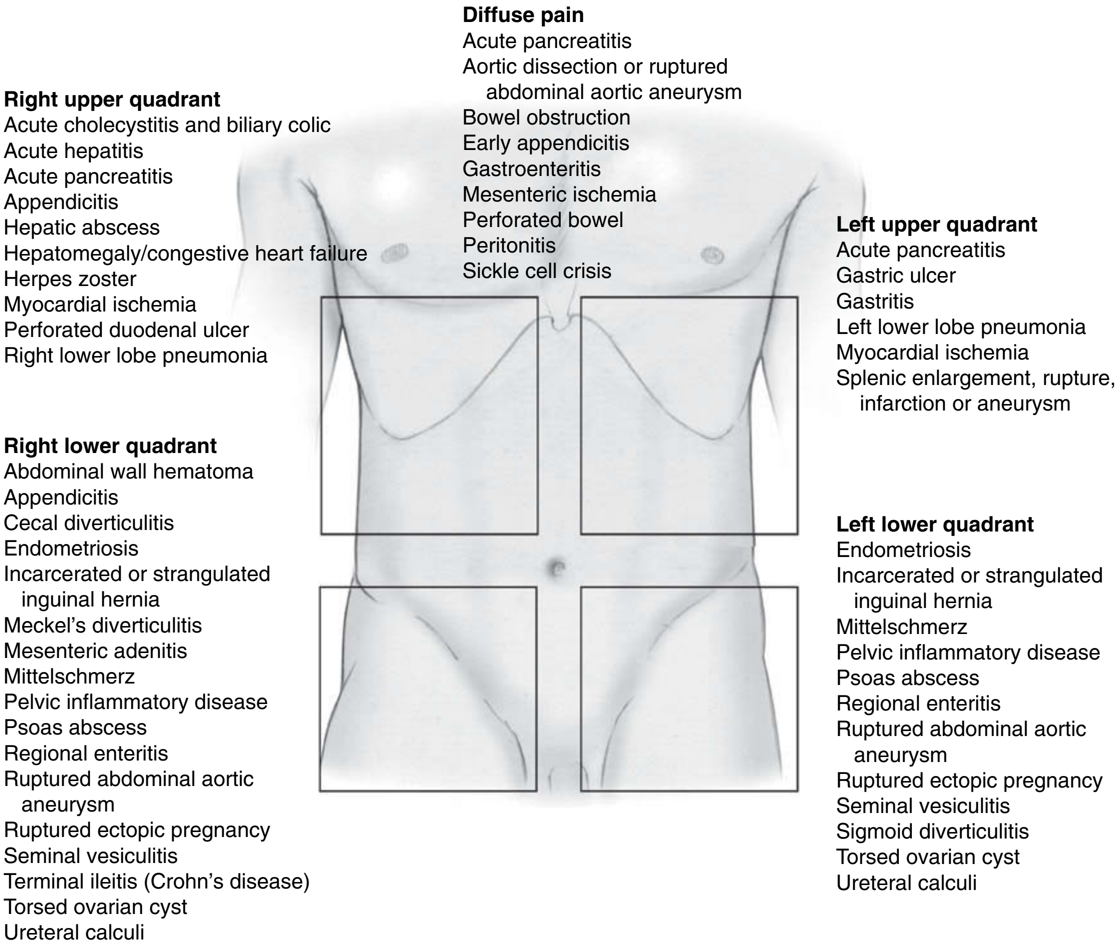

Diffuse pain
Acute pancreatitis
Aortic dissection or ruptured abdominal aortic aneurysm
Bowel obstruction
Early appendicitis
Gastroenteritis
Mesenteric ischemia
Perforated bowel
Peritonitis
Sickle cell crisis
Right upper quadrant
Acute cholecystitis and biliary colic
Acute hepatitis
Acute pancreatitis
Appendicitis
Hepatic abscess
Hepatomegaly/congestive heart failure
Herpes zoster
Myocardial ischemia
Perforated duodenal ulcer
Right lower lobe pneumonia
Left upper quadrant
Acute pancreatitis
Gastric ulcer
Gastritis
Left lower lobe pneumonia
Myocardial ischemia
Splenic enlargement, rupture, infarction or aneurysm
Right lower quadrant
Abdominal wall hematoma
Appendicitis
Cecal diverticulitis
Endometriosis
Incarcerated or strangulated inguinal hernia
Meckel's diverticulitis
Mesenteric adenitis
Mittelschmerz
Pelvic inflammatory disease
Psoas abscess
Regional enteritis
Ruptured abdominal aortic aneurysm
Ruptured ectopic pregnancy
Seminal vesiculitis
Terminal ileitis (Crohn's disease)
Torsed ovarian cyst
Ureteral calculi
Left lower quadrant
Endometriosis
Incarcerated or strangulated inguinal hernia
Mittelschmerz
Pelvic inflammatory disease
Psoas abscess
Regional enteritis
Ruptured abdominal aortic aneurysm
Ruptured ectopic pregnancy
Seminal vesiculitis
Sigmoid diverticulitis
Torsed ovarian cyst
Ureteral calculi

Figure 10.1
Differential diagnosis of acute abdominal pain by location. Adapted from Wagner DK. *Curr Topic* 1978:**1**(3).

severe pain "out of proportion" to physical examination is worrisome for mesenteric ischemia.

Does anything make the pain better or worse?

Movement aggravates parietal peritoneal pain. Pain that worsens with walking or after hitting bumps on the car ride to the hospital raises concern for conditions like appendicitis. The pain of peptic ulcer disease typically improves with eating, whereas biliary colic worsens with meals. Pain accentuated by reclining and relieved by sitting upright should raise suspicion for a retroperitoneal process such as pancreatitis. Abdominal pain relieved by vomiting suggests a gastric or proximal bowel problem, whereas relief of pain after a bowel movement suggests a colonic process. The pain of myocardial ischemia may worsen with activity and improve with rest.

Have you had the pain before?

Recurrent episodes of pain generally suggest a medical cause; however, mesenteric ischemia, biliary disease and partial bowel obstruction are exceptions. Prior pain events may occur in up to 71% of patients with cholecystitis. In patients with recurrent abdominal pain, attempt to identify triggers for the pain and obtain the results of previous laboratory studies or diagnostic imaging.

Associated symptoms

Gastrointestinal

Ask about nausea, vomiting, anorexia, constipation, diarrhea or bleeding. Nausea and vomiting may result from irritation of intra-abdominal organs or obstruction of an involuntary muscular tube (i.e., intestine, bile duct, ureter). Consequently, nausea and vomiting are common to many abdominal processes, including appendicitis. However, vomiting may also be slight or absent from many serious surgical conditions (e.g., ectopic pregnancy, intussusception). The temporal relationship of abdominal pain and vomiting is another key historical finding. Classically, patients with appendicitis or other surgical causes of abdominal pain develop pain prior to vomiting. The reverse is often true in medical conditions, where vomiting may precede pain. Any child presenting with bilious vomiting raises concern for an acute bowel obstruction.

Contrary to popular belief, anorexia is not a requisite finding for the diagnosis of appendicitis, as it is absent in 10–30% of cases. Constipation and diarrhea occur with equal frequency (15% of cases) in appendicitis. Diarrhea may accompany a partial small bowel obstruction (SBO) despite the common misconception that any bowel movement excludes this condition. Bloody diarrhea is suggestive of inflammatory bowel disease or infectious enterocolitis. A bloody or "currant jelly" (blood and mucus) stool may indicate intussusception, although this is generally a late finding. Failure to pass flatus or feces could be associated with an intestinal obstruction.

Genitourinary

Ask about dysuria, frequency, urgency and hematuria. Though dysuria and urinary frequency are classic symptoms of a urinary tract infection (UTI), they can also occur as the result of bladder irritation by an inflamed appendix or pelvic organ. Gross hematuria may indicate bladder irritation (e.g., infection, tumor) or nephrolithiasis.

Gynecologic

Ask about pregnancy, menses, contraception, fertility, sexual activity, sexually transmitted infections (STIs), vaginal discharge or bleeding, and dyspareunia. Previous gynecologic history including surgeries, previous pregnancies and infections is also important. A patient may mistake abnormal vaginal bleeding for their menses. Painful menses in a patient without a history of dysmenorrhea should raise concern for a serious gynecologic condition. Ectopic pregnancy should be considered in *all* female patients between the ages of 9 and 50 years with abdominal pain.

Pregnancy not only alters the diagnostic possibilities in a patient with acute abdominal pain but can also change the clinical findings. Advanced pregnancies make the diagnosis of appendicitis more difficult – not only does the location of the appendix change with the progression of the pregnancy, but these patients tend to have fewer clinical findings than non-pregnant patients.

Cardiopulmonary

Ask about cough, dyspnea and chest pain. Pneumonia, pulmonary embolism (PE) and acute MI may present with abdominal pain as the chief complaint. Abdominal pain may be the result of other extra-abdominal causes (Table 10.2).

Past medical

Previous abdominal surgery is an important risk factor for bowel obstruction due to adhesions. Patients with a history of cardiovascular disease, hypertension or atrial fibrillation are at risk for mesenteric ischemia and AAA. Patients with a history of cirrhosis and ascites are at risk for spontaneous bacterial peritonitis. Ongoing medical illnesses such as diabetes, heart disease, or chronic obstructive pulmonary disease (COPD) may complicate the evaluation and stabilization of patients with abdominal pain. Certain medications (i.e., nonsteroidal antiinflammatory drugs [NSAIDs], corticosteroids, antibiotics, immunosuppressants) may lead to abdominal pain or make its evaluation more challenging. Alcohol consumption places patients at risk for GI bleeding, pancreatitis, hepatitis or cirrhosis.

Table 10.2 Important extra-abdominal causes of abdominal pain

Systemic causes
Diabetic ketoacidosis
Alcoholic ketoacidosis
Uremia
Sickle cell disease
Porphyria
Systemic lupus erythematosus
Vasculitis
Glaucoma
Hyperthyroidism

Toxic
Methanol poisoning
Heavy metal toxicity
Scorpion bite
Black widow spider bite

Thoracic
Myocardial infarction
Unstable angina
Pneumonia
Pulmonary embolism
Herniated thoracic disk

Genitourinary
Testicular torsion
Renal colic

Infectious
Strep pharyngitis (more often in children)
Rocky Mountain spotted fever
Mononucleosis

Abdominal wall
Muscle spasm
Muscle hematoma
Herpes zoster

Adapted from Purcell TB. Nonsurgical and extraperitoneal causes of abdominal pain. *Emerg Med Clin North Am* 1989;7:721–40.

Physical examination

The primary goal of the physical examination is to localize the organ system responsible for disease. In addition to the abdomen, it is important to examine other body areas that may provide clues to the etiology of the pain, especially the pelvic (women), genitourinary (men), back and rectal areas.

General appearance

The general appearance of a patient is an important clinical observation. As a general rule, patients with pallor or distress are generally more acutely ill. Patients whose disease process has progressed to peritonitis tend to lie still to avoid exacerbating their pain. Patients with ureteral colic or mesenteric ischemia may writhe in pain because they cannot find a position of comfort. Nonspecific abdominal pain, gastroenteritis and ureteral colic are usually less aggravated by movement.

Vital signs

The absence of fever, often used as a marker to identify infection, can be deceiving in patients with abdominal pain. Diseases such as appendicitis and cholecystitis may present with temperatures <100.2°F (37.8°C). Elderly or immunocompromised patients may not mount a fever or may present with hypothermia despite a serious underlying illness. The majority of elderly patients with acute appendicitis or cholecystitis are afebrile in spite of higher rates of perforation and sepsis.

The presence of fever should alert the physician to the possibility of infection as the cause of pain. An acute onset of a high fever and chills make appendicitis less likely than pneumonia or pyelonephritis in the appropriate clinical setting.

Other vital signs may be helpful in assessing the degree to which a patient is affected by his or her illness. Hypotension may be a result of dehydration, sepsis or internal hemorrhage, and is a worrisome finding in an elderly patient. Tachycardia may signify occult blood loss, sepsis, volume contraction or pain. However, medications such as beta-blockers may blunt the patient's ability to mount such a response. An increased respiratory rate may result from severe pain, metabolic acidosis or an extra-abdominal cause such as PE, pneumonia or MI.

Abdomen

Inspection

Inspection may reveal distention, masses, bruising, scars from prior surgeries, or cutaneous signs of portal hypertension. Cullen's sign (a bluish umbilicus) and Grey Turner's sign (discoloration of the flank) are signs of retroperitoneal hemorrhage, although they are infrequently seen in the acute setting.

Auscultation

Auscultation is performed prior to palpation because the latter may artificially induce peristalsis. Contrary to conventional teaching, absent or diminished bowel sounds provide little useful clinical information. In one investigation, approximately half the patients with confirmed peritonitis had normal or increased bowel sounds. High-pitched or tinkling sounds can be associated with SBO, especially in the presence of abdominal distention. Low-pitched and less frequent bowel sounds are classically associated with large bowel obstruction. The auscultation of bruits might indicate the presence of an AAA in an elderly patient. In the pregnant patient, assess for fetal heart tones, which can be heard in 90% of patients by 12 weeks gestation.

Percussion

Percussion is useful for determining the size of organs and for distinguishing between distention caused by air or fluid. Tympany may be due to excessive gas in the bowel or peritoneal cavity; shifting dullness or a fluid wave suggests ascites.

Palpation

For palpation of the abdomen to be effective, it is important to first calm the patient and gain his or her cooperation. Having the patient flex the legs at the knee and hip may relax abdominal musculature, making palpation more effective. Be gentle. A rough or painful examination is not only distressing to the patient but may mislead the examining physician. Ask the patient to point with one finger to the location of greatest discomfort. Palpation should be performed systematically, beginning as far as possible from the patient's perceived location of pain. It is important to observe the patient's facial expressions for signs of pain during palpation. In older patients, careful palpation of the abdomen may also reveal a pulsatile mass suggestive of AAA.

It is rare for a serious abdominal condition to present without abdominal tenderness. By localizing tenderness to a specific abdominal region, the clinician often can narrow the diagnostic possibilities to the organs within that anatomic region. Complicating matters, however, is the fact that some patients with inflamed intra-abdominal organs do not have localizable tenderness. For example, only two-thirds of patients with appendicitis have RLQ tenderness on examination.

In addition to localizing tenderness, the patient should be assessed for signs of peritoneal irritation, the hallmark of surgical disease.

Guarding

Guarding is the reflex spasm of the abdominal wall musculature in response to palpation or underlying peritoneal irritation (Figure 10.2). Voluntary guarding can occur in response to the physician's cold hands, fear, anxiety, or being ticklish. Involuntary guarding, which has greater clinical significance, is more likely to occur with surgical illness and is not relieved by physician encouragement.

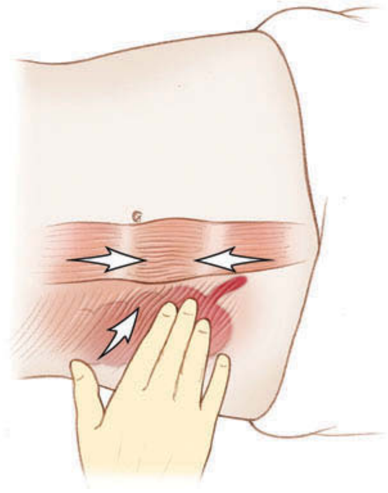
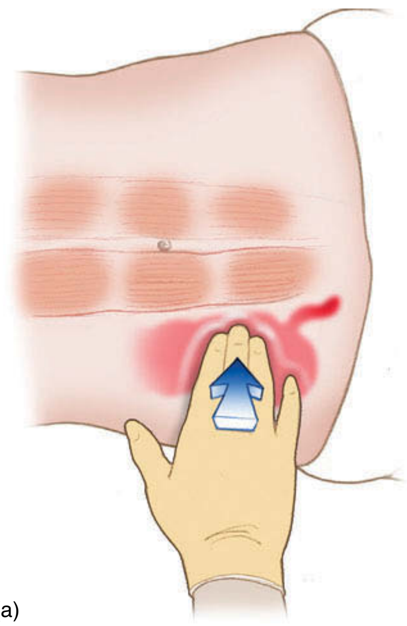


Figure 10.2
Guarding. © Chris Gralapp.

Rebound tenderness

Rebound tenderness is elicited by slow, gentle, deep palpation of an area of tenderness followed by abrupt withdrawal of the examiner's hand (Figure 10.3). Though rebound tenderness has classically been a hallmark of surgical disease, several recent studies have questioned its sensitivity, specificity and prospective utility for surgical conditions. As a result, some physicians have condemned the procedure as a painful and unnecessary holdover from the past.



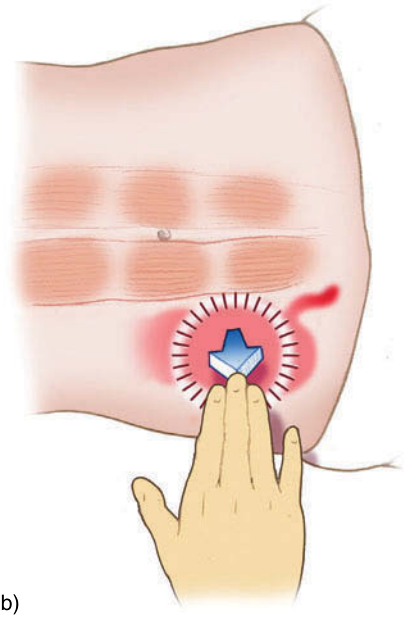


Figure 10.3
Rebound: (a) hand down; (b) hand up. © Chris Gralapp.

Alternatives to classic rebound testing include the *cough test*, where the examiner has the patient cough and looks for evidence of post-tussive abdominal pain, such as grimacing, flinching or grabbing the belly, and the *heel drop sign*, where the patient experiences pain on dropping the heels to the ground after standing on his or her toes. In children, this may be tested by having them jump up and down.

Special signs or techniques

A positive *Murphy's sign* is elicited when a patient abruptly ends deep inspiration during palpation of the RUQ. Murphy's sign is very sensitive for acute cholecystitis and biliary colic. The *psoas sign* (Figure 10.4) is performed by having the patient flex the thigh against resistance. The *obturator sign* (Figure 10.5) is performed by having the patient internally and externally rotate their flexed hip. Pain elicited by either the psoas or obturator maneuvers suggests irritation of the respective muscles by an inflammatory process such as acute appendicitis, a ruptured appendix or pelvic inflammatory disease (PID).

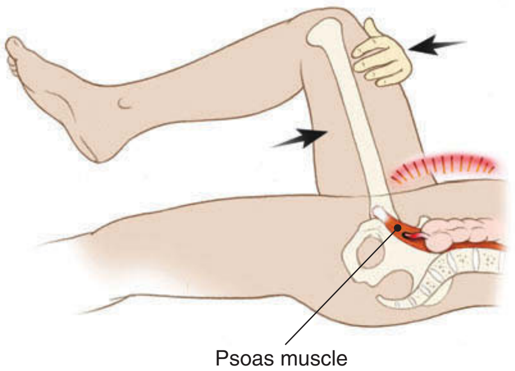


Figure 10.4
Psoas sign. © Chris Gralapp.

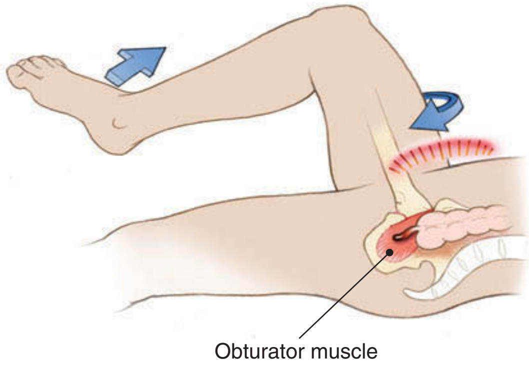


Figure 10.5
Obturator sign. © Chris Gralapp.

A positive *Rovsing's sign* is pain in the RLQ precipitated by palpation of the left lower quadrant (LLQ). This is also suggestive of appendicitis. *Carnett's sign* is increased tenderness to palpation when the abdominal muscles are contracted, as when the patient lifts his or her head or legs off the bed, and may be useful to distinguish abdominal wall from visceral pain.

Pelvic

A pelvic examination is recommended in any woman of childbearing age with abdominal pain. The pelvic examination may differentiate a gynecologic cause from other causes of pain. Cervical appearance, cervical motion tenderness (CMT), adnexal tenderness or masses, uterine size, and the presence or absence of discharge, pus or blood should be noted. Although women with appendicitis or PID may have CMT or adnexal tenderness, the presence of pus at the cervical os suggests PID. A woman with severe PID may also experience RUQ tenderness due to perihepatic inflammation (Fitz–Hugh–Curtis syndrome).

Genital

Just as every woman of childbearing age needs a pelvic examination, every male with abdominal pain should have a genital examination. The groin should be inspected and palpated for hernias, which may be the cause of an acute bowel obstruction. The external genitalia and scrotum should also carefully be evaluated for any tenderness, masses, or abnormalities.

Rectal

Recent literature has questioned the utility of the rectal examination in the diagnosis of appendicitis, as it is neither sensitive nor specific for the disease. However, the rectal examination still remains a necessary component of the evaluation of patients with abdominal pain. The diagnosis of prostate or perirectal disease, stool impactions, rectal foreign bodies and gastrointestinal (GI) bleeding all depend on the digital rectal examination. Occult blood in the right clinical setting should raise suspicion for intestinal ischemia.

Back

Gently percussing the costovertebral angles (CVA) of the back with a fist will elicit pain in patients with pyelonephritis or obstructive uropathy.

Skin

A thorough skin examination can provide important diagnostic clues. Pale, cool and moist skin suggests shock or dehydration. Jaundice may be found with biliary or hepatic disease. Petechiae and spider hemangiomas may be seen with liver disease.

Head to toe

Abdominal pain may be elicited by extra-abdominal causes, such as pharyngitis, pneumonia and MI. These conditions can be missed without a comprehensive physical examination.

Differential diagnosis

Table 10.3 lists conditions causing abdominal pain by diagnosis.

Table 10.3 Differential diagnosis of abdominal pain

Diagnosis	Symptoms	Signs	Work-up
Appendicitis	Classically vague periumbilical or epigastric pain that migrates to the RLQ Anorexia, nausea, vomiting Diarrhea Low-grade fever	Abdominal tenderness Fever (mean temperature 38°C) Voluntary or involuntary guarding Rebound tenderness Rovsing, psoas, or obturator signs CMT	Clinical diagnosis Abdominal CT Ultrasound (preferred in children and pregnant patients)
Biliary colic, cholecystitis, cholangitis	Acute crampy, colicky RUQ or epigastric pain May radiate to the subscapular area Nausea, vomiting Fever/chills may be present with cholecystitis and cholangitis	RUQ tenderness Murphy's sign Fever with cholecystitis, cholangitis	Ultrasound (preferred ED study) Radionuclide scan Liver function tests Amylase, lipase
Bowel obstruction	Crampy diffuse abdominal pain Nausea, vomiting No flatus or stool passage Bloating History of previous surgery or bowel obstruction	Abdominal distention Abdominal tenderness Fever Abnormal bowel sounds Peritoneal signs may indicate strangulation	Abdominal plain films Abdominal CT
Diverticulitis	LLQ abdominal pain Nausea, vomiting Fever Change in stool pattern (frequency or consistency) Constipation Diarrhea Rectal bleeding	LLQ tenderness, guarding, rebound Fever Heme-positive stools If perforation, potential for tachycardia, high fever, sepsis	Clinical diagnosis Abdominal CT Ultrasound Barium contrast enema
Ectopic pregnancy	Abdominal or pelvic pain Vaginal bleeding Amenorrhea Nausea, vomiting Dizziness May complain of shoulder pain (referred)	Abdominal or pelvic tenderness Adnexal tenderness Adnexal mass	Urine or serum pregnancy test Quantitative β-hCG Endovaginal ultrasound Rh type Hematocrit

(*continued*)

Table 10.3 Differential diagnosis of abdominal pain (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Gastroenteritis	Intermittent, crampy abdominal pain Poorly localized pain Diarrhea Nausea, vomiting	Nonspecific abdominal examination Absence of peritoneal signs Fever	Testing usually not necessary for uncomplicated gastroenteritis
Intussusception	Episodic colicky abdominal pain Nausea, vomiting Bloody stool Diarrhea Poor feeding Episodes of crying and drawing legs up	Palpable abdominal mass Abdominal tenderness Occult blood in stool *Currant jelly* (mucoid, bloody) stool Dehydration and lethargy between episodes	Abdominal plain films Barium or air contrast enema – gold standard, sometimes therapeutic Ultrasound Abdominal CT
Mesenteric ischemia	Gradual to acute onset Poorly localized, unrelenting abdominal pain Nausea, vomiting, diarrhea	Classically, pain "out of proportion" to examination Physical examination varies depending on the duration of ischemia May develop hypovolemia and sepsis	Serum lactate Abdominal plain films: may show *pneumatosis intestinalis*, portal vein gas or thumbprinting Abdominal CT Gadolinium-enhanced MRA Angiography
Ovarian torsion	Abrupt onset Severe unilateral abdominal or pelvic pain Nausea, vomiting	Unilateral abdominal or pelvic tenderness Tender adnexal mass	Transvaginal ultrasound with color Doppler Exclude pregnancy
Pancreatitis	Severe, dull epigastric or LUQ pain Radiation to back Vomiting	Abdominal tenderness Abdominal distention Volume depletion	Amylase Lipase Abdominal CT (with contrast)
Pelvic inflammatory disease	Lower abdominal pain – dull, constant or poorly localized Vaginal discharge Abnormal vaginal bleeding Urinary symptoms Dyspareunia	Lower abdominal tenderness Adnexal mass or tenderness CMT Mucopurulent endocervical or vaginal discharge Fever	Cultures for GC, chlamydia Pregnancy test Pelvic ultrasound to exclude tubo-ovarian abscess Consider syphilis, HIV testing
Perforated peptic ulcer	Sudden severe abdominal pain May radiate to back with posterior ulcers Nausea, vomiting Older patients may have minimal pain	Diffuse abdominal pain Acute peritonitis Rigid abdomen Volume depletion Hypotension, tachycardia, fever	Abdominal plain films: may show free air Abdominal CT
Ruptured or leaking abdominal aortic aneurysm	Severe abdominal pain Flank or back pain Radiation to groin, thigh Syncope	Pulsatile abdominal mass Diffuse abdominal tenderness Abdominal bruit, decreased pulses Hypotension Hematuria	Straight to OR Abdominal plain films ED ultrasound Abdominal CT
Testicular torsion	Sudden onset severe pain May be felt in the lower abdomen, scrotum or inguinal area Nausea, vomiting Previous episodes resolving spontaneously (41%)	Swollen, tender, firm hemiscrotum High-riding testis with transverse lie Loss of cremasteric reflex	Straight to OR Color Doppler imaging Radionuclide technetium scan
Ureteral colic	Abrupt onset of severe pain in the flank Radiates to the groin Nausea, vomiting Writhing in pain	Cannot find a comfortable position CVA percussion tenderness Benign abdominal examination Fever suggests infection Hematuria	Urinalysis may show hematuria Unenhanced abdominal CT IVP Ultrasound + KUB
Volvulus	Sudden severe colicky abdominal pain Abdominal distention May have had recurrent episodes Nausea, vomiting Constipation	Diffuse abdominal tenderness Abdominal distention Tympany Palpable mass with cecal volvulus Peritoneal signs, fever, shock with bowel infarction	Abdominal plain films: extremely distended colon Barium enema Sigmoidoscopy

CMT: cervical motion tenderness; CT: computed tomography; CVA: costovertebral angle; ED: emergency department; GC: gonococcus; hCG: human chorionic gonadotropin; HIV: human immunodeficiency virus; IVP: intravenous pyelogram; KUB: kidney, ureter and bladder X-ray; LLQ: left lower quadrant; LUQ: left upper quadrant; MRA: magnetic resonance angiography; OR: operating room; PID: pelvic inflammatory disease; RLQ: right lower quadrant; RUQ: right upper quadrant.

Diagnostic testing

Laboratory studies

Complete blood count

A complete blood count (CBC) is frequently ordered in patients with abdominal pain. Despite the association of an elevated white blood cell (WBC) count with many infectious and inflammatory processes, numerous studies have demonstrated that many patients with surgically proven appendicitis have initially normal WBC counts. Even serial WBC counts have failed to discriminate between surgical and non-surgical illness. In the patient with abdominal pain, an elevated WBC does not necessarily imply serious disease, detecting only 53% of patients with severe abdominal pathology in one study. In fact, elevations of the WBC count may lead to additional tests and increased costs without providing additional information. The WBC should never be used to make the sole diagnosis of abdominal pathology, nor should it be used in isolation to exclude reasonable diagnostic possibilities. The bottom line is that decision making in cases of abdominal pain rests primarily on a careful history and thorough physical examination, not the WBC count.

Urinalysis

The urinalysis is a rapid, cost-effective adjunctive laboratory test that needs to be interpreted with caution in patients with abdominal pain. Findings suggestive of UTI include pyuria, positive leukocyte esterase, positive nitrites and the presence of bacteria. However, up to 30% of patients with appendicitis may present with blood, leukocytes, or even bacteria in their urine. A mild degree of pyuria may be present in elderly patients at baseline. Be wary of ascribing abdominal pain to a UTI when the clinical picture does not fit. Red blood cells (RBCs) in the urine are consistent with infection, trauma, tumors and stones. Acute flank pain and hematuria suggests renal colic but may also represent a leaking or ruptured AAA.

Pregnancy test

All female patients of childbearing age with abdominal pain should have a pregnancy test. A positive pregnancy test expands the differential diagnosis (e.g., ectopic pregnancy), influences the choice of medications or adjunctive studies, and may impact disposition. Do not omit pregnancy testing in patients who report sexual abstinence, tubal ligation, or contraceptive use.

Amylase/lipase

Serum amylase, though commonly ordered, may be normal in as many as one-third of patients with pancreatitis. The serum amylase may also be elevated in other conditions, including peptic ulcer or liver disease, SBO, common duct stones, bowel infarction, ectopic pregnancy, ethanol intoxication and diabetic ketoacidosis (DKA). Serum lipase has a higher sensitivity and specificity for pancreatitis than total amylase, and is therefore the most useful test in a patient with suspected pancreatitis.

Other laboratory studies

Liver function tests may be elevated in patients with biliary or hepatic disease. Serum electrolytes may be abnormal in patients with significant vomiting or diarrhea, symptoms for more than 24 hours duration, diuretic use, or a history of kidney or liver disease. Serum glucose should be assessed in patients with diabetes or suspected diabetic ketoacidosis (i.e., abdominal pain, tachypnea and vomiting). Serum phosphate and serum lactate may be elevated in cases of bowel ischemia.

Electrocardiogram

Electrocardiograms (ECGs) should be considered for all patients with unexplained epigastric or abdominal pain. They are particularly essential in the evaluation of elderly patients with vague, poorly localized abdominal complaints. An acute coronary syndrome (ACS) or inferior MI can present with epigastric pain, diaphoresis and vomiting. Though a normal ECG in the setting of abdominal pain does not exclude MI, it makes it less likely.

Radiologic studies

Plain films

Abdominal plain films are markedly overutilized, difficult to interpret (even in experienced hands), and rarely provide useful clinical information. Plain films are unlikely to be helpful in patients with nonspecific abdominal pain, suspected appendicitis and UTIs. In fact, they may cloud the diagnosis, leading to delays in management. Plain films of the abdomen should be restricted to patients with suspected bowel obstruction, perforated viscus or foreign bodies. Even for these presentations, computed tomography (CT) is more sensitive and specific. When evaluating plain abdominal radiographs, look for abnormalities such as dilated loops of large or small bowel, air-fluid levels, abnormal calcifications (of the abdominal aorta, urinary tract, gallbladder [gallstones], or appendix [appendicolith]), or air in abnormal locations (i.e., free air under the diaphragm [Figure 10.6], in the portal vein, bowel wall, or between loops of bowel).

Ultrasound

Ultrasound has emerged as an extremely useful diagnostic modality in patients with abdominal pain. Advantages of ultrasound include lack of ionizing radiation, low cost and widespread availability. It is the preferred imaging approach for evaluating patients with RUQ pain. In patients with acute cholecystitis, ultrasound may detect gallstones, gallbladder wall thickening, pericholecystic fluid, or a sonographic Murphy's sign.

Additionally, ultrasound is useful in imaging the pelvic organs; the transvaginal approach is preferred and

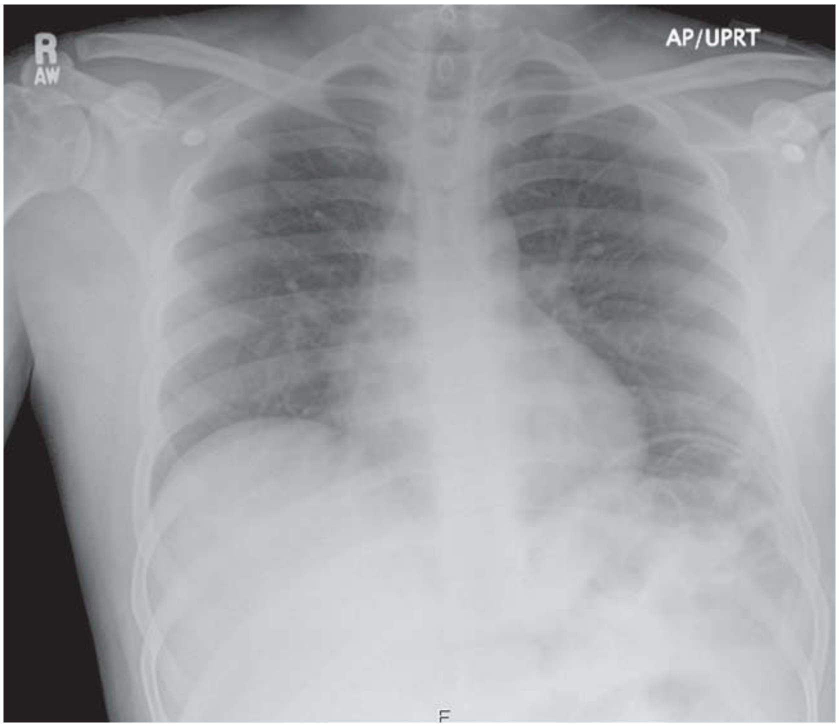


Figure 10.6
Pneumoperitoneum. AP upright chest X-ray reveals free air beneath the left hemidiaphragm consistent with pneumoperitoneum.

superior to the transabdominal approach for the diagnosis of ectopic pregnancy.

When radiation is a concern, ultrasound is the procedure of choice for identifying acute appendicitis, especially in children, women of reproductive age and pregnant patients. The primary sonographic criterion of appendicitis is demonstration of a swollen, noncompressible appendix >7 mm in diameter with a target configuration (Figure 10.7).

Goal-directed bedside ultrasonography can be performed by ED physicians to:

1. Confirm an intrauterine pregnancy, dramatically lowering the risk of ectopic pregnancy
2. Screen for the presence of an AAA (Figure 10.8)
3. Screen for the presence of free intraperitoneal fluid in patients with suspected ectopic pregnancy or abdominal trauma (see Appendix E)

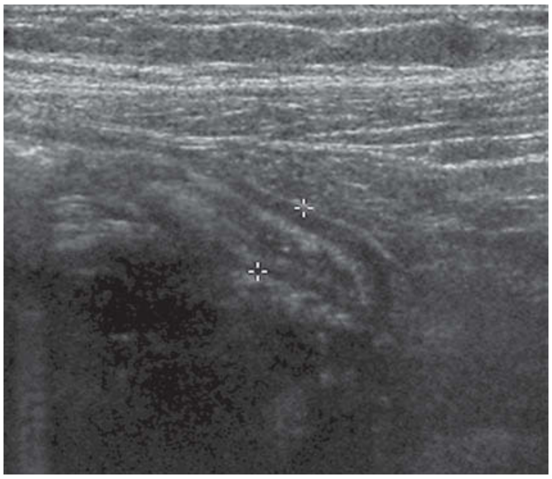


Figure 10.7
Appendicitis on ultrasound. Gray-scale longitudinal ultrasound demonstrates enlarged non-compressible appendix (cursors) >7 mm, consistent with acute appendicitis. Courtesy: Gus M. Garmel, MD.

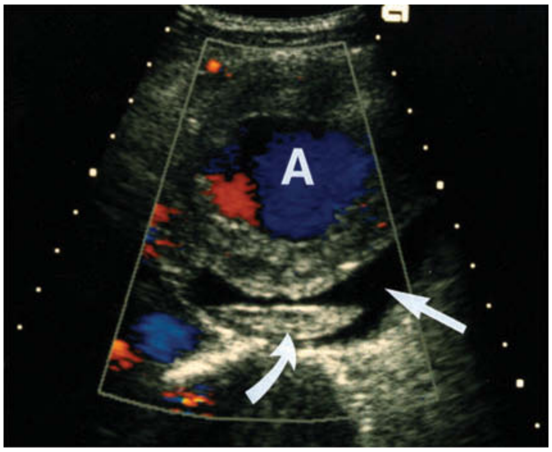


Figure 10.8
Ruptured abdominal aortic aneurysm (AAA) on transverse color Doppler sonogram. Note color flow within aneurysm (A) and retroperitoneal clot (curved arrow) and hemorrhage posterior to AAA (straight arrow). Courtesy: R. Brooke Jeffrey, MD.

Ultrasound may be difficult to perform in obese patients and those in severe pain. As ultrasound requires considerable skill, findings are operator-dependent and interpretation errors can occur. A negative ultrasound does not exclude the diagnosis of either appendicitis or ectopic pregnancy.

Abdominal computed tomography

Abdominal CT has become the modality of choice in patients with undifferentiated abdominal pain who require imaging; it allows for a panorama-like visualization of the structures of the peritoneal and retroperitoneal space, uninhibited by the presence of bowel gas or fat. Due to its exceptional accuracy, CT is often the primary imaging modality in patients with suspected appendicitis. CT findings of appendicitis (Figure 10.9) include a swollen, fluid-filled appendix, often with a calcified appendicolith or inflammatory changes in the periappendiceal mesenteric fat. After perforation, a phlegmon or abscess may be visible. CT is also useful for determining the diagnosis (and in many cases, the clinical severity) of conditions such as renal colic, bowel obstruction, bowel perforation, bowel ischemia, diverticulitis, pancreatitis, intra-abdominal abscess, AAA and solid organ injury. IV contrast is useful for identifying mesenteric ischemia, whereas oral contrast may assist with the visualization of intra-abdominal abscesses. The major drawbacks of CT are cost, contrast nephrotoxicity, radiation and availability.

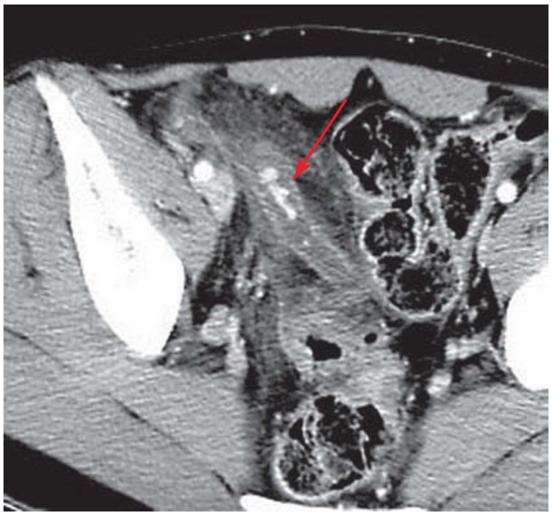


Figure 10.9
Acute appendicitis on contrast enhanced CT. Note enlarged appendix with multiple appendicoliths (arrow). Periappendiceal fat stranding is apparent. Courtesy: R. Brooke Jeffrey, MD.

General treatment principles

As with all ED patients, treatment begins with the ABCs (airway, breathing, circulation). The main goals of treatment are physiologic stabilization, symptom relief and preparation for surgical intervention when warranted.

Volume repletion

Not all patients with abdominal pain need intravenous (IV) access or IV fluids. However, many patients have some degree of volume contraction resulting from poor intake, vomiting and diarrhea, or third-spacing. Other patients may have volume loss secondary to internal bleeding (e.g., ectopic pregnancy). Crystalloids are the initial fluids of choice in both children and adults. The rate of repletion is determined by the patient's degree of hypovolemia, cardiovascular status, and response to initial therapy. Under certain circumstances, such as life-threatening hemodynamic collapse, blood products may be the initial resuscitation fluid.

Pain relief

Despite the long-held opinion that narcotic analgesia masks peritoneal signs of an acute abdomen, there is no clear evidence supporting this notion. In fact, recent studies have revealed that administration of moderate doses of analgesia and the ensuing pain relief do not cloud diagnostic findings; instead, this approach actually may aid in the diagnosis of surgical disease. In the acute setting, pain relief is typically achieved with IV titration of opioid analgesics such as morphine sulfate or fentanyl.

When combined with narcotic agents, IV ketorolac provides pain relief for patients with biliary and renal colic. Ketorolac is not recommended for treatment of undifferentiated abdominal pain as it may mask abdominal findings and increase bleeding times. Patients with epigastric discomfort may gain relief from a GI cocktail (varied combinations of an antacid, viscous lidocaine and/or Donnatal). Though the GI cocktail may be therapeutic, it is not diagnostic, as even pain from an acute MI may be relieved by this therapy.

Antibiotics

Antibiotics are indicated in patients with abdominal sepsis, suspected perforation, or the presence of peritonitis (local or diffuse). Abdominal infections are often polymicrobial and necessitate coverage for enteric Gram-negatives, Gram-positives and anaerobic bacteria. The specific regimen must take into account the patient's presentation, comorbid conditions, and local bacterial drug sensitivities and drug-resistance patterns.

Other

Control of emesis can be achieved by a number of agents. Patients in whom surgery is anticipated should be kept from eating or drinking (NPO). A nasogastric (NG) tube may be of benefit in patients with confirmed bowel obstruction or vomiting refractory to antiemetic administration.

Special patients

Elderly

Several factors make the diagnosis and management of abdominal pain in elderly patients challenging. Surgical causes of abdominal pain increase in incidence with advancing age, whereas nonspecific abdominal pain becomes less common. Typically, surgical illness in elderly patients is more rapidly life-threatening than in younger patients. Older patients are at much greater risk for vascular catastrophes such as ruptured AAA, mesenteric ischemia and MI. Elderly patients are more likely to present without the classic or expected historical or physical examination findings associated with a common disease. Because of atypical presentations and comorbidities, patient mortality and rates of misdiagnosis increase exponentially each decade after age 50. For these reasons, CT has increased diagnostic utility in elderly patients and is more likely to impact management and disposition. Of patients more than 65 years of age presenting to the ED with abdominal pain, nearly half are admitted and about one-third will require surgery.

Pediatric

The diagnosis of abdominal pain in children presents its own unique challenges. Histories must often be obtained from parents or caregivers, as children are not always able to articulate their complaint or describe their symptoms. Consequently, younger children tend to present with late symptoms of disease and have a higher incidence of perforated appendicitis compared with adults. The usual etiologies of abdominal pain in children vary from those in adults (Table 10.4). Gastroenteritis, nonspecific abdominal pain and appendicitis are more common in children, whereas biliary disease, pancreatitis and vascular disease are relatively rare. Illnesses relatively unique to children include intussusception, volvulus, pyloric stenosis and Hirschsprung's disease. Any child presenting with bilious vomiting should be presumed to have a bowel obstruction.

Pregnancy

The evaluation of abdominal pain is often more challenging in the pregnant patient. By 12 weeks gestation, the enlarging uterus extends into the abdomen, thereby impeding physical examination, altering the location of pelvic and abdominal organs, and masking peritoneal signs. Although nausea and vomiting are common early in pregnancy, their association with abdominal pain, fever, diarrhea, or peritoneal signs is abnormal. The differential diagnosis of abdominal pain in pregnant patients includes pregnancy-specific conditions (e.g., placental abruption, uterine rupture), gynecologic causes (e.g., ovarian torsion, fibroid degeneration) and abdominal conditions (e.g., appendicitis, biliary disease, bowel obstruction). Delays in diagnosis and treatment can increase maternal and fetal/newborn morbidity and mortality; therefore, rapid evaluation and early obstetric and surgical consultation are paramount. Concern about the possible fetal effects of ionizing radiation should not impede medically indicated diagnostic studies such as CT.

Immune compromised

In addition to ordinary afflictions such as appendicitis, patients with human immunodeficiency virus (HIV) presenting with abdominal pain may also have:

1. Enterocolitis with profuse diarrhea and dehydration;
2. Large bowel perforation associated with cytomegalovirus (CMV);
3. Bowel obstruction from Kaposi's sarcoma, lymphoma or atypical mycobacteria;

Table 10.4 Causes of abdominal pain by age of onset

Birth to 1 year	2–5 years	6–11 years	12–18 years
Constipation	Appendicitis	Appendicitis	Appendicitis
Gastroenteritis	Constipation	Constipation	Constipation
Hirschsprung's disease	Gastroenteritis	Functional pain	Dysmenorrhea
Incarcerated hernia	Henoch–Schönlein purpura	Gastroenteritis	Ectopic pregnancy
Infantile colic	Mesenteric lymphadenitis	Henoch–Schönlein purpura	Gastroenteritis
Intussusception	Intussusception	Mesenteric lymphadenitis	Mittelschmerz
UTI	Pharyngitis	Pharyngitis	Ovarian torsion
Volvulus	Sickle cell crisis	Pneumonia	PID
	Trauma	Sickle cell crisis	Testicular torsion
	UTI	Trauma	Threatened miscarriage
	Volvulus	UTI	

PID: pelvic inflammatory disease; UTI: urinary tract infection.

Adapted from Leung AKC, Sigalet DL. Acute abdominal pain in children. *Am Fam Physician* 2000;67(11):2321–6.

4. Biliary tract disease from cryptosporidium or CMV;
5. Drug-induced pancreatitis.

The use of antibiotics, steroids or other immunosuppressants may mask abdominal examination findings usually associated with infection, so consideration should be given to any abdominal pain complaint, no matter how slight. Steroid use can lead to demargination of leukocytes, making interpretation of the WBC count more difficult. Steroids also promote peptic ulcer disease, leading to an increased incidence of perforated viscus.

Disposition

Surgical consultation

Patients with an acute abdomen or confirmed surgical illness require urgent surgical consultation. Life-threatening diagnoses such as ruptured AAA or ectopic pregnancy require emergent consultation and expedited treatment. The most common causes of abdominal pain requiring surgical consultation are appendicitis, intestinal obstruction, perforated ulcer and acute cholecystitis. These patients should be kept well-hydrated and NPO. Early diagnosis and surgery for appendicitis prevents perforation and the associated acute (abscess formation, sepsis) and late (scar formation with bowel obstruction/infertility) complications.

Serial evaluation

Observation with serial examinations allows the emergency physician an extended evaluation of a patient with an early or atypical presentation of appendicitis or another acute abdominal process. These patients are kept in the ED or admitted to the hospital for serial abdominal examinations. Serial evaluation, preferably by the same physician, allows a patient's clinical picture to evolve or resolve over a period of time. Studies have shown that observation and repeated examinations of patients with suspected appendicitis improve diagnostic accuracy without increasing rates of perforation.

Discharge

After a thorough work-up in the ED or serial observation, patients without evidence of concerning medical or surgical illness may be discharged. Despite a patient's expectation of a firm diagnosis, it is perfectly acceptable to diagnose the patient with nonspecific or undifferentiated abdominal pain. In fact, the majority of patients are discharged from the ED with this diagnosis. Avoid forcing a diagnosis, such as acute gastroenteritis, on a patient. True gastroenteritis requires the presence of both vomiting and diarrhea.

When discharging a patient with undiagnosed abdominal pain, it is important to arrange for a repeat evaluation within 12–24 hours (either in the ED or with an outpatient clinic) and emphasize the need to return to the ED if symptoms worsen. Typically, patients are placed on a clear liquid diet and narcotic analgesics are avoided. For patients returning to the ED with worsening symptoms, the additional opportunity to establish the diagnosis should be welcomed. Typically, these patients are more likely to have appendicitis or bowel obstruction. Patients in whom reliable follow-up cannot be arranged or assured may require admission.

Pearls, pitfalls and myths

- Do not restrict the diagnosis solely by the location of the pain.
- Consider appendicitis in all patients with abdominal pain and an appendix, especially in patients with the presumed diagnosis of gastroenteritis, PID or UTI.
- Do not use the presence or absence of fever to distinguish between surgical and medical causes of abdominal pain.
- The WBC count is of little clinical value in the patient with possible appendicitis.
- Any woman with childbearing potential and abdominal pain has an ectopic pregnancy until her pregnancy test comes back negative.
- Pain medications reduce pain and suffering without compromising diagnostic accuracy.
- An elderly patient with abdominal pain has a high likelihood of surgical disease.
- Obtain an ECG in all older patients and those with cardiac risk factors presenting with abdominal pain.
- A patient with appendicitis by history and physical examination does not need a CT scan to confirm the diagnosis; they need an operation.
- The use of abdominal ultrasound or CT may help evaluate patients over the age of 50 with unexplained abdominal or flank pain for the presence of AAA.

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11 Abnormal behavior

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Scope of the problem

Patients manifesting abnormal behavior are common in emergency departments (EDs). They are some of the most challenging patients emergency physicians must treat. The causes of abnormal behavior are exceedingly diverse and require physicians to maintain a high level of vigilance to determine whether an underlying medical disorder exists. According to the National Hospital Ambulatory Medical Care Survey (NHACMS) of 2006 (published in 2008), nearly 4% of the 119 million ED visits were categorized as presentations related to mental disorders (ICD-9-CM 290–319). This percentage is likely higher as not all patients were classified, and presentations related to ingestion, drug- or alcohol-related abuse, and self-inflicted harm may not have been classified as mental disorders. Furthermore, occult depression frequently exists in adolescents, and its symptoms are often unrecognized. Many patients with abnormal behavior present "for medical clearance" or must be "medically cleared" prior to psychiatric hospitalization. "Medical clearance" should include a comprehensive medical evaluation to identify any potential underlying medical problem responsible for abnormal behavior. It is important that patients presenting with behavioral problems are treated sensitively.

Anatomic essentials

The physiology of behavior represents a complex interplay of human physiology and environmental factors. Historically, changes in behavior have been classified as being of functional (psychiatric) or organic (medical) etiology. These classifications are dated, as neuropathophysiologic mechanisms of psychiatric disease have gained prominence over the past decades. Aberrations in neurotransmitter transduction have been identified in depression (serotonin), schizophrenia (dopamine) and Alzheimer's disease (acetylcholine). Functional magnetic resonance imaging (MRI) and positron emission tomography (PET) scans of the brain have demonstrated unique differences in some individuals with mental illness. Pharmacologic therapy directed at modulation of neurotransmitters has greatly advanced the treatment and prognosis of patients suffering from these conditions.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 11.1).

Table 11.1 Abnormal behavior red flags

History	Concerning diagnosis
Children, especially toddlers	Ingestion, nonaccidental (or accidental) trauma, CNS infection, malignancy
Elderly	Organic illness, delirium, dementia, drug–drug interaction or adverse drug reaction, CNS infection (esp. fungal or uncommon organisms), sepsis, traumatic injury (esp. falls), electrolyte abnormality (esp. sodium), malignancy/metastatic disease
Age < 40 years	Substance abuse, psychiatric (functional) illness
Immunocompromised	CNS infection (esp. opportunistic), electrolyte abnormality, malignancy or metastatic disease
Diabetes	Hypoglycemia or hyperglycemia, stroke
Known or witnessed seizure disorder	Postictal phase, head injury, ingestion, substance abuse, anticonvulsant toxicity, glucose-related, electrolyte abnormality
Cardiopulmonary disease	Hypoxia, acute cardiac event
Recent illness, especially with reported fevers	CNS infection
Examination finding	**Concerning diagnosis**
Fever, rash, nuchal rigidity	CNS infection, such as meningitis, encephalitis
Toxidrome	Poisoning or ingestion (anticholinergic, opioid, alcohol or other toxin)
Raccoon eyes or Battle sign, hemotympanum, retinal or subhyaloid hemorrhage, other traumatic injury	CNS insult from injury (epidural, subdural, traumatic subarachnoid hemorrhage, contusion, concussion, closed head injury)
Focal CNS findings, including papilledema	CNS mass, intracranial hypertension, stroke
Track or needle marks	Toxic ingestion, CNS infection, endocarditis
Vital sign abnormality (i.e., sepsis)	CNS hypoperfusion

CNS: central nervous system.

History

Prior to obtaining a history of presenting illness or abnormal behavior, the safety of the patient and all staff should be ensured. Patients who are altered or violent may be unable or unwilling to give an adequate history. It is important to seek additional sources of information from family members, friends, paramedics, police, or witnesses.

Is this an acute or chronic condition?

The temporal nature of these behavioral changes is a good place to begin when obtaining the history. Sudden behavioral changes in a previously healthy person more likely herald an underlying medical disorder. In contrast, dementia is characterized by progressively worsening cognitive function, typically in an older individual.

If acute, what were the events leading up to the change in behavior?

Does an antecedent history of trauma, ingestion, medication noncompliance, or new medication(s) explain the patient's symptoms? Has the patient had a precipitating stressor recently, such as difficulty with a family member or relationship, or challenges at work or school?

Does the patient have a history of psychiatric illness?

Patients with a history of psychiatric illness are more likely to have an underlying functional disorder causing their abnormal behavior. Does the patient have a history of depression, mania, schizophrenia, or anxiety? Does the patient have a psychiatrist or psychotherapist? If so, it is important to contact that individual for additional history and consultation about disposition once underlying medical illnesses have been excluded. It is advised to gain permission from the patient to do this, although in some circumstances a patient might not be able or willing to provide consent. Many patients suffer from undiagnosed depression. The mnemonic "SIG-ME-CAPS" is helpful when evaluating patients for possible depression (Table 11.2). Other screening tools exist, such as the Beck Depression Inventory (BDI), although these may not be appropriate for use by emergency physicians in the ED.

Table 11.2 SIG-ME-CAPS mnemonic for depression screening

S	Sleep disturbances
I	Interest in hobbies (decreased)
G	Guilt (feelings of worthlessness)
M	Mood (depressed)
E	Energy (decreased)
C	Concentration (decreased)
A	Appetite (usually less, may be variable)
P	Psychomotor movements
S	Suicidal ideations or thoughts

What medications does the patient take? Is there a suspected ingestion?

Medications are commonly implicated as the etiology of acute behavioral changes. When taking a history regarding medication use, the following information should be considered:

1. What are the prescribed and over-the-counter medications (including herbal remedies) taken by the patient?
2. Is a new medication causing an adverse reaction (e.g., mefloquine for malaria chemoprophylaxis may cause psychosis) or altering behavior through a drug–drug interaction?

Serotonin syndrome is a serious central nervous system (CNS) complication that can occur in patients who have taken medication that increases endogenous levels of CNS serotonin. These medications include monoamine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs) and amphetamine-based drugs. Serotonin syndrome can occur as a side effect of a regularly scheduled medication, from cross-reactivity with another prescription or over-the-counter medication, or as a result of an overdose. It can present with mild to life-threatening symptoms, and is typically defined as the triad of neuromuscular excitability (tremor, rigidity and hyperreflexia), abnormal mental status (disorientation, delirium), and autonomic instability (diaphoresis, fever, vomiting). Severe cases require critical supportive care.

3. Is there a possibility of an accidental or intentional overdose?
4. Is the patient sharing or taking someone else's medications?

Many medications are known to alter mental status or behavior (Table 11.3). The patient should be questioned about any recent dosage adjustments. Even when patients have been taking their regularly scheduled doses, worsening renal or hepatic insufficiency or dehydration may lead to supratherapeutic drug levels (e.g., digoxin toxicity in the dehydrated elderly patient with worsening renal function), resulting in alterations of behavior. With cognitive impairment or confusion, patients make inadvertently take an incorrect dose of their medications.

Table 11.3 Drugs that cause behavior changes

Anxiolytics	Lorazepam
Antibiotics	Isoniazid, rifampin, metronidazole
Anticonvulsants	Phenytoin, phenobarbital, valproate
Antidepressants	Selective serotonin reuptake inhibitors, monoamine oxidase inhibitors
Cardiovascular drugs	Digoxin, beta-blockers, methyldopa
Others	Antihistamines, cimetidine, corticosteroids, disulfiram, mefloquine, chemotherapy agents, soporific agents

Is the patient suicidal? Is there a history of suicide attempts or gestures? Is the patient homicidal? Can the patient care for him/herself?

These questions are essential in identifying patients who require involuntary psychiatric admission for evaluation and treatment. Immediate steps should be taken to keep these patients from harming themselves or others. Inquire about red flags for suicidality. These include guns or weapons at home, pills or access to them, previous suicide attempts or recent stresses (job, financial, relationship, health). Suicidal patients are frequently encountered in the ED. In a national review, Doshi et al. demonstrated that patients with self-injury and attempted suicide compromised 0.4% of all ED visits. The authors noted that self-poisoning was the most common method, and that adolescents and young adults were the most frequently effected. Suicide is responsible for more than 30,000 deaths each year in the United States. Males are reported to be four times more likely to die from suicide than females, although females attempt suicide more often. Many people who attempt suicide don't seek medical or psychiatric care.

In addition, physicians have a *duty to warn* any individual or party who may be endangered as the result of a homicidal ideation. When assessing whether or not a patient is *gravely disabled*, consider whether or not the patient is able to shower or bathe, adequately feed themselves, ambulate safely, manage finances and make reasonable judgments. The conditions under which a person can be placed on an emergency psychiatric hold are a matter of state law and will be discussed later in this chapter.

Is there a history of substance or physical abuse?

Abnormal behavior is often the result of acute recreational drug or alcohol ingestion, or a withdrawal syndrome. Research reports that drugs and alcohol are responsible for as high as 60% of the abnormal behavior seen in EDs. There is a higher incidence of substance abuse in patients who suffer from psychiatric illness; similarly, patients with a history of substance abuse are more likely to have underlying psychiatric conditions. For patients who are depressed, substance abuse is an independent risk factor for suicide. It is important to ask patients, especially those with abnormal behavior, whether or not they are victims of physical, emotional or sexual abuse.

Associated symptoms

- *Head, eye, ear, nose and throat (HEENT)*: headache, diplopia, vision loss, pain
- *Chest*: pain, cough, shortness of breath
- *Gastrointestinal (GI):* pain, nausea, vomiting, diarrhea, incontinence, constipation
- *Genitourinary (GU)*: pregnancy, bleeding, pain, discharge, incontinence, dysuria
- *Skin*: rash, lesions, excessive moisture or dryness, pruritus
- *Neurologic*: weakness, numbness, difficulty walking, vertigo, tinnitus
- *Psychiatric*: mood, hallucinations (visual or auditory), anxiety, formication, depression, suicidal, homicidal

Physical examination

The physical examination represents a key aspect in identifying underlying medical pathology in patients with behavioral changes. In addition, it may provide clues to specific underlying psychiatric diagnoses. Physicians and psychiatrists infrequently perform complete physical examinations in patients with abnormal behavior. The medicolegal literature includes cases of fatal medical disorders inappropriately diagnosed as psychiatric illness. It is important for emergency physicians to be meticulous in gathering data from history and physical examination, including a complete set of vital signs and appropriately selected laboratory and/or imaging studies to avoid missing medical conditions responsible for abnormal behavior.

General appearance

The general appearance of the patient is a key feature of the physical examination. Is the patient alert? Is the patient violent or are there signs of impending violence, such as increased motor activity, pressured speech, threatening posture and gestures? Is the patient clean, well groomed and appropriately attired? Are there any unusual odors or coloring that might provide clues to medical diseases, including metabolic conditions or toxidromes?

Vital signs

Vital signs should be obtained as soon as safety allows. Any vital sign abnormality warrants a thorough evaluation. Many patients with underlying psychiatric illness who are evaluated in the ED do not have a complete set of vital signs documented. In particular, the temperature is often omitted. An incomplete set of vital signs is a common pitfall. Alterations in vital signs may be the only clue to an underlying medical disorder, such as bacterial meningitis, sepsis, pneumonia or other infection, or toxidrome.

Head

The head should be inspected for any evidence of trauma, including signs of a basilar skull fracture (Battle sign or raccoon eyes), soft tissue swelling or lacerations. Palpate the scalp for occult hematomas. Closely examine the head for the presence of surgical scars or shunt hardware.

Eyes

A careful ocular examination is warranted, as the eyes may provide the only clue to a patient with an underlying medical condition. Miosis (pinpoint pupils) can be caused by narcotics, cholinergic toxicity, brainstem lesions or clonidine use. Mydriasis (dilated pupils) is associated with sympathomimetics, anticholinergics, withdrawal states and post-anoxic injury. If papilledema is present, immediate computed tomography (CT) of the head should be performed, as this may signify increased intracranial pressure. Anisocoria (size difference between pupils) may indicate a space-occupying central lesion, although this may be a normal finding, or due to pharmacologic paralysis. Attention should also be directed to extraocular movements (EOMs), as alterations can be seen with Wernicke's encephalopathy or brainstem lesions. Nystagmus is an important finding associated with drug intoxication, but may be present in brainstem and posterior fossa lesions.

Neck

Assess for evidence of trauma, surgical scars, masses, nuchal rigidity, bruits, or thyromegaly.

Cardiopulmonary

Careful inspection and auscultation for evidence of pneumonia, murmurs, extra heart sounds, trauma or surgical scars is important, as such abnormalities might explain the patient's abnormal behavior.

Abdomen

Distension or pain with palpation may suggest possible underlying surgical pathology. Hepatomegaly and ascites in the setting of abnormal behavior may suggest hepatic encephalopathy. A rectal examination should be considered to assess for signs of trauma, foreign body, drugs, melena or hematochezia.

Genitourinary

In women, a careful pelvic examination should be performed to look for evidence of foreign body, sexual assault, trauma or infection. In older men, particularly those with diabetes, Fournier's disease (gangrene of the scrotum and/or perineum) or prostatitis may cause abnormal behavior due to overwhelming infection.

Skin

Assess skin turgor for signs of dehydration and malnutrition. Excessive moisture can be associated with drug ingestion or certain toxidromes. Inspect for the presence of petechiae, purpura, or ecchymosis. Is there evidence of intravenous (IV) drug usage (track marks, "skin popping," abscesses or scars from previous drainage procedures), burns, or excoriations? Are lesions suspicious for Kaposi's sarcoma present, as these might signify encephalopathy from underlying acquired immune deficiency syndrome (AIDS). Severe or unusual fungal or other rashes suggest an immune-compromised host.

Neurologic

The neurologic examination is essential in differentiating medical from psychiatric illness. A retrospective review of patients admitted to psychiatric hospitals demonstrated the neurologic examination to be the most frequently undocumented portion of the physical examination. The examination should be performed in a systematic fashion, with assessment of orientation, memory, cranial nerves, motor, sensory, reflexes and cerebellar function included and documented.

Psychiatric

Is the patient suicidal or homicidal? Determine the patient's orientation (day, date, time and location), mood (emotional state), affect (flat vs. elevated), thought content (delusions), cognitive function (Mini-Mental State Examination), speech quality (rapid, clear), and presence of hallucinations (auditory vs. visual) (Table 11.4).

Table 11.4 Mini-Mental State Examination sample items

Orientation to time "What is the date?"
Registration "Listen carefully. I am going to say three words. Please say them back after I stop. Ready? Here they are… APPLE (pause), PENNY (pause), TABLE (pause). Now repeat those words back to me." [Repeat up to 5 times, but score only the first trial.]
Naming "What is this?" [Point to a pencil or pen.]
Reading "Please read this and do what it says." [Show examinee the words on the stimulus form.]
CLOSE YOUR EYES

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A helpful mnemonic in distinguishing primary medical from psychiatric disorders is "OMI-HAT" (Orientation, Memory, Intellect, Hallucinations, Affect, Thinking). A medical (organic) etiology is more often associated with alterations in OMI, whereas psychiatric (functional) disorders are more associated with abnormalities in HAT.

The confusion assessment method (CAM) is the most useful tool for diagnosing *delirium*, an acute disturbance of consciousness with associated impaired cognition not accounted for by preexisting dementia. The CAM identifies the criteria necessary for diagnosis; other criteria that are not necessary for diagnosis (although common in delirium) include abnormal psychomotor activity, sleep–wake cycle disturbances, hallucinations, delusions and tremor. The CAM can detect delirium even in the presence of dementia (Table 11.5).

Table 11.5 Confusion assessment method (CAM)

Feature 1	Acute onset and fluctuating course
Feature 2	Inattention
Feature 3	Disorganized thinking
Feature 4	Altered level of consciousness

The diagnosis of delirium requires both features 1 and 2 to be present with either feature 3 or 4.

From Inouye SK, van Dyck CH, Alessi CA, et al. Clarifying confusion. *Ann Int Med* 1990;113:941–948.

Differential diagnosis

The differential diagnosis of abnormal behavior is broad. It includes medical and traumatic illness, effects of medications or intoxicants, and psychiatric disorders. Alterations in behavior can run the gamut from minor changes in speech to florid psychosis. Historically, several features help differentiate primary medical (organic) from psychologic (psychiatric or functional) disease (Table 11.6).

Table 11.6 Primary medical vs. psychological etiology for abnormal behavior

Primary medical (organic)	Psychological (functional)
Age < 12 or > 40 years	Age 12–40 years
Sudden onset (hours to days)	Gradual onset (weeks to months)
Fluctuating course	Continuous course
Disorientation	Scattered thoughts
Decreased consciousness	Awake and alert
Visual hallucinations	Auditory hallucinations
No psychiatric history	Previous psychiatric history
Emotionally labile	Flat affect
Abnormal vital signs/physical examination	Normal vital signs/physical examination

There are many organic causes of behavioral changes. Frequently, these are manifestations of an underlying medical problem. The mnemonic “I WATCH DEATH” is one of several proposed for the differential diagnosis of delirium, and serves as a good reminder when evaluating a patient in the ED with acute behavioral changes (Table 11.7).

Table 11.7 Differential diagnosis of delirium: I WATCH DEATH

Cause	Etiology
Infectious	Sepsis, encephalitis, meningitis, neurosyphilis, CNS abscess
Withdrawal	Alcohol, barbiturates, sedatives
Acute metabolic	Acidosis, electrolyte abnormality, hepatic or renal failure, hypoglycemia
Trauma	Head trauma, burns
CNS disease	Hemorrhage, CVA, vasculitis, seizure, tumor
Hypoxia	COPD, respiratory failure, hypotension
Deficiencies	B_{12}, niacin, thiamine
Environmental	Hypo- or hyperthermia
Acute vascular	Hypertensive emergency, subarachnoid hemorrhage
Toxins/drugs	Medications, recreational drugs, alcohols, pesticides, industrial poisons (carbon monoxide, cyanide, solvents)
Heavy metals	Lead, mercury

CNS: central nervous system; COPD: chronic obstructive pulmonary disease; CVA: cerebrovascular accident.

Diagnostic testing

As with all patients seen in the ED, diagnostic testing should be guided by a careful history and physical examination. Patients with a prior history of psychiatric illness, normal vital signs and a normal physical examination may not require diagnostic tests in the ED. In a recent survey of emergency physicians, most felt that “routine” laboratory testing was not a necessary part of the medical screening examination of psychiatric patients. However, nearly one-third of those respondents reported that routine testing is required by their local psychiatric facilities.

Few studies have examined the yield of routine laboratory testing as part of the medical screening examination of the psychiatric patient. At a county ED, Henneman et al. prospectively studied the utility of a standardized medical evaluation in 100 alert patients 16–65 years of age presenting with first-time psychiatric symptoms without obvious signs of intoxication or suicidality. This evaluation included a complete history and physical examination (H&P), complete blood count (CBC), creatine phosphokinase (CPK), electrolyte and renal panel, prothrombin time, calcium, drug and alcohol screening, head CT and lumbar puncture if febrile. They reported that 63 patients had an underlying medical condition, with abnormal findings of H&P in 33 patients, CBC in 5, electrolyte and renal panel in 10, CPK in 6, drug and alcohol screen in 28, head CT in 8, and lumbar puncture in 3. The authors noted that all infections were detected by fever or lumbar puncture. This study sharply contrasts with the majority of literature, which reports a yield for routine screening as low as 0.05%. Most emergency physicians agree that mandatory testing of patients presenting for medical clearance is costly and time-consuming, and clinically insignificant abnormalities may subject an otherwise medically stable patient to additional testing and unnecessary delays in transfer. However, selective and directed diagnostic testing to identify possible medical conditions resulting in abnormal behavior is always appropriate.

Specific diagnostic testing

In patients with normal behavior, self-reporting of drug or alcohol use has been shown to be 92% sensitive and

91% specific for identifying a positive drug screen. Drug screens and blood alcohol levels are frequently ordered on patients in the evaluation of abnormal behavior. These tests assist with the diagnosis in obtunded patients. In addition, the absolute value of the blood alcohol level can be used to estimate the rate at which an intoxicated patient should sober (30–60 mg/dL/hr). Some psychiatric centers require blood alcohol levels and toxicologic screens before accepting a patient in transfer. Ecstasy, gamma hydroxybutyrate (GHB) and ketamine cause abnormal behavior but are not detected by routine urine drug screens.

Some literature reports that hypoglycemia is responsible for up to 10% of abnormal behavior seen in ED patients. Based on these numbers and the rapidity in which treatment should be rendered, immediate bedside testing of blood sugar is important for all patients who present with acute alterations in behavior.

Screening electrocardiograms (ECGs) are generally not necessary in the evaluation of abnormal behavior unless the patient has abnormal vital signs, symptoms or findings suggestive of acute coronary syndrome, or significant risk factors for a cardiac event (age >50 years, cocaine or stimulant use/abuse, or strong family history). If there is a suspicion that a patient ingested a tricyclic antidepressant, beta-blocker, calcium channel blocker, antidysrhythmic or other medication known to affect cardiac conduction, an ECG should be obtained and reviewed. Evaluation of the QT interval on ECG is important before initiating treatment with antipsychotic agents that prolong this interval.

Chest radiography is indicated in patients with cough, tachypnea, fever, or hypoxia. A low threshold for obtaining a chest X-ray in an elderly patient is essential, as pneumonia may present solely with abnormal behavior. CT scanning of the brain is reserved for patients with a headache, focal neurologic deficits, or those at risk for subdural hematomas (elderly, anticoagulant use, recent falls, trauma, or dialysis).

Lumbar puncture (LP) should be performed in patients suspected of having a subarachnoid hemorrhage (despite a negative head CT) or CNS infection. As a rule, anyone with fever, nuchal rigidity and altered mental status should have an LP. Patients who are immunocompromised may not mount a fever even in the presence of fulminant meningitis; therefore, they should have an LP whether or not fever is present. Most clinicians advocate obtaining a CT scan prior to LP in anyone with focal neurologic findings in order to assess for masses or radiologic signs of increased intracranial pressure.

General treatment principles

Ensure safety

The primary treatment principle of any patient presenting with abnormal behavior is ensuring the safety of the staff and the patient. The patient must be prevented from hurting others or himself. As a general rule, safety measures should be initiated as soon as possible (if needed), in a rapid, collaborative, rehearsed and stepwise fashion proceeding from the least to the most restrictive.

The setting for obtaining the history is important, especially with a potentially violent patient. The interview should be conducted in an environment of privacy but not isolation. Security personnel should be stationed outside the room in which the interview is being conducted. While in the room, the examiner should always remain between the patient and the door. Ideally, the room should have two points of exit so that both the physician and the patient have access to an exit should they feel threatened. During the history and physical examination, the physician should act as an advocate for the patient, not an adversary. Physicians should allow the patient to feel in control, while setting limits about inappropriate behavior. Interviewing the patient in a seated position has been shown to be effective in decompressing violent patients. Avoid prolonged eye contact and talk in a calm manner without being condescending. If at any time an examiner feels unsafe, he or she should leave!

Rule out conditions that require immediate action

Once the safety of the patient and staff has been established, the next step is to determine whether the altered behavior is a symptom of an underlying medical problem. Blood glucose, oxygenation status, fever and hemodynamic compromise should be rapidly addressed.

Determine the need for emergency psychiatric admission

Every state has conditions and laws set forth to provide for the involuntary admission of a mentally ill patient. These laws allow for a patient to be held for a set period of time (usually 72 hours) for further psychiatric evaluation and treatment if they are deemed dangerous to themselves, dangerous to others, or gravely disabled. Some states also have laws specific to alcohol or drug intoxication that make it possible to hold a patient for evaluation and treatment.

Implement physical or chemical restraint when necessary

Many patients who are agitated can be “talked down” using a calm and soothing voice. Inform the patient that you are his advocate and want to help. Speak clearly while remaining nonjudgmental. Ask the patient why he is upset and what could be done about it. For some patients, it may be appropriate to bargain using food or drink to gain control of the situation. The patient can be offered medication, either oral or parenteral, to calm him down. If these verbal interventions fail, proceed to a higher level of intervention called a “show of force.” A minimum of five trained staff are needed, one to control each extremity and one to control the head. An additional person serves as the leader. To begin, the security personnel gather around the leader to promote an image of confidence. The leader tells the patient to calm down or he will be restrained. The patient is then given a few seconds to back down. Many patients will respond to this demonstration of force. If a patient remains agitated or combative, it is then necessary to apply physical restraints. At the signal of the leader, the team controls the patient's extremities and head. Caution should be exercised at all times, as violent patients are prone to kick, swing, bite, spit and scratch while being restrained. The patient is taken down in a backward motion and then rolled over. The leader informs the patient why restraints are necessary. Restraints are then applied and the patient is properly positioned in either a prone or recumbent orientation. Avoid placing patients in the supine position as this is uncomfortable and increases the risk of aspiration.

Physical restraints serve as a bridge to chemical restraint. The goal of chemical restraint is rapid tranquilization. Two classes of drugs are used in the ED for chemical restraint: antipsychotics and anxiolytics. It is important to be familiar with these medications in the emergency setting. Cooperative patients should be offered oral medications as first-line agents. Traditionally, antipsychotics (known as neuroleptics) are the preferred first-line agent for controlling an agitated or violent patient. Haloperidol (Haldol) is the most common antipsychotic used in the ED for rapid chemical control of an agitated patient. The recommended adult dose is 5–10 mg IV or intramuscular (IM), repeated every 15–30 minutes until sedation is achieved. Haloperidol is a "low-potency" antipsychotic. It is associated with increased risk of extrapyramidal symptoms (EPS), which include dystonia (acute torticollis, oculogyric crisis and opisthotonos), akathisia, pseudoparkinsonism and tardive dyskinesia in the case of chronic use. EPS occurs < 1% of the time, and typically responds to anticholinergic medications, such as diphenhydramine 25–50 mg PO/IM/IV and benztropine 1–2 mg PO/IM/IV. "High-potency" antipsychotics (e.g., chlorpromazine) are associated with lower rates of EPS but have a higher incidence of prolonged sedation, cardiovascular toxicity and orthostatic hypotension, making them poorly suited for controlling an acutely agitated patient.

Ziprasidone, olanzapine and risperidone are newer "atypical" antipsychotics available in an oral formulation. Ziprasidone is also approved for IM injection to rapidly control agitated behavior and psychotic symptoms in patients with acute exacerbations of schizophrenia. Limited data suggest that patients may tolerate ziprasidone better than Haldol; however, this drug may cause greater QT prolongation that other antipsychotics. The recommend initial dose for ziprasidone is 20 mg PO or 10–20 mg IM. Olanzapine is also available in both an oral disintegrating tablet and parenteral form. The recommended initial dose of olanzapine is 5–10 mg orally or 10 mg IM for acute agitation. The oral formulation may be preferred over the injectable form (IM or IV administration) by some patients. Many of these drugs are rapidly adsorbed sublingually.

Droperidol, once preferred by many emergency physicians for rapid sedation and tranquilization of an acutely agitated patient, has received a "black box" warning by the FDA due to its potential to precipitate torsades de pointes in patients with underlying QT prolongation. One study estimates the incidence of this to be 4 in 1,100. It is important to note that many antipsychotic drugs can precipitate torsades de pointes. Antipsychotics should not be used in pregnant or lactating females, phencyclidine overdose, or anticholinergic-induced psychosis.

Anxiolytics may be used as single-line agents (especially when drug or alcohol intoxication or withdrawal is suspected), or as an adjunct to antipsychotics to control a violent patient. Benzodiazepines are the anxiolytics of choice in this situation – especially those with rapid onset and short half-lives. Lorazepam is one mainstay and can be given at a dose of 1–2 mg PO/IM/IV every 30 minutes. Numerous studies have shown that anxiolytics decrease the dosage requirements of antipsychotic agents when they are used in conjunction. Patients require lower doses of medication and the incidence of EPS is reduced. "HAC" is a mnemonic for Haldol (5 mg), Ativan (2 mg) and Cogentin (1 mg). This combination of medications can be given as a single IM injection. Care should be exercised when using multiple agents in elderly patients, as over-sedation is a concern. Midazolam is another short-acting benzodiazepine with very rapid onset of action, and has been given safely at a dose of 5 mg IM.

Perform frequent rechecks

The medical and psychiatric evaluation or transfer of a patient often takes time to complete. It is important that patients with abnormal behavior are frequently rechecked for over- or under-sedation, abnormal vital signs, seizures, emesis, or respiratory compromise. Older patients, those with significant comorbidities, and those with abnormal vital signs should be closely monitored while their disposition is being established. Patients who are agitated may need additional medication to achieve sedation. The Joint Commission mandates that patients who are physically restrained must have frequent reevaluation for extremity trauma, aspiration, respiratory compromise, pressure sores and skin injury, as well as repeated vital signs documented. This is good practice, as physically restrained patients are at increased risk for cardiovascular collapse for a number of reasons.

Special patients

Elderly

Elderly patients who manifest behavioral changes represent a special challenge. Alterations in behavior have been reported to be more common precursors of physical illness than fever, pain, or tachypnea. Urinary tract infections are often implicated as a cause of abnormal behavior in the elderly, especially females. Therefore, a low threshold should exist for obtaining a urinalysis in any elderly patient with a change in behavior. If the evaluation of an elderly patient is unrevealing, yet concern for an underlying medical problem remains, the patient should be admitted to a medical floor for further observation and evaluation.

Pediatric

In a recent national review by Sills, over 400,000 pediatric mental health visits occur annually, with 1.6% of all ED visits for this reason by individuals under 18 years of age. A 2009 study by Mahajan et al. reported this number to be as high as 3.3%. Unspecified neurotic state and depressive disorder are the most commonly diagnosed conditions in these patients. Nearly 14% of these patients were seen for suicide attempts. The World Health Organization estimates that by the year 2020, childhood psychiatric disorders will become one of the top five causes of morbidity, mortality and disability among children. Over the past few decades, a growing number of children have been prescribed psychoactive medications.

There is a higher incidence of ingestions and psychiatric illnesses in pediatric patients with abnormal behavior than adults. When a child with a psychiatric illness presents to the ED, there is often a breakdown of the family's support system. It is important to attempt to uncover what is not working at home. Furthermore, school, work, or social stressors may be even more problematic without a supportive home environment.

Suicide is currently the fourth leading cause of death in children 10–14 years of age and the third leading cause in children 15–19 years of age. A retrospective study by Porter demonstrated that adolescents with somatic complaints were infrequently screened for depression in the ED. Emergency care provides physicians an opportunity to intervene in children at risk for major depression or suicide.

Pediatric and adolescent patients requiring admission for psychiatric evaluation and treatment typically go to specialized facilities that deal only with pediatric patients. Because of a nationwide shortage of pediatric psychiatric beds, pediatric patients commonly experience extended stays in the ED.

Immune compromised

Patients who are immune compromised may not demonstrate abnormal vital signs even with serious medical illness. This is frequently demonstrated in patients with AIDS. In patients with a history of HIV, it is important to determine the history of any AIDS-defining illness. The patient and all medical records should be queried for recent lymphocyte counts. A low threshold for diagnostic testing should be maintained. Patients with HIV are susceptible to CNS infections such as toxoplasmosis, cytomegalovirus (CMV), herpes encephalitis, cryptococcal and bacterial meningitis, or CNS lymphoma with minimal focal neurologic findings. For this reason, any immunocompromised person with abnormal behavior, even if afebrile, should have a CT scan of the brain as part of the medical work-up before an LP.

Disposition

Admission

Patients with underlying medical problems as the cause for abnormal behavior require admission to the hospital for further evaluation and treatment. Patients with progressive dementia may no longer be safe in their current living situation, and might benefit from a social services evaluation or hospital admission. Skilled nursing facility placement may be difficult directly from the ED, but this possibility should be considered. Patients in whom underlying medical pathology cannot be safely eliminated should be admitted to a medical bed for further testing. Patients who are suicidal, homicidal, or gravely disabled should be placed on an emergency psychiatric hold and admitted to a psychiatric facility for further evaluation and treatment.

Depressed patients who do not actively endorse suicidal ideation can be difficult to disposition. One mnemonic and scoring system for the assessment of suicide risk is "SAD PERSONS" (Table 11.8). Scores of ≤ 6 are associated with low risks of suicide, whereas scores > 6 represent a higher risk of suicide and warrant hospitalization. Caution is necessary in any patient with the possibility of suicidal behavior, and liberal use of consulting psychiatric services is recommended.

Table 11.8 Assessment for suicide risk: SAD PERSONS

Sex: Male	1 point
Age: < 19, > 45 years	1 point
Depressed	2 points
Previous suicide attempt	1 point
Ethanol or any substance	1 point
Rational thinking absent	2 points
Separated or divorced	1 point
Organized suicide plan	2 points
No social support	1 point
Stated future attempt	2 points

Scores of ≤ 6 are associated with low risks of suicide, whereas scores > 6 represent a higher risk of suicide and warrant hospitalization. Caution is necessary in any patient with the possibility of suicidal behavior.

In 2004, the FDA required the manufacturers of medications used to treat depression to include a black box warning that there is an increased risk of suicide in children and adolescents. In 2007, the warning was expanded to include adults less than 25 years of age. Subsequent analysis performed by independent researchers has not verified this risk. Nonetheless, it is reasonable to carefully monitor children, adolescents and young adults who have recently been started on an antidepressant for increased symptoms of suicidal thoughts or gestures.

Consultation

A current list of on-call psychiatric care providers available to evaluate and treat patients with psychiatric emergencies should be maintained at each hospital. Attempts should be made to contact the patient's primary psychiatrist, psychologist, or therapist to assist with disposition. When contacting a mental health care provider, be certain to relay the events leading up to the ED visit, physical examination (including a complete set of vital signs), all treatment rendered in the ED, laboratory data (if any), and the status of the "medical clearance." Offering your assessment of the situation and your opinion of the circumstances to a consultant is appropriate.

Transfer

Depending on the hospital, patients requiring involuntary or voluntary psychiatric admission may need to be transferred to a psychiatric care facility after the medical screening examination has been completed. It is important that physician-to-physician communication occurs prior to transfer, and that the staff confirms bed availability before this transfer occurs. It is never appropriate to allow a family member or taxi service to transfer a patient for involuntary psychiatric admission. Caution should also be used for transfer arrangements of voluntary psychiatric admissions.

Observation/discharge

Most patients with abnormal behavior will not be released unless they are observed for an extended period in the ED. Patients who are discharged should have emergency medical and psychiatric causes of their abnormal behavior excluded. Family members or a responsible adult (preferably with transportation) should be involved in the discharge process. Patients suffering from mild drug ingestions or alcohol intoxication are frequently discharged from EDs after observation. Patients who, while intoxicated, threaten to commit suicide should be evaluated by a mental health professional even if they deny this claim once sober. In addition, patients with a stable psychiatric condition may be discharged if they are not suicidal (danger to self), homicidal (danger to others), or gravely disabled. In this situation, speaking directly with the patient's primary mental health provider is always preferred. Patients who are discharged should have intact support networks, a safe place to stay and reliable follow-up, preferably arranged prior to discharge. An appointment the next day or contact from the patient's psychiatrist or therapist is preferred. In most instances, a patient with abnormal behavior who is being released from the ED should be asked to "contract for safety" (sign an agreement stating they will not harm themselves) in the presence of a family member or reliable adult before leaving.

Pearls, pitfalls and myths

- Limited history from limited sources
- Incomplete review of systems
- Incomplete review of medications without considering drug–drug interactions or adverse effects
- Failure to document vital signs
- Failure to address abnormal vital signs
- Limited or incomplete physical examination, including vital signs and neurologic examination
- Unreasonable assumption of psychiatric illness without considering medical or traumatic etiologies, ingestion, intoxication, or adverse drug interaction

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12 Alcohol-related emergencies

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Scope of the problem

Alcohol abuse is a worldwide problem that places significant strain on health care resources, especially in the United States. The physiologic and psychologic issues associated with alcohol abuse and alcohol-related disease account for as many as 15% of emergency department (ED) visits. Binge drinking may result in coma and even death, particularly in adolescents and young adults. When considering intoxication, withdrawal syndrome, or alcohol-related injury or diseases, alcohol-related emergencies are common reasons people seek emergency medical care. Many of the diseases that develop from chronic alcohol abuse may be life-threatening. The physiologic, psychologic and social effects of alcohol abuse are likely to be devastating to patients and their families, an important consideration when caring for patients suffering from alcohol-related diseases.

Anatomic essentials

The term *alcohol* refers to ethanol or ethyl alcohol. The primary metabolism of ethanol is hepatic via alcohol dehydrogenase. The metabolism of ethanol has been studied to great extent. Typically, it follows zero-order kinetics for most people; however, it may follow first-order kinetics in high doses or chronic abusers. It is therefore best to describe alcohol metabolism as non-linear. As alcohol consumption increases, alcohol dehydrogenase activity increases. The average rate of alcohol metabolism in an adult is roughly 20–30 mg/dL/hr; occasional drinkers metabolize alcohol more slowly, and chronic alcohol abusers generally metabolize faster.

The hepatocytes are subject to direct chemical effects of ethyl alcohol, although almost all body systems are affected in some fashion. There is evidence that both sustained and binge drinking have the same toxic effect on the hepatocytes. In addition, the toxic effect of alcohol on hepatocytes can result in a chemical hepatitis.

Although the primary damage from alcohol is due to hepatic toxicity, alcohol affects nearly every organ system. The neurologic, endocrine, pulmonary, immunologic, hematologic and gastrointestinal systems are adversely affected in chronic alcoholics. Alcohol is primarily a depressant neurotransmitter in the central nervous system (CNS), mimicking GABA neuroregulators. In chronic alcohol abuse, GABA receptors are up-regulated; in alcohol withdrawal, the absence of GABA stimulation results in a hyperadrenergic state, with signs and symptoms of anxiety, tremors, diaphoresis, tachycardia and hypertension.

Chronic alcoholics often consume few calories other than alcohol, resulting in metabolic derangements and vitamin deficiency syndromes that can be life-threatening. Due to the multi-system influence of alcohol, patients are at risk for numerous medical conditions (Table 12.1).

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 12.2).

History

The primary history in a patient with alcohol abuse varies depending on the clinical presentation. In some cases, patients will present with an alcohol-related syndrome such as withdrawal; in other situations, only a careful history will identify a disease process related to alcohol consumption. The history commonly focuses on withdrawal symptoms, neurologic status, metabolic derangements, problems with bleeding and/or hemostasis, and secondary trauma related to excess ingestion of alcohol. Though a careful history can frequently identify alcohol-related conditions, many patients refuse to acknowledge that alcohol is a problem. Given the complex nature of addiction, patients may avoid questions related to their drinking habits. In order to establish a therapeutic relationship with the patient, it is important for emergency practitioners to demonstrate empathy rather than assign blame. Equally important is not allowing a history of prior alcohol abuse to overly influence medical decision making, such as attributing a change in mental status in a chronic alcoholic to intoxication rather than a possible life-threatening condition.

The CAGE questionnaire can quickly assess for the presence of ethanol abuse. The following four questions should be asked by emergency physicians and primary care providers:

C. Have you ever felt that you need to **C**ut down on your drinking?
A. Have people **A**nnoyed you by criticizing your drinking?
G. Have you ever felt bad or **G**uilty about your drinking?
E. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (i.e., an **E**ye-opener)?

Table 12.1 Alcohol-associated conditions

Diagnosis	Symptoms	Signs	Work-up
GI bleeding	Usually upper GI (gastritis, PUD, varices) Hematemesis Melena Fatigue from anemia	Pallor Tachycardia Hypotension Heme-positive emesis or stool	Immediate resuscitation with crystalloid and blood products Labs, coags, type and crossmatch Gastroenterology consultation
Alcoholic pancreatitis	Epigastric pain Nausea Vomiting	Epigastric tenderness Tachycardia Hypotension Dehydration	Volume replacement Labs, lipase (amylase if lipase unavailable) US or CT if necrotizing or pseudocyst suspected
Alcoholic hepatitis	Abdominal pain Nausea Vomiting Jaundice	RUQ tenderness Fever Elevated transaminases (AST > ALT) Elevated bilirubin	Volume replacement Labs, LFTs, coagulation factors Carries high mortality; generally requires admission
Alcoholic ketoacidosis	Fatigue Dehydration Abdominal pain Nausea Vomiting	Dehydration Nonspecific exam findings	Volume replacement Labs with elevated anion gap acidosis Give food or IV glucose to return to fed state and to correct acidosis
Trauma	Depends on type of trauma	Various injuries	Resuscitation as necessary Diagnostic imaging and labs Alcohol counseling: disinhibition, poor judgment and aggression lead to trauma
Vitamin and nutritional deficiency syndromes (such as Wernicke's encephalopathy)	Memory loss Confusion Confabulation	Gait disturbances Ophthalmoplegia	Thiamine replacement Folate replacement Return to fed state with glucose or a meal Balanced diet and vitamin rich food Hydration

ALT: alanine aminotransferase; AST: aspartate aminotransferase; CT: computed tomography; GI: gastrointestinal; IV: intravenous; LFTs: liver function tests; PUD: peptic ulcer disease; RUQ: right upper quadrant; US: ultrasound.

Table 12.2 Alcohol-related emergencies red flags

History	Concerning diagnosis
Acute or chronic ingestion	Acute alcohol toxicity, coma, malnutrition, starvation or alcoholic ketoacidosis, Wernicke-Korsakoff syndrome, hypoglycemia, hyponatremia, coagulopathy, seizures, alcohol withdrawal
Alcohol substitute	Ethylene glycol, methanol, or isopropyl alcohol toxicity, Listerine toxicity
Falls	Closed head trauma, subdural or intracranial hemorrhage, contusions, fractures, spinal cord injury, other injuries
Dyspnea	Aspiration pneumonia, tuberculosis, pulmonary infection
Weakness	Malnutrition, co-ingestion, liver disease, anemia
Neurologic symptoms	Stroke, electrolyte derangement, abnormal metabolism
Examination finding	**Concerning diagnosis**
Bruises, ecchymoses	Trauma, coagulopathy
Tremor, fasciculations	Seizure (withdrawal), liver disease, hypoglycemia
Muscle atrophy, cachexia	Malnutrition, immunodeficiency
Asterixis, spider angioma, telangiectasia, caput medusa, ascites, jaundice	Liver disease, portal hypertension, coagulopathy
Abnormal vital signs (sympathetic overdrive), altered sensorium, agitation	Delirium tremens
Encephalopathy, psychosis, confabulation, ataxia, visual disturbance	Wernicke-Korsakoff syndrome

Responses are either "yes" or "no," with 1 point given for each "yes" response. A score of 0–1 suggests the normal range and an individual at low risk of problem drinking (i.e., no apparent problem). It is important to remember these questions are for screening purposes only. A score of ≥2 is thought to be clinically significant and may indicate alcohol dependence. This score suggests a risk of problem drinking or alcoholism. Additional questions may further determine the risk for acute alcohol-related illness.

How much do you drink on a typical day?

Although self-reporting is an unreliable method for determining the quantity of alcohol consumed, it may differentiate casual social drinking from abuse. Many patients will have periods of significant drinking followed by brief periods of abstinence. Binge drinking, defined as four or more drinks within a 2-hour period, can have the same long-term effects as chronic daily consumption of alcohol. Some patients may feel that drinking certain kinds of alcohol reduces their health risk (e.g., beer is less toxic then distilled liquor); however, the long-term effects of alcohol abuse are the same regardless of the type of alcohol consumed.

Have you ever had a seizure when you stop drinking?

This question is important to assess for potential alcohol withdrawal symptoms. A history of seizures, anticonvulsant use, delirium tremens with hospitalization for drinking, or head injury helps emergency physicians assess the risk for more complicated withdrawal symptoms. Seizures may occur due to hyponatremia; beer potomania is uncommon, yet can cause hyponatremia.

Have you been vomiting?

Vomiting can be present in more severe alcohol-related conditions such as pancreatitis, alcoholic hepatitis and head injuries, as well as intoxication. Vomiting blood may represent upper gastrointestinal bleeding, which may be life-threatening in a chronic alcoholic due to liver disease, portal hypertension, esophageal varices, or coagulopathy. Inability to maintain hydration status due to vomiting may be an indication for admission or additional observation.

Have you had a fever?

Chronic alcohol abuse impairs the immune system; therefore, infections such as pneumonia, cellulitis, abscess or endocarditis are common in chronic alcoholics. A febrile alcoholic is at serious risk for sepsis and complex infections with high mortality. In addition, conditions such as alcoholic hepatitis can produce a fever.

Have you had any abdominal pain?

Abdominal pain is a concerning symptom in an alcoholic patient. Pancreatitis, gastritis, ulcers, perforation and alcoholic hepatitis are several potential causes of abdominal pain. Abdominal pain may also be the result of electrolyte abnormalities with acidosis.

Do you notice any blood in your stool or emesis, nosebleeds, or bleeding gums?

Alcoholics have many reasons for bleeding. End-stage liver disease can cause bleeding due to loss of the liver's synthetic function. In addition, alcohol abuse causes thrombocytopenia secondary to marrow suppression. Platelet dysfunction is also common. Bleeding due to alcohol abuse should raise serious concern, and may signify end-stage liver disease.

Have you noticed that you've been bruising more easily?

This question explores whether there is additional evidence of bleeding and loss of synthetic function of the liver. Bruising is also likely to be present following trauma. Multiple falls and resulting traumatic injuries are common; many patients may attempt to explain away bruising. Closed head injuries from falls are common in alcoholic patients and are often more serious than might be expected due to platelet and coagulation abnormalities.

Have you been seeing things that are not there or hearing voices?

Visual hallucinations are common with severe alcohol withdrawal, and may portend severe withdrawal syndromes. Auditory hallucinations are generally not related to alcohol withdrawal, and may be part of an underlying psychiatric disorder.

Have you noticed your skin looking yellow or dark urine?

This line of questioning can screen for signs of alcoholic liver disease. It is not uncommon for alcoholic patients to come to the emergency department after developing jaundice, which may represent end-stage liver disease or acute alcoholic hepatitis.

Past medical

Patients should be asked about underlying cardiovascular, gastrointestinal, neurologic, endocrine, hematologic, renal, psychiatric and immunologic diseases that may be exacerbated by chronic alcohol abuse or alcohol withdrawal. For example, a patient with dilated cardiomyopathy may decompensate from the adrenergic stress of acute alcohol withdrawal. Furthermore, in a patient with coronary artery disease or a tenuous myocardial blood supply, the tachycardia of dehydration or acidemia may cause acute cardiac ischemia from demand ischemia, anemia of chronic disease, or adrenergic excess.

Physical examination

Given that alcohol affects the entire body, a thorough physical examination is always necessary.

General appearance

The general appearance is quite varied in patients suffering from alcohol abuse. The acutely intoxicated patient often presents disheveled and without much attention to appropriate modesty. They may be verbally or physically aggressive, somnolent, unresponsive, or anywhere in between. They often smell of ethanol byproducts, urine, vomit, stool or the outdoors, depending on their circumstances. They may have signs of obvious traumatic injury or injuries at various stages of healing. Patients in acute alcohol withdrawal may appear quite ill, diaphoretic, tremulous or agitated. It is important to screen for subtle or obvious areas of trauma, especially evidence of closed head injury.

It is important to remember that patients suffering from chronic alcohol abuse are from all economic and cultural backgrounds. They are likely to be of all age groups, including adolescents and the elderly, and do not always fit a specific "stereotype." A person suffering from severe alcohol-related disease may be well dressed and groomed, and very good at hiding their drinking history.

Vital signs

Special attention to the vital signs is critical when assessing a patient with alcohol abuse. Vital signs can be used to predict the severity of the condition as well as response to therapy. Patients may be hyperthermic, especially in acute withdrawal or delirious states, or hypothermic, due to the vasodilatory effects of alcohol and possible environmental exposure. A few important points should be remembered:

- Heart rate can quickly determine the severity of alcoholic withdrawal. Patients can appear quite calm but may be significantly tachycardic while experiencing alcohol withdrawal.
- Hypertension is common in withdrawal. Severe hypertension in the face of altered mental status can be the result of *delirium tremens (DT)*, as well as intracranial bleeding.
- Hypotension can signify serious hypovolemia secondary to trauma, dehydration, or gastrointestinal bleeding.
- Fever is a serious sign in alcoholic patients. Regardless of the cause, the presence of a fever in the alcoholic patient mandates rapid, decisive evaluation.

Head, eye, ear, nose and throat

It is critical to identify new or old injuries and any evidence of head trauma. Lacerations, bruising, contusions and step-off fractures of the skull and face must be appreciated. Bony crepitus and subcutaneous emphysema may signify facial fractures. The eyes and pupils should be examined for scleral icterus, ophthalmoplegia, extraocular movements, nystagmus and asymmetric pupils in the setting of altered mental status. The nose should be inspected for evidence of fracture, bleeding or nasoseptal hematoma from traumatic injuries. The oropharynx should be assessed for hydration, tongue fasciculations, infections and trauma, as well as intraoral injuries that can be present in withdrawal or alcohol-related seizures.

Neck

If the potential for trauma exists, the cervical spine should be palpated for tenderness. Cervical spine immobilization may be necessary, or may have been placed by paramedics in the field. Caution against aspiration must be maintained in a supine, immobilized patient who is intoxicated. Cervical spine fractures are a concern in patients suffering from chronic alcohol abuse, as they may have had prior falls or unknown traumatic injuries. Therefore, clinicians should maintain a low threshold for obtaining diagnostic imaging of the cervical spine.

Chest

Inspect the chest for bruising related to trauma, and palpate for the presence of point tenderness or instability. The lungs should be auscultated anteriorly and posteriorly in all fields for asymmetry, consolidative changes, or decreased breath sounds. Wheezing or rhonchi is common in alcohol abusers, with gross or microaspirations causing pneumonia or bronchitis.

Abdomen

Locate any tenderness, palpate the liver edge, and identify evidence of end-stage liver disease. Examine the abdomen for the presence of ascites. Tenderness (especially with fever) in the face of ascites may signify spontaneous bacterial peritonitis. Individuals who are acutely or chronically intoxicated may still develop abdominal pathology, such as appendicitis, cholecystitis, spontaneous bacterial peritonitis (in the setting of ascites), hepatomegaly, splenomegaly, diverticulitis, splenic rupture or infarction, hepatitis, and hepatic or splenic abscesses. Acute abdominal aortic aneurysm (AAA) remains a life-threatening condition in alcoholics.

Rectal

If blood loss is suspected from gastrointestinal hemorrhage, rectal examination for hemoccult testing of stool should be performed. Hemorrhoids that may result in blood loss should be identified, as well as any rectal or perirectal masses that might suggest an abscess.

Extremities

With the patient undressed, look at the skin and extremities for evidence of trauma and bruising. Examine for fine motor tremors, which often signify alcohol withdrawal. The skin may be hairless, waxy, cool and dry, with multiple healing scars from injuries and abscesses, indications of peripheral vascular disease.

Neurologic

The neurologic examination is probably the most important component of the physical examination in patients suffering from alcohol-related disease. It is important to perform a careful mental status examination. Pay close attention to thought content and identify possible confabulation. Head trauma, delirium tremens and encephalopathy all cause altered mental status. Perform a thorough motor examination. Asterixis may represent end-stage liver disease and encephalopathy. Assess cranial nerves, cerebellar function and deep tendon reflexes. When safe, the patient should ambulate (with assistance) to test balance, proprioception and proximal muscle strength. Severe withdrawal symptoms, dehydration, motor weakness and CNS toxicity may make ambulation extremely difficult.

Differential diagnosis

The differential diagnosis for alcohol-related emergencies can be divided into three common categories: acute alcohol intoxication, alcohol-associated conditions, and alcohol withdrawal syndrome. Acute alcohol intoxication is a common complaint in the emergency department. It is important that emergency physicians are familiar with agents that mimic alcohol as a cause of altered mental status (Table 12.3), as treatments differ despite similar presenting complaints. Other alcohol-associated conditions may range from simple, reversible metabolic syndromes to more fulminant, life-threatening conditions. Emergency physicians must be able to rapidly recognizing these conditions as well (Table 12.1). Patients with alcohol withdrawal commonly seek emergency care at different stages of their withdrawal syndrome. Withdrawal symptoms are uncomfortable and potentially life-threatening. Table 12.4 lists the common presentations of alcohol withdrawal, as well as other drugs that mimic alcohol withdrawal.

Table 12.3 Alcohol and intoxicants that cause altered mental status

Diagnosis	Symptoms	Signs	Work-up
Acute alcohol intoxication	Altered mental status Disinhibition Nausea/vomiting CNS depression	Normal vital signs usually Respiratory depression at very high levels of ethanol Nystagmus	Mild anion gap acidosis from ethanol alone Ethanol level to confirm diagnosis Chemistry panel for ketoacidosis
Methanol/ethylene glycol/ isopropyl alcohol intoxication	Altered mental status Nausea/vomiting Abdominal pain CNS depression Visual findings (snowstorm) in methanol intoxication	Tachypnea to compensate for metabolic acidosis (methanol, EG) GI irritation, including hemorrhagic gastritis, rhabdomyolysis, renal dysfunction (IPA) Blindness and papilledema or papillitis (methanol) Kidney stones, hematuria, proteinuria (EG causes calcium oxalate stone formation and renal tubule dysfunction) Fruity breath (IPA metabolized into acetone)	Suspect methanol or EG with large anion gap acidosis and osmolar gap Hypocalcemia and calcium oxalate crystals in urine in EG toxicity Elevated creatinine (false) Hypoglycemia Osmolar gap with IPA ingestion; anion gap unlikely Need specific toxicology levels for confirmation
Benzodiazepine intoxication	Altered mental status CNS and respiratory depression	Extreme sedation without other toxidrome findings Normal vitals Midrange pupils Exam otherwise unremarkable	Urine toxicology Gas chromatography confirmation of specific compounds Can reverse with flumazenil in acute setting only (*caution: may trigger seizures in chronic abusers or in those with co-ingestion*)
Barbiturate intoxication	Altered mental status CNS depression	Extreme sedation HR/BP/RR depression Decreased tone/reflexes Midrange pupils Exam otherwise unremarkable	Urine toxicology Gas chromatography confirmation of specific compounds
Opiate intoxication	Altered mental status CNS and respiratory depression	Extreme sedation HR/BP/RR depression Respiratory depression most consistent sign Miosis Decreased bowel sounds	Urine toxicology Gas chromatography confirmation of specific compounds Can reverse with naloxone

BP: blood pressure; CNS: central nervous system; EG: ethylene glycol; GI: gastrointestinal; HR: heart rate; IPA: isopropyl alcohol; RR: respiratory rate.

Table 12.4 Alcohol and other drug withdrawal syndromes

Diagnosis	Symptoms	Signs	Work-up
Mild alcohol withdrawal	Tremors Abdominal pain Nausea Vomiting Hallucinations	Diffuse tremor Tachycardia Hypertension Tachypnea Mental status normal, or agitated from hallucinations	Volume repletion Labs if associated conditions suspected Ethanol level rarely useful GABAergic medications for control (benzodiazepines, barbiturates)
Alcohol withdrawal seizures	Above symptoms plus: Seizures	Generalized seizures only; no partial complex seizures May have multiple seizures but all occur within an 8–12 hour (short) period, not days Can occur anytime during abstinence No status epilepticus Mental status normal after short postictal phase	Volume replacement Labs for associated conditions GABAergic medications for control (benzodiazepines, barbiturates, propofol if intubated) Phenytoin does not stop seizures, no role in ethanol withdrawal Seizure precautions
Delirium tremens (DT)	Above symptoms plus: Hyperthermia Altered mental status Adrenergic instability	Severe tremor Tachycardia Hypertension Tachypnea Diaphoresis Fever Profoundly altered mental status	Volume replacement Labs for associated conditions GABAergic medications for control (benzodiazepines, barbiturates, propofol if intubated) ICU admission
Benzodiazepine withdrawal	Nightmares Restlessness Insomnia Paranoia Tremor Confusion Psychosis Seizures	Altered mental status Often more mood disturbances than ethanol withdrawal Tachycardia Hypertension *Severe:* DT-like presentation	Volume replacement Labs for associated conditions GABAergic medications for control (benzodiazepines, barbiturates, propofol if intubated) Prefer long-acting GABA agonists for smoother recovery (diazepam, chlordiazepoxide, phenobarbital) Symptoms may last months
Barbiturate withdrawal	Hyperactivity Anxiety Insomnia Confusion Seizures	Altered mental status Often more mood disturbances than ethanol withdrawal Tachycardia Hypertension Hyperreflexia *Severe:* DT-like presentation	Symptomatic relief Observation in a monitored setting
Opiate withdrawal	Agitation Anxiety Insomnia Sweating Yawning Lacrimation Piloerection Abdominal cramping Nausea Vomiting Diarrhea Myalgias	Mood disturbances Tachycardia Increased bowel sounds No seizures or delirium No life-threatening findings from opiate withdrawal	Volume replacement Labs for associated conditions Give either long-acting opiates (methadone or buprenorphine) or clonidine to decrease symptoms from central adrenergic discharge

Diagnostic testing

Diagnostic studies are utilized to evaluate alcohol intoxication, withdrawal and associated conditions. For acute alcohol intoxication, history and clinical judgment are used to diagnose intoxication rather than reliance on labs. Patients with alcohol withdrawal generally require more extensive evaluation than patients with simple intoxication. However, depending on the patient's comorbid conditions and overall state of health, additional testing is likely to be needed. For alcohol-associated conditions (e.g., alcoholic ketoacidosis, gastrointestinal hemorrhage, alcoholic hepatitis, pancreatitis, infections, traumatic injuries), laboratory tests are ordered depending on the patient's presenting signs and symptoms, as these conditions may be difficult to detect through history and physical examination alone, especially in unreliable patients.

Laboratory studies

Point-of-care glucose test

The most important test in an intoxicated patient fortunately can be done rapidly at the bedside (or in the field) with reliable results. The bedside (point-of-care) fingerstick glucose is essential in patients with altered mental status, alcoholism, seizures or stroke-like symptoms.

Hypoglycemia may be life-threatening, is easily corrected, and is common in alcohol abusers who have low glycogen stores and often rely on alcohol for caloric intake. In patients with glucose levels performed in the field, consider repeating them if the transport time was long or the patient is in the ED for extended observation, especially if the patient's mental status changes or is not normal.

Serum ethanol level

Blood ethanol levels should be used in conjunction with the patient's history and clinical condition to differentiate intoxication from other conditions, not merely "confirm" intoxication. In other words, if a patient who is acting "intoxicated" has an ethanol level that doesn't explain his or her level of altered mental status, other conditions must be investigated to explain their clinical presentation. A serum ethanol level should not be obtained for legal purposes; this is the purview of law enforcement agencies. If a patient has a history of recent alcohol intake and an examination consistent with intoxication, confirmation of alcohol use with a serum ethanol level is generally of little value. A single ethanol level in a given patient does not always correlate with the level of clinical intoxication. For instance, an occasional alcohol drinker may be quite intoxicated at an ethanol level of 60 mg/dL, whereas a chronic alcoholic may not become altered until 300 mg/dL or higher.

Obtaining a repeat ethanol level is also not required when deciding if an intoxicated patient is ready for discharge from the ED. Because most individuals metabolize ethanol at approximately 20 mg/dL/hr (chronic alcoholics metabolize ethanol slightly faster), a patient's approximate ethanol level can be estimated following a period of observation. There is no defined ethanol level at which a patient is "safe" for discharge. If a patient is able to communicate appropriately, ambulate safely, eat and drink successfully, and understand discharge instructions, he is likely ready for discharge. Ideally, a patient who has been drinking and is now clinically "sober" should not be allowed to drive, should have a place to go, and should be released with another adult (who has been given and understands the discharge instructions). Social services, shelter information, chemical and/or alcohol dependency literature, telephone numbers and education (intervention) should be provided the patient. In rare cases, practitioners may feel obligated to obtain ethanol levels as confirmatory documentation to include in the medical record, although this is not necessary. In California, psychiatric facilities will not accept intoxicated patients who require hospitalization for psychiatric purposes until an ethanol level is documented to be less than 100 mg/dL. Many EDs now use breathalyzers to estimate a serum ethanol level, which expedites confirming that it has decreased to an acceptable level.

Complete blood count

Alcohol abusers should be evaluated with a complete blood count (CBC). Alcoholic patients may have a leukocytosis due to infection, or leukopenia secondary to chronic bone marrow suppression. Evaluate the hemoglobin and hematocrit, looking for anemia from occult bleeding. The mean corpuscle volume (MCV) is typically increased in alcoholics due to folate deficiency and other factors, such as the direct effect of alcohol on the red cell membrane or bone marrow. MCV can be elevated in elderly patients whether or not they consume alcohol. Chronic alcoholic patients may have depressed platelet counts secondary to the toxic effects of alcohol on the bone marrow.

Metabolic panel

It is critical that the electrolytes are evaluated in alcoholic patients, especially those acutely intoxicated with altered mental status, chronic and elderly alcoholics with multiple comorbidities, and those having seizures, alcohol withdrawal, or delirium tremens. Electrolyte disturbances are common due to poor nutritional status and renal dysfunction. The potassium level should be noted, as hypo- or hyperkalemia may result in life-threatening dysrhythmias. The bicarbonate level should be noted as well, as low bicarbonate may indicate the presence of an anion gap acidosis (especially in patients with alcoholic ketoacidosis). In addition, ingesting methanol or ethylene glycol (toxic alcohols that cause significant morbidity) can cause profound anion gap metabolic acidoses. The patient's glucose should be noted (see above). Patients with long-standing alcohol abuse may have limited gluconeogenic capacity in addition to depleted glycogen stores, resulting in hypoglycemia and altered mental status. Kidney disease is not uncommon in patients with substance abuse.

Liver function tests

Liver function tests (LFTs), especially hepatic transaminases, are commonly elevated in a patient with chronic alcohol abuse. The aspartate aminotransferase (AST) is typically much greater than the alanine aminotransferase (ALT) in alcoholic liver disease. The gamma-glutamyl transferase (GGT) is a liver enzyme that is elevated in alcoholics. Also noting the bilirubin is important, because a rising bilirubin in the face of fever and right upper quadrant tenderness suggests alcoholic hepatitis, a condition that can cause rapid decompensation.

Lipase

A serum lipase should be ordered if alcoholic pancreatitis is suspected. Chronic pancreatitis may not result in an elevated lipase level. Neither serum lipase nor amylase correlates well with the amount of patient suffering due to alcohol-related pancreatic disease.

INR and tests of coagulation

The international normalized ratio (INR) should be checked if there is a concern regarding the synthetic function of the liver. Coagulation studies can be used as surrogate indicators of hepatic function and are readily available in the emergency department. Factor VII, which is vitamin K–dependent and produced by the liver, has the shortest half-life of the factors in the extrinsic arm of the coagulation cascade. Thus, the prothrombin time (PT) and possibly INR may be abnormal, indicating liver dysfunction.

Urinalysis

A point-of-care urine dipstick is important to look for evidence of infection, ketones, blood and urobilirubin.

Calcium, magnesium, phosphorus

These labs should be obtained in individuals with chronic alcoholism or malnutrition, as they are essential to metabolism. Hypocalcemia is common in ethylene glycol ingestion, and abnormalities of all three of these ions may occur in any alcohol-related condition. It is important to note that serum hypomagnesemia only occurs in the most severe stages of whole-body hypomagnesemia; therefore, a normal serum magnesium level does not accurately reflect magnesium stores. Because of this, clinicians often empirically give magnesium with other vitamins to alcoholics even with a normal serum magnesium level.

Serum osmolality

The serum osmolality can be both measured and calculated. It is measured by freezing-point depression.

Calculating the serum osmolality (osm) requires the serum sodium (Na^+), glucose (Glu), blood urea nitrogen (BUN) and alcohol (EtOH) levels.

$$\text{serum osm} = 2\text{Na}^+ + \text{Glu}/18 + \text{BUN}/2.8 + \text{EtOH}/4.2$$

Any difference between the calculated and measured serum osmolality is known as an *osmolar gap* (normal <10 mOsm/kg). An elevated osmolar gap signifies that osmotically active substances such as methanol, ethylene glycol, isopropanol, acetone or mannitol are present in the patient's serum. The contribution of these osmotic agents can be roughly estimated using approximations of their molecular weights and conversion factors.

Ethylene glycol, methanol, isopropyl alcohol (isopropanol)

It is also important to keep in mind that patients who drink large quantities of ethanol may also ingest other alcohols, such as isopropyl alcohol, methanol or ethylene glycol, as ethanol substitutes. This may be especially true when a patient suffers from uncomfortable withdrawal symptoms and has no access to regular ethanol. Emergency physicians must always consider this possibility when evaluating a critically ill patient. These agents may be ingested in suicide attempts. Specific levels of these agents can be ordered at most hospital labs, although these tests often must be sent out for processing.

Radiologic studies

Imaging of any type must be directed by the history and physical examination. Emergency physicians often (and perhaps correctly) have a low threshold to obtain diagnostic imaging because of the frequent episodes of blunt trauma that accompany alcohol intoxication and alcohol withdrawal. Acute, subacute and chronic traumatic injuries are common findings in the evaluation of chronic alcoholic patients. In fact, a good initial approach to evaluating patients with either alcohol intoxication or alcohol withdrawal is to completely expose them and perform a complete examination evaluating for occult injuries.

Chest radiographs may be necessary to investigate for pneumonia (possibly from aspiration), rib fractures and pneumothorax, and to assess cardiac silhouette and mediastinal structures. Evidence of head injury in an altered, chronic alcoholic patient often necessitates computed tomography (CT) of the brain. Because of liver dysfunction, coagulopathy, platelet disorders, and their tendency to fall or have altercations, alcoholic patients are at increased risk for intracranial trauma, including subdural and epidural hematoma, petechial hemorrhage, contusion and skull fractures. Focused assessment with sonography for trauma (FAST) scanning of the abdomen may be necessary to rule out intra-abdominal bleeding. Abdominal CT imaging may be necessary to look for hemorrhagic pancreatitis or other conditions that cause hypotension, abdominal pain, or altered mental status. Ultrasound of the chest is used by emergency physicians at many centers to assess for pneumothorax, pericardial effusion and cardiac dysfunction.

General treatment principles

Acute ethanol intoxication

The effects of alcohol intoxication or agents that mimic alcohol intoxication are summarized in Table 12.3. Treatment of acute alcohol intoxication is mainly supportive, but begins with an assessment of the ABCs (airway, breathing, circulation). As the patient metabolizes ethanol, the CNS depressant effects and GI effects should gradually resolve, and patients usually return to their baseline status. In the ED, clinicians frequently must manage intoxicated patients who are agitated. Disinhibited, intoxicated patients commonly require sedation, as these patients may be belligerent and present a safety threat to themselves and the ED staff. The goal of chemical sedation is to minimize the use of physical restraints, which have been associated with higher morbidity and mortality when compared with chemical restraint.

Common classes of the agents used for sedation include benzodiazepines and butyrophenones. The ideal sedative agent would result in rapid tranquilization without significant cardiovascular or respiratory effects, and would metabolize over several hours. Benzodiazepines (e.g., lorazepam, diazepam and midazolam) are the most commonly used sedative agents in the agitated, alcoholic patient. In order to achieve adequate chemically induced sedation in the intoxicated patient, large doses of these agents may be required, which may cause significant respiratory depression.

Other agents that have been used to chemically restrain intoxicated and agitated patients are butyrophenones haloperidol and droperidol. The advantage of these agents over benzodiazepines is greater sedation with little to no respiratory depression. Several trials comparing these agents with benzodiazepines have found them to be as effective for chemical restraint with fewer side effects. Unfortunately for emergency physicians, at the time of this publication, droperidol has a black box warning from the FDA. Butyrophenones are not as effective for the sole treatment of alcohol withdrawal, however, as they lack GABA effects.

Alcohol-associated conditions

Common conditions that result from alcohol abuse are summarized in Table 12.1. The treatment of alcohol-associated conditions depends on the specific pathophysiologic process responsible.

Metabolic and nutritional deficiencies are common in patients suffering from alcohol abuse. Patients fail to consume diets consisting of complex carbohydrates and vitamins; instead, ethyl alcohol is often the only (or predominant) carbohydrate they ingest. Ketoacidosis from lack of glucose and Wernicke's encephalopathy due to thiamine deficiency are examples of metabolic and nutritional deficiencies common in alcoholics. Replacing vitamins such as thiamine and folate is very important in patients suffering from chronic alcohol abuse. In addition, returning the person to a "fed" metabolic state is important. A balanced meal can have significant benefit in the ED; not only is it a test to see if the patient can tolerate oral intake, but it provides calories and carbohydrates needed for metabolism. Intravenous glucose should be provided if the patient is unable to eat. Fluids and glucose will rapidly correct alcoholic ketoacidosis. With proper treatment, alcoholic ketoacidosis can resolve within hours. Thiamine deficiency and the resulting altered mental status may take several days and large quantities of vitamin B1 to correct. Patients with severe thiamine deficiency generally require admission. There is a theoretical risk of precipitating Wernicke's encephalopathy if glucose is given to a thiamine-deficient patient; consequently, thiamine is generally administered simultaneously with the glucose. Alcoholic patients are typically magnesium and phosphate depleted. These should be replaced, although caution is necessary if renal dysfunction is present.

Patients with abdominal pain may require evaluation for occult infection, gastrointestinal bleeding, pancreatitis, hepatitis, perforated ulcer, or ketoacidosis. If there are signs of gastrointestinal bleeding, coagulation profiles and blood type and crossmatch should be ordered. Patients with peptic ulcers or esophageal varices may present in hemorrhagic shock and require volume resuscitation, immediate blood transfusion, fresh frozen plasma, intubation for airway protection, and early gastroenterology consultation. The specific management of gastrointestinal bleeding is covered in Chapter 29.

Traumatic injuries associated with alcohol intoxication require careful evaluation. Patients may be victims of trauma from others or from falls due to unsteady gait. Patients who are intoxicated may have contact with law enforcement and sustain injuries during altercations. Patients who require physical restraint in the ED to prevent injury to themselves or staff may injure themselves while in restraint, either because of the restraints or because of their movements while restrained. The Joint Commission requires that patients who are physically restrained be continuously monitored. Chemical restraints used alone or in combination with physical restraints may also result in adverse reactions, especially respiratory compromise. Therefore, patients who require chemical and/or physical restraints must have frequent and close supervision while in the ED.

Intoxicated patients should be observed and serially reexamined until they are clinically sober. This is done to ensure they have a reasonable ability to care for themselves following release, as well as to identify occult injuries or conditions that might have been missed during their initial evaluation. Serial assessments of the patient's vital signs, mental status and abdominal examination should be timed and documented in the medical record. Once a patient is considered clinically sober, this clinical assessment should be documented to support their final disposition.

For patients who ingest methanol, ethylene glycol or isopropyl alcohol (isopropanol), specific therapies are indicated in addition to careful assessment of the ABCs and supportive care. For both methanol and ethylene glycol ingestions, which cause profound anion gap acidosis (uncommon in isopropanol), sodium bicarbonate therapy has been recommended. Fomepizole (4-methylpyrazole) has been studied as an antidote and has essentially replaced the administration of ethanol (parenteral or enteral) in the treatment of methanol and ethylene glycol ingestion. In methanol ingestion, folinic (or folic) acid may be of value to increase formate metabolism. The administration of calcium may be required if hypocalcemia is present in ethylene glycol ingestion, as should pyridoxine, thiamine and magnesium cofactors to assist in its metabolism. Emergent hemodialysis is recommended for patients who have ingested toxic alcohols and are hemodynamically unstable, have measured concentrations >50 mg/dL of methanol or ethylene glycol, fail to correct their symptoms of acidemia with above treatments, or have visual impairment (methanol) or renal failure (ethylene glycol). As charcoal does not bind these toxic alcohols (including ethanol) and may be associated with aspiration, it is not recommended unless co-ingestion is suspected.

Alcohol withdrawal syndromes

Common conditions associated with ethanol withdrawal are summarized in Table 12.4. The treatment of alcohol withdrawal is based on the degree of withdrawal the patient is experiencing. The Clinical Institute Withdrawal Assessment for Alcohol, revised (CIWA-Ar, or simply CIWA) protocol has been adopted by many clinicians to evaluate and treat patients experiencing withdrawal symptoms from alcohol. Its use is predominantly in the inpatient setting, where it has been validated (it has yet to be validated in the ED setting). However, familiarity with this protocol and scoring system is important because patients with alcohol-related conditions are being monitored and managed in emergency observation settings more commonly and for longer periods of time (Table 12.5). Because

Table 12.5 Clinical Institute Withdrawal Assessment for Alcohol scale, revised (CIWA-Ar)

Patient:________________Date:_______________Time:_______________(24-hour clock, midnight = 00:00) **Pulse or heart rate, taken for one minute:________________Blood pressure:________________**	
NAUSEA AND VOMITING – Ask “Do you feel sick to your stomach? Have you vomited?” Observation: 0 No nausea and no vomiting 1 Mild nausea with no vomiting 2 3 4 Intermittent nausea with dry heaves 5 6 7 Constant nausea, frequent dry heaves and vomiting	**TACTILE DISTURBANCES –** Ask “Have you any itching, pins and needles sensations, any burning, any numbness, or do you feel bugs crawling on or under your skin?” Observation: 0 None 1 Very mild itching, pins and needles, burning or numbness 2 Mild itching, pins and needles, burning or numbness 3 Moderate itching, pins and needles, burning or numbness 4 Moderately severe hallucinations 5 Severe hallucinations 6 Extremely severe hallucinations 7 Continuous hallucinations
TREMOR – Arms extended and fingers spread apart Observation: 0 No tremor 1 Not visible, but can be felt fingertip to fingertip 2 3 4 Moderate, with patient’s arms extended 5 6 7 Severe, even with arms not extended	**AUDITORY DISTURBANCES –** Ask “Are you more aware of sounds around you? Are they harsh? Do they frighten you? Are you hearing anything that is disturbing to you? Are you hearing things you know are not there?” Observation: 0 Not present 1 Very mild harshness or ability to frighten 2 Mild harshness or ability to frighten 3 Moderate harshness or ability to frighten 4 Moderately severe hallucinations 5 Severe hallucinations 6 Extremely severe hallucinations 7 Continuous hallucinations
PAROXYSMAL SWEATS Observation: 0 No sweat visible 1 Barely perceptible sweating, palms moist 2 3 4 Beads of sweat obvious on forehead 5 6 7 Drenching sweats	**VISUAL DISTURBANCES –** Ask “Does the light appear to be too bright? Is its color different? Does it hurt your eyes? Are you seeing anything that is disturbing to you? Are you seeing things you know are not there?” Observation: 0 Not present 1 Very mild sensitivity 2 Mild sensitivity 3 Moderate sensitivity 4 Moderately severe hallucinations 5 Severe hallucinations 6 Extremely severe hallucinations 7 Continuous hallucinations
ANXIETY – Ask “Do you feel nervous?” Observation: 0 No anxiety, at ease 1 Mild anxious 2 3 4 Moderately anxious, or guarded, so anxiety is inferred 5 6 7 Equivalent to acute panic states as seen in severe delirium or acute schizophrenic reactions	**HEADACHE, FULLNESS IN HEAD –** Ask “Does your head feel different? Does it feel like there is a band around your head?” Do not rate for dizziness or lightheadedness. Otherwise, rate severity. 0 Not present 1 Very mild 2 Mild 3 Moderate 4 Moderately severe 5 Severe 6 Very severe 7 Extremely severe
AGITATION Observation: 0 Normal activity 1 Somewhat more than normal activity 2 3 4 Moderately fidgety and restless 5 6 7 Paces back and forth during most of the interview, or constantly thrashes about	**ORIENTATION AND CLOUDING OF SENSORIUM –** Ask “What day is this? Where are you? Who am I?” 0 Oriented and can do serial additions 1 Cannot do serial additions or is uncertain about date 2 Disoriented for date by no more than 2 calendar days 3 Disoriented for date by more than 2 calendar days 4 Disoriented for place or person
	Total CIWA-Ar Score______ Rater’s Initials______ Maximum Possible Score 67

The CIWA-Ar is not copyrighted and may be reproduced freely. This assessment for monitoring withdrawal symptoms requires approximately 5 minutes to administer. The maximum score is 67 (see instrument). Patients scoring less than 10 do not usually need additional medication for withdrawal.

From Sullivan JT, Sykora K, Schneiderman J, et al. Assessment of alcohol withdrawal: The revised Clinical Institute Withdrawal Assessment for Alcohol scale (CIWA-Ar). *British Journal of Addiction* 1989;84:1353–7. Reproduced with permission.

ethanol is a GABA agonist, alcohol withdrawal leads to CNS excitation. Treatment is therefore focused on administration of GABA agonists to reopen chloride channels and diminish CNS excitation. Mild alcohol withdrawal can often be treated as an outpatient after initial stabilization in the ED, especially if the patient has good support systems in place. Extensive diagnostic testing may not be necessary in patients suffering from mild alcohol withdrawal symptoms.

The most frequently used agents to control alcohol withdrawal are benzodiazepines, especially diazepam and lorazepam. Alcohol withdrawal peaks in 2–3 days and can last 5–7 days; therefore, longer-acting agents are generally preferred to prevent frequent re-dosing. In addition, long-acting agents wear off slowly, leading to a smoother and more predictable experience for the patient. This allows patients to take subsequent doses of medication before significant withdrawal symptoms recur. Parenteral benzodiazepines (like diazepam and lorazepam) are generally used to treat moderate to severe alcohol withdrawal in the ED, especially if the patient is vomiting or not willing to take oral medication. The half-life of diazepam is 30–50 hours and active metabolites have a half-life of up to 100 hours, which makes it a much better choice than lorazepam, with a half-life of only 10–20 hours. However, it is best to use an oral agent in the ED, as this is what the patient will be released with. Oral agents also provide less euphoria and have a wider margin of safety. When prescribing oral benzodiazepines, chlordiazepoxide is a common choice. The half-life of chlordiazepoxide is 5–30 hours, but active metabolites have a half-life of up to 200 hours. Oxazepam is also commonly used, with a half-life of 4–15 hours. These (and other) benzodiazepines should be used with caution in elderly patients.

Severe alcohol withdrawal includes significant vital sign changes, CNS excitation and seizures. Medical therapy is similar to mild alcohol withdrawal with one key exception — the seizure should be treated. Most experts feel that lorazepam is a better parenteral agent than diazepam to stop an actively seizing patient. The rationale for this recommendation is that lorazepam is less lipophilic, remains in the vascular space longer, and therefore has longer peak anticonvulsant effects. In contrast, intravenous diazepam is rapidly absorbed into lipophilic tissue, thereby allowing seizures to recur as the therapeutic plasma concentration decreases. Occasionally, chronic alcoholics who are familiar with the medical system feign seizures to get a parenteral dose of a benzodiazepine.

Delirium tremens (DT) is an uncommon but life-threatening condition characterized by severe alteration of mental status and abnormal vital signs. Mortality is usually due to decompensation from adrenergic instability. Treatment of DT requires aggressive GABA-based receptor treatment (GABAergic therapy), volume replacement, and correction of any electrolyte abnormalities. Despite aggressive therapy and modern intensive care unit (ICU) care, the mortality rate of DT remains approximately 20–30%. These patients require large doses of benzodiazepines and frequently continuous benzodiazepine infusions. The side effects of continuous infusions of benzodiazepines include respiratory depression and prolonged sedation, even after discontinuation of the infusion (due to delayed release from fat stores). The addition of barbiturates, such as phenobarbital, can provide a sustained baseline level of GABA-agonism and decrease the amount of benzodiazepines required for therapy. In severe cases requiring intubation, propofol is a strong GABA agonist and may be more effective than benzodiazepines and barbiturates.

Other medications that may have a therapeutic role in alcohol withdrawal include haloperidol, gabapentin and clonidine. Haloperidol is an effective adjunctive medication to help control the hallucinations and delirium in alcohol withdrawal and DT. Haloperidol has no effect on GABA receptors and therefore does not help the underlying withdrawal process. Gabapentin, a structural analogue of GABA, has recently shown promise in reducing the symptoms of alcohol withdrawal and alcohol craving. Despite extensive studies, the mechanism of action of gabapentin remains poorly understood. Clonidine may also have some utility in limiting central adrenergic discharge and CNS stimulation, but evidence is limited.

Phenobarbital is an underutilized medication for the treatment of alcohol withdrawal. When used as a second-line agent after benzodiazepines, phenobarbital can further reduce the signs and symptoms of withdrawal. Some clinicians also use it for mild alcohol withdrawal, as its long half-life (80–120 hours) makes it desirable for outpatient treatment. A single intravenous dose in the ED, typically between 130 and 260 mg, followed by repeated doses of 65–130 mg as necessary to control tremulousness and withdrawal symptoms, will remain in the patient's system for as long as the patient is at risk for alcohol withdrawal symptoms. Phenobarbital is metabolized slowly; therefore, it can function as a self-tapering agent. It is important to understand that there are currently no universally accepted dosing guidelines for phenobarbital. The ultimate dose of this (and other agents) required by a patient depends on the patient's tolerance to barbiturates and GABA-agonism.

Disposition

Patients with uncomplicated acute alcohol intoxication should be observed until they are clinically sober. Clinically "sober" implies that the patient has returned to their baseline mental status; ambulates safely; can eat and drink; has a safe place to return; is not suicidal, homicidal or gravely disabled; and can care for themselves. Clinical examination, rather than a specific serum ethanol level, best evaluates a patient's sobriety. Chronic alcoholics with acute intoxication should be carefully observed because they may develop withdrawal symptoms requiring treatment. Patients who are intoxicated and lack the capacity for reasonable decision making should not be allowed to leave the ED against medical advice. Whenever possible, family members should be involved in clinical care decisions. Mental health, risk management and law enforcement personnel should be consulted if necessary.

If a patient has an alcohol-related condition, such as gastrointestinal bleeding, pancreatitis, hepatitis, or traumatic injury, disposition is usually determined by that particular condition. Intoxicated patients should always be reassessed once deemed clinically sober to exclude alcohol-related conditions that might have been missed on initial assessment. Chronic alcoholics often have a poor social situation and limited ability to return for reevaluation, which should be considered when making a disposition decision, including follow-up care.

The disposition of patients with alcohol withdrawal, whether mild or severe, should be based on their response to treatment and risk of subsequent respiratory depression (based on the amount of benzodiazepines or barbiturates they received). Patients with mild alcohol withdrawal are often treated in the ED and discharged home, with an outpatient regimen of oral benzodiazepines, close follow-up instructions, and prearranged follow-up care. The amount of medication a particular patient requires depends on many variables. One approach is to titrate medical therapy to effect (symptom response) rather than a specific amount of medication. Patients with prolonged somnolence after medical therapy require admission. In contrast, patients whose symptoms are controlled, are awake, alert, ambulate safely, are able to eat and drink, and can understand instructions can be managed as outpatients. Upon discharge, it is important to warn the patient about refraining from alcohol consumption since the combination of sedating medications and alcohol can lead to respiratory depression.

DT, the most severe sequela of alcohol withdrawal, requires aggressive treatment and ICU admission. These patients have a high mortality rate even with aggressive therapy. Intensivists should be consulted early in the ED course. Under-recognition of DT is a common reason for delayed treatment. As a general rule, alcohol withdrawal syndromes do not cause altered mental status. Even a subtle change in mental status is a hallmark of more severe alcohol withdrawal syndrome. Early, aggressive therapy can prevent further clinical decompensation. High-dose sedatives are required to treat the hyperadrenergic state. Response to therapy in the ED is measured by improvement in vital signs. In order to reach the requisite doses of sedative needed for treatment of DT, patients frequently require mechanical ventilation for ventilatory support.

Many patients being treated for alcohol-related illnesses or withdrawal need restraints, either chemical, physical, or both, to prevent further injuries to themselves or the ED staff. Great care must be taken to encourage those not restrained to remain in the ED to receive a comprehensive evaluation and appropriate treatment and referral.

Special patients

Pregnant

Women who are pregnant should not drink excessive amounts of alcohol, and should refrain from ingesting any toxic alcohols. Fetal alcohol syndrome, fetal malformations, folate depletion, miscarriage due to bleeding disorders, and increased likelihood of falls with abdominal trauma are concerns. Women who are known to be pregnant or newly diagnosed in the ED should be counseled regarding potential ill effects of alcohol abuse during pregnancy. Because depression and intimate partner violence are common during pregnancy, women who are pregnant should be screened for these conditions and referred appropriately, if necessary.

Children

Infants and toddlers are at increased risk of profound hypoglycemia and death due to inadequate glycogen stores. Therefore, parents need to be counseled to keep alcohol away from their infants and toddlers. Older children may experiment with alcohol to mimic their parents or older siblings, to mimic television or movie actors, to get “buzzed,” or due to peer pressure.

Young adults

Peer pressure at school, parties, or at home may result in young adults drinking alcohol. As young adults may not have the appropriate ability to control their intake or understand the relationship between exposure, time course of ingestion, and food, they may “miscalculate” how much alcohol they ingest and its effects. Furthermore, there are numerous drinking “games” that result in high serum alcohol levels, coma and death. Drinking 21 shots of alcohol in celebration of turning 21 years of age has resulted in numerous fatalities from acute alcohol intoxication. Driving is impaired after even one drink, especially in inexperienced drivers, resulting in traffic injuries and fatalities. Disinhibition from alcohol ingestion and intoxication has resulted in numerous injuries and deaths from risky behaviors, suicides and violence.

Psychiatric

Patients presenting with abnormal behavior (see Chapter 11), with or without previous psychiatric illness, often will ingest alcohols (including toxic alcohols). Patients may do this in an attempt to kill or harm themselves. Patients who require psychiatric hospitalization for suicidal behavior or grave disability (prior to intoxication) may be challenging to admit on a psychiatric unit because of medical conditions that might develop (seizures, withdrawal, delirium tremens, hypoglycemia). A particularly challenging patient may claim to be suicidal while intoxicated, but may deny these statements and threats once sober. This is dangerous if emergency physicians overlook the impact of this statement. The patient is likely to become intoxicated again, may not have the desire, opportunity, or funds to seek follow-up therapy, and may make an attempt at taking their life during their next contact with alcohol. It is therefore prudent practice to have a mental health professional, which may include someone from an alcohol and chemical dependency program, speak with this patient once sober if they are not immediately placed on an involuntary psychiatric hold based on their initial statement or actions.

Taking disulfiram

Patients taking disulfiram to treat alcoholism who are exposed to ethanol may experience flushing, tachycardia, warmth, urticaria, pruritus, lightheadedness, headache, nausea, vomiting, palpitations, chest pain and dyspnea. Certain medications, such as some cephalosporins, metronidazole, trimethoprim-sulfamethoxazole, nitrofurantoin, griseofulvin, chlorpropamide and tolbutamide, may result in patients experiencing disulfiram-like reactions following ethanol exposure. A similar reaction may also occur with exposures to *Coprinus* mushroom species and chemicals carbon tetrachloride or trichloroethylene. This reaction itself is not life-threatening.

Pearls, pitfalls and myths

- Always adhere to basic emergency care principles in a patient with alcohol-related illness or emergencies, including the ABCs (airway, breathing, circulation).
- Patients with acute alcohol intoxication require a thorough evaluation to prevent missed illness and injury. Do not frame the patient's complaint around only alcohol, nor prematurely reach a diagnosis related to the known history of alcohol abuse.
- Observation (often prolonged) of intoxicated patients is required until they are clinically sober. Progress notes should document a patient's status during this observation period while sobering, in addition to their condition at discharge.
- A serum blood ethanol level has limited utility in ED care, unless it is unexpectedly low in a patient with altered mental status who appears "intoxicated," as this should result in the aggressive search for an alternative explanation.
- Treatment of alcohol withdrawal should focus on symptomatic improvement of the patient's symptoms and vital signs, not the absolute amount of medications.
- Benzodiazepines and phenobarbital are the mainstay of therapy in the treatment of acute alcohol withdrawal.
- Delirium tremens is a life-threatening condition that can be subtle in onset. Early and aggressive treatment, including admission to an ICU, helps reduce mortality.
- It is important not to overlook the metabolic and nutritional conditions that can result from chronic alcohol abuse.
- Hemodialysis is emergent treatment for methanol, ethylene glycol and isopropanol, and should be initiated rapidly if patients are hemodynamically unstable, ingested amounts or serum concentrations are known to be high, specific physical examination or laboratory findings exist, or clinical improvement to aggressive therapy is not occurring.

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13 Allergic reactions and anaphylactic syndromes

Steven Go, MD

Scope of the problem

In the emergency department (ED), health care professionals sometimes attempt to reassure patients by comparing the pain of minor procedures to "a little bee sting." However, the estimated prevalence of acute anaphylactic reactions to insect stings is as high as 5% of the US population, resulting in about 100 deaths annually. In the broader scheme, anaphylaxis from any cause has been estimated to occur at rates from 7.9–9.6 per 100,000 patients, with about 1,500 deaths per year in the US. The symptoms of allergic reactions occur on a spectrum – from mild cases of pruritus to cardiovascular collapse – and it is not always easy to predict when the former will evolve quickly into the latter. The failure to rapidly diagnose and treat these conditions often results in untoward outcomes. Therefore, it is imperative that emergency physicians have a solid understanding of the diagnosis and management of allergic reactions and anaphylaxis.

Anatomic essentials

The term *anaphylaxis* comes from the Greek words for "against" and "protection." In 2006, Sampson et al. more formally defined anaphylaxis as an acute illness after exposure to an antigen involving skin and/or mucosa with either some degree of respiratory compromise and/or reduced blood pressure or symptoms of end-organ dysfunction.

The mechanism begins when the body produces immunoglobulin E (IgE) during initial exposure to an antigen. On subsequent exposure, IgE binds to mast cells, causing release of vasoactive products. These products, histamine being chief among them, lead to smooth muscle spasm, bronchospasm, mucosal edema, angioedema, and increased capillary permeability. Such reactions are generally immediate; however, it has been suggested that mast cells or basophils can also release new mediators in a delayed fashion, which results in a second phase of symptoms.

Anaphylactoid reactions are syndromes that present as anaphylaxis, but not through an IgE-mediated mechanism. In addition, they often do not require a prior exposure to the antigen. From a practical perspective, however, anaphylaxis and anaphylactoid reactions are often clinically indistinguishable, and will therefore be addressed together as ***anaphylactic syndromes***.

Inciting causes of anaphylactic syndromes are legion, including but not limited to insect bites and stings, food exposure, medications (especially by parenteral administration), latex exposure, exercise (with or without concurrent food exposure), seminal fluid and idiopathic factors.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 13.1).

History

Although history is important in confirming both the diagnosis and etiology of acute allergic syndromes, it is vital to remember that the length of the history must be proportional to the stability of the patient.

Do you have trouble breathing or talking?

As always, airway management must take top priority. In allergic syndromes, airways can be compromised by angioedema, and the sometimes brief window of opportunity for securing the airway may close rapidly. If impending airway collapse is not quickly recognized by the emergency physician, a bad outcome is almost certain to follow. An affirmative nod to this question requires immediate transfer to a monitored area of the ED, where airway emergencies can be best handled.

When did the symptoms start and how long have they been going on?

The symptoms of anaphylaxis typically start within seconds to minutes of exposure to the offending antigen; however, they may start as late as 24 hours after exposure. In general, the sooner symptoms appear after exposure, the more severe the clinical course. A *biphasic response*, where severe symptoms recur up to 72 hours (mean 6–10 hours) after the initial symptoms resolve, occurs in about 20% of treated patients. Persistent anaphylactic reactions consist of continual symptoms for 5–32 hours despite medical therapy.

Do you have any known allergies? Any new exposures? Has this happened before?

Identification of the inciting antigen is not always possible, but should be attempted in order to discontinue exposure to that antigen (e.g., new make-up, perfume, topical medication). Previous incidents and known allergies may provide a clue to the etiology of the current attack or point to specific cross-reactivities that may exist (i.e., penicillins and cephalosporins). Frequent previous incidents may identify carcinoid syndrome, hereditary angioedema, or factitious anaphylaxis.

Table 13.1 Allergic reactions and anaphylactic syndromes red flags

History	Concerning diagnosis
Previous admission or emergency airway intervention, epinephrine requirement, rapidly progressive course, GI symptoms	Anaphylactic shock, airway compromise, prolonged or severe disease course
Difficulty speaking, dysphonia, dysphagia, throat tightness, oropharyngeal/facial swelling, shortness of air, anxiety/confusion	Airway compromise, angioedema, respiratory failure
Syncope, near-syncope, dizziness, orthostatic symptoms, anxiety/confusion	Anaphylactic shock
Recent contrast during imaging study	Anaphylactic syndrome from contrast allergy
Chest pain, shortness of breath (esp. elderly), palpitations	Cardiac ischemia (from hypoxia, stress, or epinephrine), dysrhythmia
Family history of similar symptoms or history of similar episodes with no antigen	Hereditary angioedema, carcinoid syndrome
Abdominal pain in pregnant woman	Miscarriage caused by anaphylactic syndrome
Examination finding	**Concerning diagnosis**
Oropharyngeal/facial swelling, muffled voice or hoarseness, stridor, tachypnea, air hunger, increased work of breathing, hypoxia (late sign)	Airway involvement, angioedema
Wheezing, diminished breath sounds, cyanosis, hypoxia (late sign), altered mental status	Respiratory failure
Hypotension, tachycardia, thready pulse, diffuse erythema/flushing, altered mental status	Anaphylactic shock
Irregular pulse	Dysrhythmia (from hypoxia, stress, or epinephrine)
Mucous membrane involvement	Angioedema, Stevens-Johnson's syndrome

What were the surrounding events when the symptoms occurred?

If the symptoms occurred in conjunction with the introduction of emotional stress, a vasovagal reaction may be suspected. If the symptoms began during or shortly after a meal, a potential food antigen is possible. Because restaurants do not generally disclose the precise ingredients in their dishes, many patients may not realize they have consumed foods that they know cause them problems. Anaphylaxis can occur in conjunction with vigorous exercise, especially in conditioned athletes in adverse climates.

Has anyone in your family had symptoms like this before?

If affirmative, hereditary angioedema should be suspected. Many antigens and exposures cause difficulty for an entire family.

Associated symptoms

Anaphylactic syndromes can present in various ways (Table 13.2). Increased vascular permeability can result in urticaria and angioedema, and is sometimes preceded by a feeling of flushing and warmth. Laryngeal edema can quickly lead to airway compromise and may present with stridor, hoarseness, a feeling of airway obstruction and dysphagia. Nasal congestion can further hamper respirations. Bronchospasm presents with dyspnea, wheezing and "tightness" in the chest. Hypotension can present with syncope or dizziness, which are sometimes harbingers of vascular collapse. Other associated symptoms include gastrointestinal (GI) symptoms, such as nausea, vomiting, abdominal pain and diarrhea, which may sometimes be bloody. Signs and symptoms of shock may be present in severe cases. Uterine muscle contractions can cause pelvic cramping and miscarriage in pregnancy. In anaphylaxis, any of these symptoms can be present, either together or in isolation. Skin findings are present in up to 90% of cases. However, the absence of skin signs should not be falsely reassuring.

Past medical

Patients with a history of cardiac or pulmonary disease are at greater risk of death. Patients taking beta-blockers who develop anaphylaxis are often refractory to therapy and are at extremely high risk.

Physical examination

General appearance

The general appearance of the patient is of crucial importance. Patients experiencing allergic reactions who appear sick are probably ill or about to be very ill. Any difficulty speaking, respiratory distress or agitation should provoke immediate treatment. An expressed fear of impending doom is often eerily prescient.

Table 13.2 Symptoms and signs of anaphylactic syndromes

Presentation	Symptoms	Signs
Airway edema	Sensation of throat tightness, dysphagia, dysphonia	Respiratory distress, stridor, muffled voice or hoarseness, coughing, sneezing, nasal congestion
Angioedema	Swelling without pruritus	Edema, especially of face, eyelids, lips, tongue, uvula, eyes, hands and feet
Bronchospasm	Dyspnea, chest tightness	Wheezing, coughing, retractions, tachypnea
Distributive shock	Dizziness, syncope, near-syncope, anxiety, weakness, confusion	Hypotension, tachycardia
Gastroenteritis	Nausea, vomiting, diarrhea, bloating, abdominal cramping	Diffuse abdominal pain without peritoneal signs; may have normal examination
Increased secretions	Rhinorrhea, bronchorrhea, increased lacrimation	Nasal congestion, increased tracheal and bronchial secretions, drooling, tearing, conjunctival erythema
Urticaria	Pruritus or tingling, rash or swelling, flushing	Raised erythematous welts of various sizes on the skin surface; usually pruritic

Vital signs

Temperature is usually normal. Cardiovascular involvement is suggested by hypotension, tachycardia, or dysrhythmias. Hypotension with a relative bradycardia can be associated with insect sting anaphylaxis. Pulse oximetry is typically normal until airway compromise is nearly complete; therefore, a normal reading does not rule out airway involvement.

Integument

Inspection may reveal urticaria (Figure 13.1), angioedema, erythema, flushing and pruritus. Diaphoresis and/or cyanosis indicates the presence of shock.

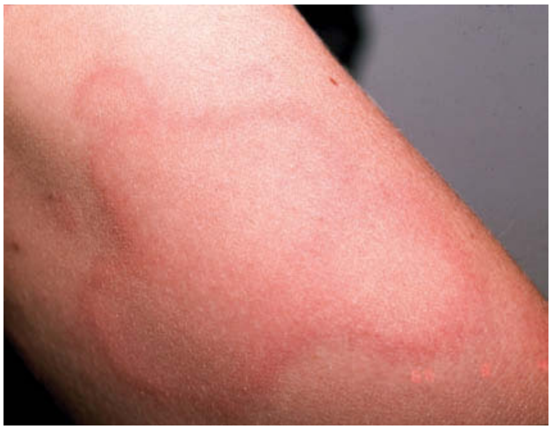

Figure 13.1
Urticaria. Courtesy: Steven Shpall, MD.

Head and neck

Inspection may reveal swelling of the eyelids, lips (Figure 13.2), tongue or oral mucosa. Lip or facial cyanosis indicates severe respiratory compromise. Drooling, the inability to manage secretions, and the size and appearance of the uvula and tongue should all be noted. The posterior oropharynx should be inspected for patency. A hoarse or muffled voice signals potential airway compromise, as does dysphagia. Stridor should be identified. Eye itching, conjunctival injection and tearing can occur. Nasal congestion, rhinorrhea and sneezing may also be present. Observing the patient's Mallampati classification (Figure 2.8) may be useful in helping determine what type of airway stabilization method is appropriate if acute airway compromise occurs, but its role in the management of anaphylaxis has not been clearly delineated in the literature.

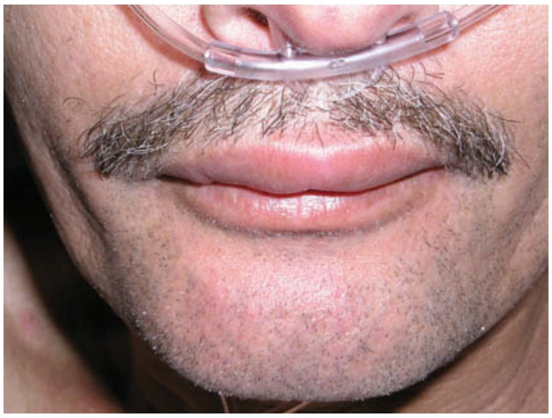

Figure 13.2
Angioedema involving the upper lip. Courtesy: Leland Robinson, MD and Steven Go, MD.

Pulmonary

Wheezing indicates bronchospasm if enough airflow is present to wheeze. A quiet chest is an even more dangerous sign because it indicates severe compromise of the patient's ventilatory status. Increased respiratory effort is also dangerous.

Cardiac

Tachycardia is most common, but other dysrhythmias may be present.

Abdomen

Crampy abdominal pain as a result of edema, smooth muscle contraction or vascular engorgement can be present. However, true peritoneal signs should not be present. Tenesmus can also occur.

Extremities

Patients with anaphylaxis commonly have a rapid, weak, thready pulse. Cyanosis of the nail beds occurs with severe respiratory compromise.

Neurologic

Altered mental status, agitation, lightheadedness or unconsciousness are signs of a severe reaction. Seizures are uncommon, but may occur. Otherwise, the neurologic examination should be normal.

Differential diagnosis

There are numerous entities that can mimic anaphylaxis. It can be very difficult to differentiate them in the acute phase. Therefore, clinical syndromes that appear to be anaphylaxis should be treated as anaphylaxis until proven otherwise (Table 13.3).

Diagnostic testing

Diagnostic testing in the ED is of little utility in the emergent diagnosis and management of anaphylactic syndromes. Confirmatory skin testing is beyond the scope of emergency medicine.

Laboratory studies

Serum histamine, tyramine, mast cell tryptase, and specific IgE levels have been mentioned in the literature to possibly confirm the diagnosis of anaphylaxis. However, histamine has an extremely short half-life; therefore, a meaningful level is difficult to measure. More importantly, these tests are more appropriate to confirm the diagnosis after the patient has been stabilized. They play no role in determining whether to suspect anaphylaxis or to treat it.

Electrocardiogram and radiologic studies

Electrocardiogram (ECG) and radiologic studies are generally nonspecific. However, case reports exist that describe concurrent acute coronary syndromes (ACS) with resultant characteristic ECG changes. A definite causative relationship between anaphylaxis and ACS has yet to be found.

General treatment principles

The guiding treatment principle is to rapidly determine that the patient needs treatment. Anaphylaxis often occurs without warning, and a delay in appropriate therapy may prove fatal. A high level of suspicion must be maintained. In addition, for obvious reasons, there are few prospective controlled trials for the treatment of anaphylactic shock. Therefore, it should be remembered that treatment recommendations in the literature are largely based on anecdotal clinical experience.

Although the following treatment strategies should occur simultaneously, it is helpful to conceptualize them in a few basic categories.

Antigen removal

If the inciting antigen is still present (e.g., the stinger of a bee, article of clothing), it should be removed promptly.

Epinephrine administration

Epinephrine administration is the cornerstone of treatment. Moreover, the failure to administer epinephrine quickly has been implicated as a key factor leading to anaphylactic death. ***Therefore, epinephrine should be given liberally whenever an anaphylactic syndrome is suspected***. The usual dose of epinephrine is 0.3–0.5 mg of 1:1,000 solution given intramuscularly (IM). Data have shown that epinephrine absorption is superior with the IM route, especially when it is injected in the anterior-lateral thigh. Some experts have recommended intravenous (IV) epinephrine administration, but given the potential hazards of this route (e.g., fatal dysrhythmias, myocardial infarction, cerebral vascular events, organ ischemia) and the lack of conclusive advantages, it is probably prudent to avoid the IV route except in cases of cardiopulmonary extremis refractory to IM epinephrine. Epinephrine should be used with care in those with known cardiac disease, pregnancy, or in patients taking beta-blockers or other drugs that have potential adverse drug interactions (see Special patients). However, it cannot be overemphasized that ***in anaphylactic shock, there are no absolute contraindications to epinephrine***.

Airway control

The most common mistake in airway management is the failure to recognize the need for early airway control. For any patient with an allergic reaction, the status of the airway must be determined, documented and monitored closely. An oral airway is preferable to a surgical airway, if possible. If laryngeal edema is present, early elective airway control

Table 13.3 Differential diagnosis of anaphylaxis-like syndromes

Diagnosis	Symptoms	Signs	Work-up
Carcinoid syndrome	Recurrent episodes of flushing of the face and neck, palpitations, facial swelling, GI symptoms (especially diarrhea, which can be debilitating). Dyspnea may also occur.	Hypotension, no urticaria. Facial edema, malar telangiectasia, flushing, wheezing. May hear murmur if cardiac involvement.	Increased serum and urine levels of serotonin metabolite, 5-HIAA.
Chinese restaurant syndrome (MSG symptom complex)	History of eating MSG-containing foods. Dyspnea, flushing, sweating, tightness in the chest, burning sensation at the back of the neck into arms and chest, headache, nausea, palpitations, oral numbness and burning.	Wheezing, flushing, hypotension, and dysrhythmias can occur. True anaphylaxis may occur.	History. No definitive test. Symptoms typically resolve in 2 or 3 hours.
Factitious anaphylaxis	Anxiety present.	No objective signs of anaphylaxis.	History and examination. Diagnosis of exclusion. Munchausen's anaphylaxis is true anaphylaxis that the patient causes surreptitiously.
Hereditary angioedema	Swelling of lips, tongue and upper airway with possible respiratory compromise. Sometimes abdominal pain or non-pruritic swelling of extremities. Often develops after trauma (e.g., dental procedure). Lack of antigen exposure. Family history of these events and/or history of recurrent episodes in the absence of antigen.	Angioedema is usually seen in the lips, face and oral mucosa. Absence of urticaria or pruritus.	Decreased C1-esterase inhibitor levels. Decreased serum C4. Fiberoptic laryngoscopy may reveal upper airway edema.
Pheochromocytoma	Headache, sweating, palpitations, tremor, nausea, weakness, constipation, abdominal pain, weight loss.	Hypertension, fever, weight loss, pallor, tremor, neurofibromas, café au lait spots, tachydysrhythmias.	Elevated levels of urine catecholamines. Hyperglycemia, hypercalcemia, erythrocytosis.
Scombroid poisoning	Exposure to fish of the Scombridae family or related fish (tuna, mackerel, mahi-mahi, sardines, anchovies). Rapid onset of facial flushing, peppery taste, dizziness, palpitations, nausea, headache, diarrhea, abdominal pain.	Diaphoresis, facial rash, urticaria, edema, abdominal tenderness. Respiratory distress, tongue swelling, blurred vision and vasodilatory shock may occur.	Elevated level of urine histamine. FDA analysis of tainted fish. Typical resolution of symptoms within 8–10 hours.
Serum sickness	Fever, malaise, headache, arthralgias, GI symptoms, associated with urticaria occur 7–10 days after exposure to antigens.	Fever, rash (may be scarlatiniform, urticarial, morbilliform, or polymorphous) lymphadenopathy, arthritis, arthralgias. Rarely cardiopulmonary involvement.	Elevated sedimentation rate. Possible elevated creatinine. CBC with eosinophilia. Depressed complement levels.
Systemic mastocytosis	Not associated with a particular antigen exposure.	Presents as anaphylaxis.	No available test to differentiate from anaphylaxis.
Vasovagal reactions	Occurs during stress (e.g., injection, dental procedures). No pruritus. Absence of respiratory obstruction or skin symptoms.	Slow, strong, steady pulse. Blood pressure normal or elevated. Skin cool. Pallor without cyanosis.	Monitoring and ED observation. Symptoms relieved by recumbency.
MCSLC	See specific disorder	See specific disorder	See specific disorder

CBC: complete blood count; ED: emergency department; FDA: Food and Drug Administration; GI: gastrointestinal; 5-HIAA: 5-hydroxyindoleacetic acid; MCSLC: miscellaneous causes of sudden loss of consciousness (i.e., seizure, cardiac dysrhythmias, pulmonary embolism, foreign-body aspiration); MSG: monosodium glutamate.

Table 13.4 Anaphylactic syndrome drug dosages

Drug	Adult dose	Pediatric dose
Parenteral adrenergic agents		
Epinephrine	0.3–0.5 mg 1:1,000 solution IM Q 15 min 0.1 mg 1:10,000 solution slow IV push	0.01 mg/kg (minimum 0.1 mL) 1:1,000 solution IM Q 15 min 1 mcg/kg (minimum 0.1 mL) 1: 10,000 solution slow IV push
Epinephrine (intravenous) infusion	1–10 mcg/min titrate to effect	0.1–1 mcg/kg/min titrate to effect
Inhaled β-agonists		
Albuterol	0.5 mL 0.5% solution in 2.5 mL NS via nebulizer Q 15 min	0.03–0.05 mL/kg 0.5% solution in 2.5 mL NS via nebulizer Q 15 min
H_1-receptor antagonists		
Diphenhydramine (Benadryl)	25–50 mg IV/IM Q 4–6 hrs 50 mg PO Q 4–6 hrs	1–2 mg/kg IV/IM Q 4–6 hrs 2 mg/kg PO Q 4–6 hrs
H_2-receptor antagonists		
Ranitidine (Zantac)	50 mg IV over 5 min 150 mg PO BID	0.5 mg/kg IV over 5 min 0.25–2 mg/kg/dose PO Q 12 hrs (maximum 150 mg Q 12 hrs)
Cimetidine (Tagamet)	300 mg PO/IV/IM Q 6 hrs	Not recommended for children
Corticosteroids		
Methylprednisolone (Solu-Medrol)	40–250 mg IV/IM Q 6 hrs	1–2 mg/kg IV/IM Q 6 hrs
Prednisone	20–60 mg PO QD	1 mg/kg PO QD
Antidote, refractory hypotension		
Glucagon	1 mg IV Q 6 min until hypotension resolves, followed by 5–15 mcg/min infusion	Dosing not definitively established

BID: two times a day; IM: intramuscular; IV: intravenous; NS: normal saline; PO: per os; QD: once daily.

is preferable to expectant management. By the time extreme respiratory distress develops, achieving an airway may be impossible. Rapid sequence intubation (RSI) should be used with great caution in these patients, as unseen lower airway edema may preclude an oral endotracheal airway. In such cases, giving paralytics would be unwise. If *immediately available*, fiberoptic intubation may be a safer option. In any event, equipment and personnel necessary to establish an emergent surgical rescue airway should ideally be close at hand when managing the airway.

Ventilatory support

Any component of bronchospasm should be treated with beta-agonist bronchodilators, supplemental oxygen and corticosteroids. Arterial blood gases may be useful in determining the level of ventilatory compromise, although the decision to intubate for ventilatory compromise remains largely a clinical one.

Circulatory support

Fluid resuscitation with crystalloid should be given for hypotension and other signs of shock (as colloid has been associated with allergic reactions). Large quantities of crystalloid may be required to maintain a satisfactory blood pressure. Central venous pressure monitoring may be helpful in guiding therapy. For refractory cases, vasopressors such as norepinephrine may be required. The patient should be kept recumbent (or in the Trendelenburg position) until the blood pressure stabilizes, as the seated or upright posture has been associated with poor outcomes. Orthostatic blood pressure measurements have no role in the evaluation of anaphylactic syndromes.

Secondary medications

Antihistamines can be useful in treating cutaneous manifestations of allergic reactions, but their utility in acute anaphylaxis is unclear because robust evidence of their efficacy is lacking. Therefore, they should be viewed as adjunctive treatments to epinephrine and fluids in this circumstance. Some studies have shown that in acute allergic urticaria, the addition of H_2-antagonists to H_1-antagonists resulted in improved outcomes (e.g., resolution of urticaria) in patients compared to those treated with H_1-blockade alone. However, more well-controlled clinical trials concerning the role of H_2-antagonists in anaphylactic syndromes are needed before definitive recommendations can be given.

Corticosteroids likely have no benefit in the acute phase of anaphylaxis, given their delayed onset of action. They *may* reduce the possibility of a biphasic reaction; however, published data demonstrating this are lacking. Nevertheless, many expert treatment guidelines recommend that corticosteroids be administered in anaphylactic syndromes. Therefore, they should probably be given early to all patients unless strong contraindications exist.

Beta-agonist bronchodilators are frequently used when wheezing is present. Although wheezing may improve with beta-agonists, they do not treat the underlying mechanism of the anaphylaxis syndrome, and such improvement does not obviate the need for epinephrine.

If anaphylactic shock proves refractory to IV epinephrine, preliminary evidence suggests that alternative vasopressors such as metaraminol, methoxamine, or vasopressin may be of some value. However, because the evidence is limited to case reports, optimal dosing has not been determined.

Norepinephrine and glucagon may also be useful in refractory hypotension. Glucagon may be particularly useful in hypotensive patients taking beta-blockers, but definitive supportive data for its use are lacking.

Recently, there has been much interest in studying the potential role of heparin (which acts as a histamine binder and mast cell mediator inhibitor) in anaphylaxis. Published data at this time include a pilot study in which heparin demonstrated the ability to reverse induced anaphylactic shock in a porcine model, as well as several case reports in humans. Research trials for this use of heparin are ongoing; therefore, its routine use in anaphylaxis is premature.

Special patients

Potential drug interactions

Beta-blockers are proallergenic, and also amplify the production of anaphylactic mediators that potentiate the severity of allergic reactions. Beta-blockers may also blunt the usually favorable response to epinephrine treatment. A glucagon infusion may be useful in treating hypotension in anaphylaxis patients who are taking beta-blockers. In addition, these patients may develop severe hypertension upon epinephrine administration secondary to unopposed alpha-adrenergic effects. Dysrhythmias may also occur.

Adverse reactions may also occur during epinephrine therapy in patients who are using tricyclic antidepressants, monoamine oxidase inhibitors, antipsychotics, clonidine, dopaminergics and ergotamines. Epinephrine should be used at reduced dosages in these cases, and phentolamine (to treat hypertension) and antidysrhythmic agents should be readily available.

Resistant bronchospasm

Resistant bronchospasm may occur in patients who are taking beta-blockers. Sometimes higher than usual dosages or frequency of bronchodilators (beta-agonists and anticholinergics) are necessary for these patients. Inhaled epinephrine may be useful when IM epinephrine fails to relieve bronchospasm. Other therapies mentioned in the literature include IV magnesium, vitamin C, naloxone, atrial natriuretic factor and glucagon; however, evidence of benefit for these medications is inconclusive.

Disposition

Much like treatment, disposition recommendations in the literature are generally based on clinical experience, with many experts recommending an extended period (4–8 hours) as the *minimum* time a patient with a significant anaphylactic syndrome should be closely observed.

Patients with mild allergic reactions limited to peripheral cutaneous findings (not involving the airway) and without evidence of anaphylaxis may be treated symptomatically and discharged with careful follow-up instructions, including avoidance of the inciting antigen.

Patients with more severe reactions (e.g., mucosal swelling, wheezing) but without evidence of shock should be treated aggressively and observed for at least 8 hours. If the patient makes a prompt recovery without complications and remains asymptomatic, he may be safely discharged with cautionary discharge instructions, scheduled corticosteroids to prevent a late-phase reaction, histamine receptor antagonists for symptomatic treatment, and close follow-up. In the absence of contraindications, patients should also be given a prescription for an epinephrine injector with verbal and written instructions on how to use it. These patients should be referred to an allergist for determination of the inciting allergen and for possible desensitization therapy.

The subset of the above patients with significant preexisting comorbidities (e.g., advanced age, cardiopulmonary disease) should probably be admitted for observation. In addition, most experts suggest admitting any patient who requires multiple doses of epinephrine, regardless of response to therapy.

All other patients with anaphylactic syndromes should be observed in the ED or hospital as described.

For all discharged patients, the prevention of future allergic reactions should be stressed. The patient should be urged to remove inciting antigens from their environment. This may require a physician's note to an employer to request that the patient be allowed to avoid a workplace antigen. In certain cases in which desensitization for unavoidable antigens may be necessary, referral to an allergist is appropriate. Finally, the inciting antigen (if known) should be well-documented in the patient's medical record, especially if the antigen is a medication or latex.

Pearls, pitfalls and myths

Pitfalls

- Failure to administer epinephrine early and as needed in the patient's treatment
- Failure to recognize the subtle early presentation of anaphylaxis
- Failure to recognize the need for acute and definitive airway management
- Failure to recognize the contraindications for RSI in anaphylaxis patients

- Failure to anticipate difficulties in the treatment of patients taking beta-blockers
- Failure to observe patients for an adequate length of time
- Failure to admit high-risk patients
- Failure to anticipate the possibility of a biphasic allergic reaction
- Failure to appropriately administer and prescribe corticosteroids
- Failure to counsel the patient to avoid antigen triggers
- Failure to prescribe an epinephrine auto-injector for susceptible patients and to properly instruct them regarding its use
- Failure to make an appropriate referral to an allergist

Myths

- Patients with anaphylaxis always look sick on initial presentation.
- Epinephrine should only be used for patients in extremis.
- Airway compromise always follows a linear time course.
- Antihistamine agents are first-line treatments for anaphylaxis.
- Once patients get better, they never relapse.
- If the patient does not react immediately after exposure to an antigen, they will not have a significant anaphylactic reaction.

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14 Altered mental status

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Scope of the problem

The patient with altered mental status (AMS) presents significant challenges for emergency physicians, including potential life threat, rapid decision making and astute detective work. The etiology may be chronic or acute, life-threatening or benign, reversible or irreversible. One of nearly a dozen different organ systems might be implicated or harmed. Within minutes, a diligent emergency physician must narrow the differential to a manageable number of possible diagnoses and initiate appropriate treatment.

Terminology

AMS is an alteration of a patient's level of cognitive (knowledge-related) ability, appearance, emotional mood, speech and thought patterns.

Level of consciousness relates to one's level of awareness and responsiveness to his or her surroundings.

Lethargy is generally referred to as mild to moderate depression in level of consciousness. It implies an abnormal state of drowsiness or sleepiness from which it may be difficult to arouse the patient.

Stupor is a more profound depression of one's level of consciousness. It is an extreme form of lethargy requiring a greater stimulus to produce a lesser degree of arousal.

Coma is an abnormal state of deep unconsciousness from which a patient cannot be awakened.

Organic illness refers to impairment of normal anatomic and/or physiologic activity resulting in abnormal mental functioning. *Functional illness* generally refers to a physical disorder with no known or identifiable organic basis to explain the symptoms, as in psychiatric illness.

Delirium is an acute confusional state with an organic etiology. The key to this definition is that there is an alteration in both the level and the content of consciousness. Unrecognized delirium can result in significant morbidity and mortality. If treated, the majority of cases are reversible.

Dementia is an insidious deterioration of higher cortical function with an organic etiology. In distinct contrast to delirium, affected patients have a normal level of consciousness. Although acute insults and deterioration in mental status may be reversible, underlying dementia is rarely completely corrected.

Acute psychosis is a functional disease that needs to be distinguished from delirium and dementia. The hallmark of psychosis is loss of the ability to distinguish reality from fantasy. It can be very difficult to distinguish an acutely psychotic patient from one who is delirious.

Abulic state (akinetic mutism) is the inability to respond or act. For example, responsiveness may be so depressed in a patient with frontal lobe dysfunction that it may take the patient several minutes to answer a question.

Locked-in syndrome may leave the patient unable to respond, except for the ability to move the eyes in upward gaze. This occurs from destruction of pontine motor tracts.

Psychogenic unresponsiveness is a form of functional, nonphysiologic unresponsiveness.

Delirium versus dementia versus acute psychosis

Emergency physicians must make a concerted effort to distinguish delirium from other causes of altered mental states. This distinction can be difficult to make, but is critical to the patient's ultimate outcome. The etiologies of delirium are extensive, and many causes have the potential for serious morbidity or mortality. Distinguishing features between these three conditions are identified in Table 14.1.

Table 14.1 Delirium versus dementia versus acute psychosis

	Delirium	Dementia	Acute psychosis
Definition	Acute confusional state	Insidious deterioration in higher cortical functions	Loss of the ability to distinguish reality from imaginary
Organic vs. functional	Organic disease	Organic disease	Functional disease
Onset	Hours to days	Months	Hours to days
Course	Fluctuating course	Progressive course	Stable course
Level of consciousness	Altered	Normal	Normal
Hallucinations	Visual – common	None	Auditory – common
Orientation	Altered	Altered	Normal
Vital signs	Widely variable and fluctuating	Normal	Variable
Miscellaneous	Extreme agitation is common Reversible in >80%	Consider medications, thyroid disease and infections as a cause for exacerbations	Fixed delusions First attack common in patients <40 years old

Anatomic essentials

Arousal requires a healthy, functioning reticular activating system (RAS) and cerebral cortex. The midbrain portion of the RAS is key, and may be viewed as a driving center for higher structures. Loss of the midbrain reticular formation (MRF) produces a state in which the cortex appears to be waiting for the command to function. This ascending midbrain reticular activating system extends upward into the hypothalamus and thalamus. Every major somatic and sensory pathway stimulates the RAS at all levels, either directly or indirectly. Awareness and arousal also depend on proper functioning of the cerebral cortex. Unconsciousness will result from severe disruption of anatomic or physiologic function of either the MRF or both cerebral hemispheres. These critical structures may be compromised by structural, chemical or infectious etiologies. Unilateral insults to the cerebral cortex will not result in unconsciousness unless the brainstem is also affected.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 14.2).

History

What is/was the timing and course of events since the onset of change in mental status or level of consciousness?

A critical distinction between dementia and delirium is the time course. Therefore, this is a key question for the patient and family member(s). Dementia is generally insidious in onset compared with delirium, which is acute and dramatic. With respect to acute psychosis versus delirium, the distinction is less clear. However, patients suffering from a state of delirium will often have a waxing and waning course compared with the continuous nature of functional illness.

What methods can the physician use to help overcome difficulties obtaining a detailed history from the patient?

As with most medical problems, the quality of the history often dictates the success and timeliness of the emergency department (ED) evaluation. One of the major inherent difficulties in the evaluation of patients with AMS is the inability to get a meaningful and reliable history from the patient. All other sources for medical history must be tapped: paramedics, relatives, friends, medic alert tag, wallet, personal physician, hospital records and pill bottles. Social services and law enforcement may assist in the search for critical information about these patients. In addition to present history, the importance of obtaining past history (including suicidal ideation/suicide attempts), current medications and social history (substance abuse and living situation) cannot be overemphasized.

Physical examination

Vital signs

A thorough physical examination and rapid bedside testing may be more enlightening in patients with AMS than other problems presenting to the ED. An elevated temperature may indicate an infectious etiology or, if pathologically high (≥106 °F), may suggest heat stroke, drug interactions, or an intracranial process. The respiratory rate and pattern may suggest intracranial pathology or an acid–base disorder. The heart rate, rhythm and electrocardiogram (ECG) can offer a number of clues about toxins (digoxin, tricyclic antidepressants [TCAs], beta-blockers), metabolic derangements (high or low calcium or potassium), or closed head injury (with deep inverted T waves). Elevated blood pressure, a widened pulse pressure (systolic minus diastolic blood pressure), and slow heart rate (Cushing reflex) may be consistent with elevated intracranial pressure. Pulse oximetry may direct the clinician to causes of hypoxia and whether or not low oxygenation relates to the patient's overall presentation.

Head, ears, eyes, nose and throat

Head

A number of components of the general physical examination are particularly helpful. *Breath odor* may provide clues to the presence of diabetic ketoacidosis (DKA), liver failure (fetor hepaticus), or a number of toxins (e.g., alcohol, insecticides [onion odor], paint or glue, gasoline, cyanide [bitter almond odor], and arsenic [garlic]). The head should be examined for signs of acute or recent trauma (hemotympanum, cephalohematoma, cerebrospinal fluid [CSF] leak, Battle sign, raccoon eyes) and for past surgery (shunt, cranial defect, surgical scars).

Eyes

The pupillary exam is essential as it can provide information about structural and metabolic abnormalities. One must look for pupillary size and the presence of asymmetry. Examining the direct and consensual response to light will determine the integrity of the afferent function of the optic nerve and efferent function of the third cranial nerves. A unilateral dilated pupil in an altered patient is due to herniation until proven otherwise (Figure 14.1). The mass causing the pathology is usually on the same side as the dilated pupil, as demonstrated in the figure. In the awake, alert patient, the primary life-threatening cause for a unilateral dilated pupil is compression of cranial nerve III by a mass (such as a posterior communicating artery aneurysm). There are a number of other causes for pupillary dilation that are not serious (traumatic

Table 14.2 Altered mental status red flags

History	Concerning diagnosis
Diabetes	Hypoglycemia, hyperglycemia
Polyuria, polydipsia, polyphagia	Diabetic ketoacidosis, hyperosmolar hyperglycemic state
Fever, chills, rigors	Occult or obvious infection, bacteremia, septicemia, sepsis, pneumonia, UTI, meningitis
Head trauma	Concussion, contusion, hemorrhage, post-traumatic seizure (postictal)
New or change in medication	Drug interaction or adverse reaction causing change in mental status
Recent surgery	Post-op infection (urine, lung, wound, abscess), post-anesthesia malignant hyperthermia
Headache and/or stiff neck	CNS infection, subarachnoid hemorrhage, postictal phase following seizure
Environmental exposures	Heat stroke or hypothermia
Recent travel abroad	Uncommon infections (malaria, dengue, typhoid fever)
Cancer with recent chemotherapy	Cancer-related fever, tumor fever, neutropenic fever, tumor lysis syndrome, opportunistic infection, hypercalcemia
Intravenous drug use	Endocarditis, epidural abscess, osteomyelitis, necrotizing fasciitis
Examination finding	**Concerning diagnosis**
Hypoxia, rales, rhonchi, asymmetry or abnormal lung auscultation	Pneumonia, respiratory failure
Nystagmus	Recreational drug injection, brainstem infarction
Flank tenderness	Pyelonephritis, renal abscess, psoas muscle abscess
Scleral icterus, jaundice	Liver failure, hepatitis
Neck mass	Thyroid disease, abscess
Petechial rash	Meningococcemia, Rocky Mountain Spotted Fever, thrombotic thrombocytopenic purpura
Battle sign, raccoon eyes, hemotympanum, clear (CSF) rhinorrhea, scalp contusion or cephalohematoma	Traumatic brain injury, concussion, contusion, hemorrhage, skull fracture, pneumocephalus
Kernig's or Brudzinski's sign	Meningitis
Cardiac murmur, Janeway lesions, splinter hemorrhages, Roth spots, needle track marks	Endocarditis, complex skin infections
Back or vertebral (spinal) tenderness	Lumbar osteomyelitis or epidural abscess
Abdominal tenderness with or without guarding	Acute abdominal process (appendicitis, cholecystitis, mesenteric ischemia, diverticulitis)
Evidence of an immunocompromised host (thrush, wasting, diffuse adenopathy)	Opportunistic infection, such as cryptococcal meningitis
Unusual odors	DKA (fruity, ketones) Cyanide (almonds) Arsenic (garlic) Insecticides (onions) Hydrocarbons (chemistry lab, glue, paints) Hydrogen sulfide (rotten eggs)

CNS: central nervous system; CSF: cerebrospinal fluid; DKA: diabetic ketoacidosis; UTI: urinary tract infection.

mydriasis, intentional or accidental topical medications, Adie's pupil, anisocoria). Bilateral pupillary constriction (pinpoint pupils) may represent an opiate overdose or pontine lesion.

The *funduscopic examination* is a critical, often underutilized component of the eye examination. Flame hemorrhages are characteristic of hypertensive bleeds. Increased intracranial pressure will produce changes associated with papilledema, in which the disc margins of the optic nerve are indistinct (Figure 14.2). Earlier subtle findings of increased intracranial pressure include absent venous pulsations, although this is not specific; later, findings include blurred disc margins and engorged vessels. Funduscopic changes associated with diabetes (neovascularization, hemorrhages, exudates) or with methanol ingestion (optic disc hyperemia and retinal edema) may provide clues to the cause of the patient's altered level of consciousness.

Eye movements are generally more helpful than commonly thought. Assuming the brainstem is intact, most comatose patients exhibit slowly roving eye movements. In contrast, malingering or hysterical patients feigning

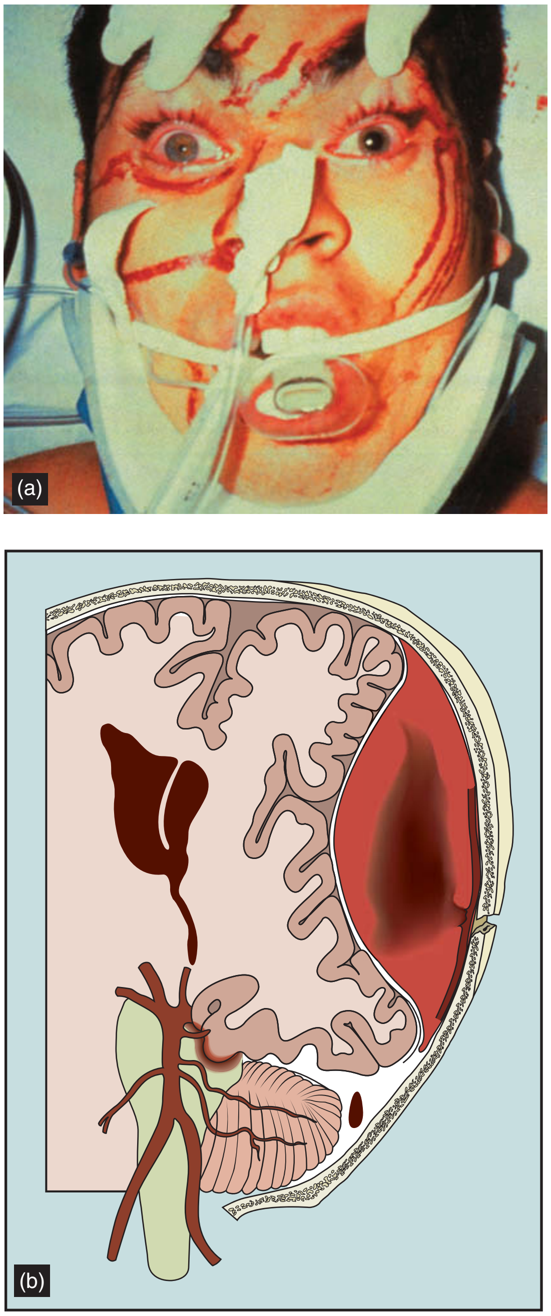

Figure 14.1
(a) Photograph of patient with transtentorial herniation from blunt head trauma. The right pupil is constricted normally; the patient's left pupil is fixed and dilated. (b) Illustration of an epidural hematoma with acute mass effect and compression of the ipsilateral cerebral peduncle resulting in uncal herniation. Reproduced from D. Mandavia et al, *Color Atlas of Emergency Trauma*, Cambridge University Press, Cambridge, 2003.

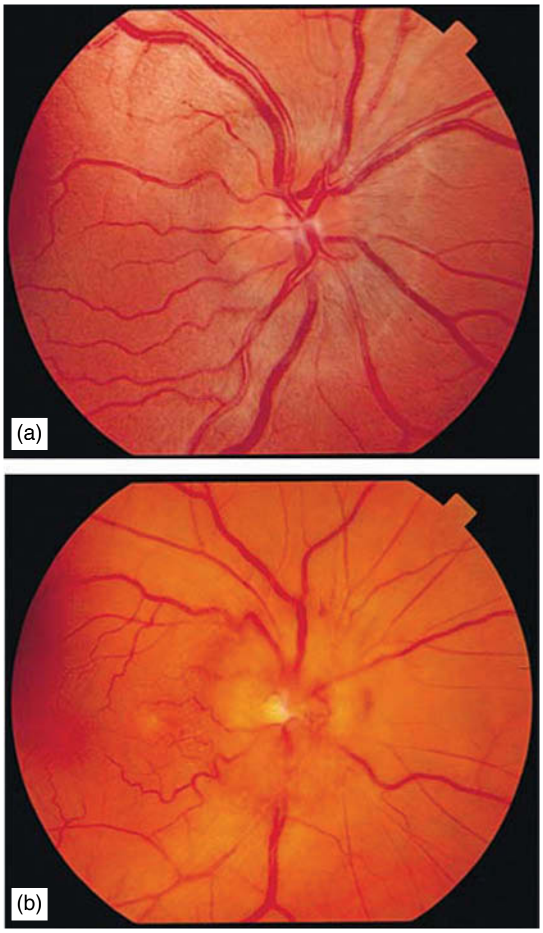

Figure 14.2
(a) Early papilledema with disc elevation, blurring of the margins, hyperemia and venous engorgement in the right eye. (b) Acute papilledema with increased elevation and hemorrhages on the disc surface in the right eye. Reproduced with permission from Tasman W et al, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

coma have spontaneous eye movements that tend to be rapid and rigid. If both eyes cross midline, the brainstem is intact. When the eyes are fixed in one direction, they commonly "look" toward the side of a hemorrhage or away from a destructive lesion.

Eyelid tone may help to differentiate organic disease from hysteria. Hysterical patients may offer some resistance to an examiner's attempt to open their eyes. They tend to close their lids quickly. "Fluttering" eyelids are commonly seen in patients feigning unresponsiveness. Patients with organic coma offer no resistance to lid opening, then close the lids slowly and incompletely. Unilateral ptosis may be seen in patients with a third cranial nerve palsy, or when there is disruption of the sympathetic chain in association with *Horner's syndrome*.

The *oculocephalic reflex (doll's eyes)* depends upon the medial longitudinal fasciculus (MLF), which receives constant input about the patient's head position from the semicircular canals. Without cortical input, the eyes are typically directed straight ahead and remain fixed in the orbit as the head is turned. The oculocephalic reflex is elicited by rotating the head briskly from side to side (Figure 14.3). If the brainstem is intact, the eyes deviate opposite to the direction of head rotation (head rotated right, eyes deviate left). Confusion often arises because

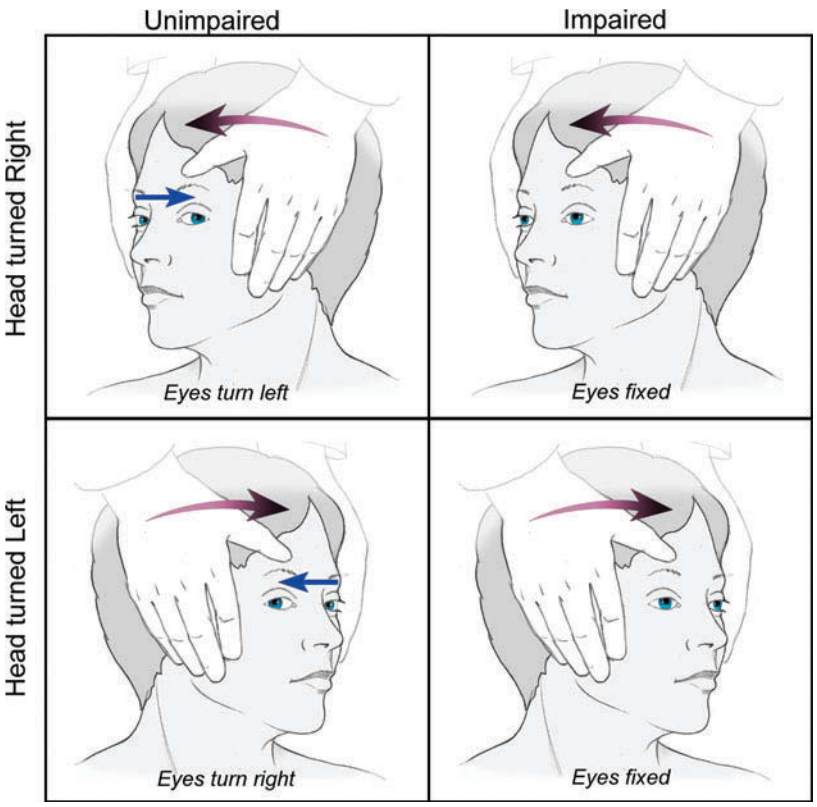

Figure 14.3
Oculocephalic reflex (Doll's eyes). © Chris Gralapp.

the patient's eyes continue to look straight ahead. In other words, they remain focused in the same direction, possibly giving one the impression that they did not move. The examiner must remember that in order for the eyes to remain fixed in a given direction, when the head is turned, the eyes had to move. Cervical spine injury must be excluded before performing this maneuver.

Oculovestibular testing (cold calorics) is another underutilized ED maneuver that yields important clinical information about the integrity of the brain and brainstem (Figure 14.4). The test is performed by positioning the patient supine with the head elevated 30° in order to isolate input from the horizontal semicircular canals. In this position, given intact tympanic membranes, 10–20 mL of ice-cold water or saline is injected into the auditory canal. Cooling of the mastoid bone causes alteration in endolymphatic flow within the canals. Information is then transmitted to vestibular nuclei and pontine gaze centers, triggering the eye movements.

If the practitioner always uses cold water and remembers the three points described in Table 14.3 and Figure 14.4, the examination can be extremely helpful.

Neck

The neck must be examined for the presence of nuchal rigidity (meningismus) that often occurs when the meninges become inflamed by blood or infection. Thyroid or parathyroid disease may be responsible for an alteration in mental status or level of consciousness. The neck should be examined for an enlarged thyroid or the presence of a surgical scar suggesting previous thyroid or parathyroid surgery.

Pulmonary

Hypoxia and hypercarbia are uncommon causes of an altered level of consciousness. However, findings consistent with severe lung disease and respiratory distress should raise consideration for these entities. One must look for the barrel chest of the patient with emphysema, wheezing and a prolonged expiratory phase in patients with obstructive lung disease, and for evidence of consolidation (e.g., egophony, vocal or tactile fremitus, or whispered pectoriloquy) in those with pneumonia.

Table 14.3 Cold calorics

Response	Interpretation
Both eyes deviate, nystagmus (slow phase toward stimulus, fast back to midline)	Patient is not comatose
Both eyes tonically deviate toward cold water	Coma, but intact brainstem
No eye movement or movement only of eye ipsilateral to the stimulus	Brainstem damage; internuclear ophthalmoplegia (brainstem structural lesion)

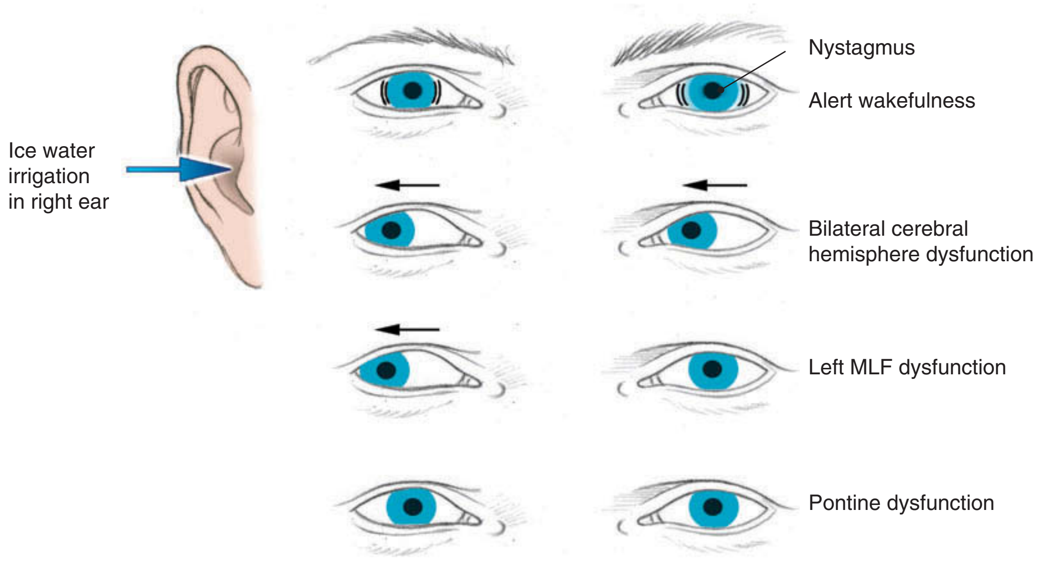

Figure 14.4
Oculovestibular testing (cold calorics). MLF: medial longitudinal fasciculus. © Chris Gralapp.

Cardiac

The cardiovascular system is rarely the primary source for mental status changes; yet dysrhythmias, valvular heart disease and severe heart failure can be implicated. The heart should be examined for the presence of an irregular rhythm or extra heart sounds, including an S3, murmurs and/or rubs. New murmurs may be appreciated following valve damage from endocarditis.

Abdomen

A thorough abdominal examination should be performed, looking for signs of infection, organomegaly, mass or obstruction. Any cause of abdominal infection can lead to an alteration in consciousness, especially in elderly patients. Localized tenderness, absent bowel sounds, rebound tenderness and rigidity are all signs of a serious intra-abdominal process. Bowel sounds may also be abnormal in certain ingestions, such as cholinergics and anticholinergics. Liver failure is a common cause of altered consciousness. An enlarged liver or a small, hard nodular liver may be noted during examination. Severe alcoholics may have stigmata of end-stage liver disease, such as spider angiomas, caput medusae and ascites. The spleen should also be examined for size, shape, tenderness and nodularity. Late findings of catastrophic abdominal processes such as pancreatitis and ruptured abdominal aortic aneurysm (AAA) include periumbilical ecchymosis (Cullen's sign) and flank ecchymosis (Grey Turner's sign).

Rectal

Digital rectal examination may identify a mass suggestive of malignancy or melena associated with an upper gastrointestinal bleed. Rectal tone should also be noted, as this may be important in discriminating between organic and functional illness.

Skin

The skin examination may reveal signs of local or systemic disease. Skin temperature and hydration offer clues about infection, blood sugar (moist with hypoglycemia and dry with hyperglycemia), and certain toxins (moist from cholinergic agents and dry and/or red from anticholinergic agents). The brow covered with "uremic frost" may suggest renal disease. Spider angiomas, palmar erythema and jaundice may be apparent in patients with long-term liver disease. Hypothyroid patients often develop coarse, dry hair; thinning of the lateral aspect of the eyebrows; and dry, rough, pale skin. Needle tracks, petechiae and other rashes may provide significant clues of underlying disease processes.

Neurologic

The neurologic examination for patients with AMS must be thorough, beginning with careful observation. *Automatisms* (involuntary acts carried out as protective mechanisms) such as yawning, hiccups, sneezing and swallowing may be present with brainstem or frontal lobe pathology. Abnormal respiratory patterns (central neurogenic hyperventilation, apneustic, Cheyne-Stokes, ataxic) are suggestive of lesions at various levels of the brainstem (Table 14.4). The key is to recognize the abnormal respiratory pattern rather than remember the exact level of the brainstem implicated. Body posture is also important (discussed in the section on the motor examination).

Table 14.4 Progressive brainstem dysfunction – rostral caudal progression with increasing pressure

Level of lesion	Respiratory pattern	Other
Early brainstem compression	Cheyne-Stokes	Small but reactive pupils Plantar reflex becomes extensor
Midbrain and upper pons	Central neurogenic hyperventilation	Dilated nonreactive pupils Will see spontaneous decerebrate posturing
Pons and upper medulla	Quiet respirations – normal rate	Loss of Doll's eyes + Cushing reflex No response to painful stimuli
Medulla	Slow irregular respirations leading to apnea	Widely dilated, fixed pupils, hypotension

Mental status assessment

Glasgow Coma Scale (GCS): This 15-point scale was developed to assess head-injured patients; however, it is commonly used to describe or serially assess the mental status of a patient. The lowest score is 3, the maximum (best) score is 15. The score is calculated by assigning the number associated with the patient's best response to eye, verbal and motor assessments (Table 14.5). It is important to compare two GCS measurements to capture fluctuating changes consistent with delirium.

AVPU: Awake, verbal, painful, unresponsive. This simplified four-part scale is used to describe the patient's level of consciousness (Table 14.6).

Orientation assessment: Person, place and time.

Memory: Test short-term memory by asking the patient to remember three common objects and recall them 3 minutes later.

Specific mental status examinations: Two tests have been developed and studied to assist non-psychiatrists in the evaluation of mental status. Both tests are easy to perform and accurate with a high degree of interobserver reliability. The mini-mental status examination (MMSE) is an excellent test, but is geared mostly toward the evaluation of dementia and content of thinking. The best bedside test for identifying delirium is the confusion assessment method (CAM).

Table 14.5 Glasgow coma scale (GCS)

Eye opening	Verbal response	Motor response
4 Spontaneous	5 Oriented	6 Obeys commands
3 To voice	4 Confused	5 Localizes
2 To pain	3 Inappropriate words	4 Withdraws
1 None	2 Incomprehensible sounds	3 Decorticate posturing
	1 None	2 Decerebrate posturing
		1 None

Table 14.6 AVPU

A	Awake and aware
V	Responds to verbal stimuli
P	Responds to painful stimuli (A painful stimulus potentially or actually damages body tissue. Typical maneuvers used in the ED include forcefully grinding the knuckles of one's fist into the sternum or squeezing two toes together while a firm object is wedged between them.)
U	Unresponsive

The astute physician will perform the CAM while observing the patient's responses during the history and physical examination. The clinician must pay attention to the patient's thinking, communication skills and level of consciousness to complete the assessment. The patient is considered delirious if he or she has an acute onset illness with a fluctuating course, disorganized thinking or altered level of consciousness, and easy distractibility. Examples of being easily distracted include the patient who interrupts the history and physical examination by striking up a conversation with other patients or health care providers, or changes the focus to items on the walls. The patient with disorganized thinking will communicate with disconnected sentences or will change topics from moment to moment, making it difficult or impossible to follow the line of thinking.

CAM (to diagnose delirium):

1. Acute onset with a fluctuating course, and
2. Easily distracted, inattentive
   and
3. Altered level of consciousness, or
4. Disorganized thinking

A positive CAM test includes numbers 1 *and* 2 plus number 3 *or* 4.

Cranial nerves (CNs) must be tested as part of a complete neurologic examination. Portions of this examination were completed during the evaluation of the eyes. The remaining CNs are tested to help identify and localize CN lesions.

Motor and sensory testing assess for tone, focality and evidence of herniation. Determining the extent or severity of alteration of consciousness is aided by evaluating motor responses to verbal and painful stimuli. Purposeful movements indicate a functioning brainstem and cerebral cortex. Posturing may be apparent in the presence of a diffuse metabolic or toxic insult, or secondary to herniation. *Decorticate posturing* (flexion of arms and hyperextension of legs), *decerebrate posturing* (arms and legs extended and internally rotated), or both may occur as herniation progresses. Abnormal motor tone may suggest an acute cardiovascular accident (CVA) or spinal cord injury (if tone is absent). Focality identified on the motor or sensory examination may help identify the level of a cord lesion or confirm that the insult is in the brain. Tone may be increased by the presence of various toxins, or in neuroleptic malignant syndrome (NMS). Severe hypothermia, massive overdose of sedatives or hypnotics, hypoglycemia, CVA, and the postictal state can mimic structural neurological diseases.

Deep tendon reflexes (DTRs) may help localize the level of lesion or place the insult in the central or the peripheral nervous system. Symmetry of responsiveness is the key to assessment of DTRs. The plantar (Babinski) reflex becomes abnormal in many patients with upper motor neuron pathology, especially related to the corticospinal tracts. The response is abnormal if the big toe dorsiflexes and the other toes fan outward. An asymmetric response is also significant.

Differential diagnosis

During the evaluation of patients with AMS, the process of generating and eliminating diagnoses evolves. The diagnostic possibilities are so numerous and broad that emergency practitioners need to be organized and systematic in their approach. *It is imperative to develop a process that works for you and to use it regularly*. Many physicians use mnemonics to help them focus the broad differential. One common mnemonic for the altered patient is AEIOU TIPS (Table 14.7). The head-to-toe approach is intuitive, simple to remember and apply, and nearly 100% inclusive (Table 14.8). Start from the head and progress down the body, considering diagnostic possibilities associated with each anatomic system as they are encountered.

Table 14.7 Mnemonic for ALOC differential diagnosis

AEIOU TIPS	
A	Alcohol, other toxins, drugs
E	Endocrine, electrolytes
I	Insulin (diabetes)
O	Oxygen, opiates
U	Uremia (renal, including HTN)
T	Trauma, temperature
I	Infection
P	Psychiatric, porphyria
S	Subarachnoid hemorrhage, space-occupying lesion, seizure

ALOC: altered level of consciousness; HTN: hypertension.

Table 14.8 Differential diagnosis of altered mental status

Head

1. Supratentorial
    (a) Unilateral hemispheric disease with herniation
        – Abscess
        – Hemorrhage (including traumatic)
        – Infarction
        – Tumor (primary or metastatic)
    (b) Concussion/contusion
    (c) Infectious – meningitis/encephalitis, expand if immunocompromised
    (d) Seizure (postictal period)/nonconvulsive status
    (e) Subarachnoid hemorrhage
    (f) Cerebral vascular accident
    (g) Wernicke's encephalopathy
    (h) Functional (psychiatric)
2. Infratentorial
    (a) Basilar artery occlusion
    (b) Brainstem tumors
    (c) Cerebellar hemorrhage
    (d) Pontine hemorrhage
    (e) Traumatic posterior fossa hemorrhage

Mouth

1. Toxins – medications
2. Toxins – drugs
    (a) Alcohols
    (b) Anticholinergics
    (c) Anticonvulsants
    (d) Barbiturates
    (e) Carbon monoxide
    (f) Cyanide
    (g) Hallucinogens
    (h) Heavy metals
    (i) Opiates
    (j) Phenothiazines
    (k) Salicylates
    (l) Sedative/hypnotics
    (m) Sympathomimetics
    (n) Tricyclic antidepressants

Neck

1. Thyroid disease
    (a) Hyperthyroidism (thyroid bruits, tender goiter, tender neck mass)
    (b) Hypothyroidism (goiter)
    (c) Post-operative endocrinopathy
2. Parathyroid disease (hypercalcemia)

Chest/heart

1. Hypoxia
2. Hypercarbia
3. Congestive heart failure (CHF)
4. Pulmonary emboli
5. Rhythm disturbances
6. Murmurs (especially new)

Abdomen

1. Liver – hepatic encephalopathy
2. Kidney – renal insufficiency, electrolyte abnormalities, infection
3. Adrenal insufficiency (endocrinopathies)
4. Pancreas (diabetes: hyper- or hypoglycemia)
5. Peritonitis
    (a) Cholecystitis
    (b) Appendicitis
    (c) Spontaneous bacterial peritonitis
    (d) Perforated viscus

Abdomen – vascular

1. Mesenteric ischemia
2. Abdominal aortic aneurysm

Skin

1. Temperature – hypothermia, heat stroke
2. Color – liver failure, renal failure, hypoxia
3. Rash – vasculitis, thrombotic thrombocytopenic purpura (TTP), toxic shock, endocarditis

Miscellaneous

1. Sepsis
2. Hyperviscosity syndromes
3. Vasculitis (cerebritis)

Diagnostic testing

Laboratory and radiographic testing of unstable, critically ill or confused patients fall into two categories. The first group of essential tests can be completed in 10 minutes or less. The etiology of a patient's altered mental status can be narrowed substantially by identifying the timing of illness onset, performing a thorough history and physical examination, and utilizing these rapid tests.

These relatively inexpensive tests (not arranged in any particular order) yield a tremendous amount of useful information:

1. Glucose (dextrostick or glucometer)
2. Pulse oximetry (hypoxia)
3. Hematocrit (blood loss)
4. Breathalyzer (ethanol)
5. Urinalysis (infection, hyperglycemia, ketosis, dehydration, toxicology)
6. ECG and cardiac monitor (electrolyte abnormalities, toxins, acute cardiac disease, hypercalcemia [short QT], tricyclics [rapid rate with a wide QRS], digitalis toxicity [various blocks and asymmetric T wave inversions])
7. Arterial blood gas (acidosis, hypercarbia, hypoxemia)

Other tests to consider (which may take longer to obtain results):

1. Complete blood count, electrolytes (particularly important is sodium), blood urea nitrogen (BUN), creatinine (Cr), serum osmolality, calcium and magnesium
2. Carboxyhemoglobin level
3. Directed drug screen
4. Chest X-ray
5. Head computed tomography (CT): done before lumbar puncture (LP) to rule out focal lesions and hemorrhage
6. LP with CSF analysis
7. Peritoneal tap (if ascites fluid present)
8. Thyroid function studies
9. Cervical spine X-rays if trauma cannot be excluded
10. Magnetic resonance imaging (MRI) – herpes encephalitis, Wernicke's encephalitis
11. Electroencephalography (EEG)

General treatment principles

As with all ED patients, emergency treatment begins with the ABCs (airway, breathing, circulation). The main goals of treatment are physiologic stabilization, symptom relief and specific diagnosis-driven treatment plans. The first four recommendations are commonly known by the acronym *DON'T* (dextrose, oxygen, naloxone and thiamine).

Dextrose

Dextrose should be administered if glucose testing indicates hypoglycemia. Glucometers are extremely reliable and rarely miss true hypoglycemia. One ampule of D50 (25 g of glucose) will raise the serum glucose by approximately 130 mg/dL (the range is from 30–250 mg/dL). Administer 50 mL of 50% dextrose intravenous (IV) in adults, 4 mL/kg of 25% dextrose (0.5–1 g/kg) IV in children, and 5 mL/kg of 10% dextrose (0.5 g/kg) IV in neonates.

Oxygen

Oxygen saturations should be assessed by pulse oximetry on all patients with AMS. Supplemental oxygenation via nasal cannula or face mask should be provided to patients with oxygen saturations less than 92%, those with possible carbon monoxide poisoning, and those under significant stress. Certain patients with chronic obstructive pulmonary disease (COPD) retain CO_2. They may develop AMS from CO_2 narcosis if they have inadvertently received high-flow oxygen for an extended period of time. However, all hypoxic patients with increased work of breathing (WOB) and AMS should receive oxygen therapy.

Naloxone

Naloxone is relatively benign and should be considered in all hypoventilating patients with altered levels of consciousness and possible opioid exposure. The standard dose of naloxone is 2 mg IV in adults and 0.1 mg/kg IV in children (maximum of 2 mg IV). The effects of a narcotic ingestion can partially be reversed with small aliquots of naloxone in the range of 0.1 to 0.4 mg IV. In contrast, the ingestion of some of the synthetic opioids and combination medications (e.g., diphenoxylate/atropine, methadone, propoxyphene, pentazocine) may require as much as 10 mg of naloxone to reverse. Naltrexone, a long-acting narcotic antagonist, has a limited role in the ED because of the risk associated with sending patients home who may have ingested longer-acting narcotics (these patients may decompensate after discharge).

Thiamine

Thiamine administration should be considered in all patients with altered consciousness unless the cause is known. Thiamine is a safe, effective and inexpensive treatment for Wernicke's encephalopathy. Thiamine depletion-induced encephalopathy is characterized by the abrupt onset of ophthalmoplegia, ataxia and confusion. If not treated promptly, it may progress to irreversible Korsakoff's psychosis (confabulation, retrograde amnesia and impaired ability to learn). Although Wernicke's encephalopathy is rare, it is reversible if recognized and treated early. Physicians must remember that alcoholism is not the only cause of thiamine deficiency. Patients are at risk for Wernicke's if they have any cause of malnutrition or vitamin deficiency, including hyperalimentation, anorexia, bulimia, pregnancy and malignancy. Thiamine dosing is 100 mg slow IV push in adults; the dose for children is 10–25 mg IV.

Volume replacement

Many altered patients are dehydrated because of prolonged agitation and excessive stimulation over hours to days. Care must be taken as diseases such as renal failure or water intoxication may produce delirium in volume-overloaded patients.

Temperature control

Fever may be a manifestation of a disease process, and can contribute to ongoing cell damage. Fever may be secondary to heat stroke, sepsis, aspirin toxicity or drug overdose, and should be treated with the appropriate interventions. One may use acetaminophen (rectal suppository is an option) or ibuprofen (in the absence of contraindications), cooling blankets, wet sheets with a fan, or ice packs in the axillae or groin to reduce the temperature.

Flumazenil

Flumazenil is a competitive benzodiazepine antagonist. Independently, the drug is benign. Its use is limited in emergency medicine because blocking benzodiazepines from their receptor sites can unmask epileptogenic potential. Patients addicted to benzodiazepines may seize from this pharmacologically-induced withdrawal state. Patients who ingested drugs or medications that can cause seizures along with benzodiazepines may seize once the “protective” effect of the benzodiazepines has been removed. Yet flumazenil may serve a diagnostic and therapeutic role in selected cases. Administration of flumazenil may awaken patients who have acutely ingested benzodiazepines, preventing unnecessary endotracheal intubation and its associated complications. It may also eliminate the need for expensive testing and possible admission. The risk of seizures may be ameliorated by careful titration of flumazenil; increments of the smallest dose recommended may decrease the chance of seizure. Clinicians should not eliminate flumazenil from the arsenal of critical care medications solely based on the risk of seizures. As with all medications, the risks and benefits must be carefully considered before administration. The dose of flumazenil is 0.2–1 mg IV in adults.

Physostigmine

Physostigmine is a cholinergic drug that can diagnose and treat overdoses of anticholinergic substances and agents with anticholinergic-like properties. The goal of physostigmine administration is to reverse the patient’s anticholinergic signs and symptoms. Physostigmine is administered in small 0.5-mg aliquots IV up to a maximum of 2 mg. Administration end points include clear reversal of anticholinergic signs and symptoms, the development of cholinergic symptoms (salivation, lacrimation, urination, defecation, gastric irritation, emesis), or delivery of 2 mg of drug. Physostigmine must be used with great caution, however; many centers do not use this drug at all. When administered to patients who have co-ingested agents that cause myocardial depression (such as TCAs), the result can be cardiac standstill.

Antibiotics

Antibiotics are indicated in patients with a suspected infectious cause for their AMS. When considered, these drugs should be administered early in the ED course, often before a source of infection has been identified. Infections of the urinary tract, lungs, skin, genitalia, or meninges are often implicated. The choice and dose of antibiotic must be directed by the organisms suspected. Whenever possible, blood and urine cultures should be obtained prior to the administration of antibiotics. In cases of suspected bacterial meningitis, antibiotics should be administered before performing the LP.

Miscellaneous

The myriad possible etiologies for AMS preclude a discussion of all possible emergent treatments. The potential exists to prepare the patient for neurosurgery, treat significant metabolic abnormalities, initiate therapy for thyroid dysfunction, or support the patient until drugs are metabolized or the postictal period passes. Specific antidotes and treatment for drug ingestions are discussed in greater detail in Chapter 41.

Special patients

Geriatric

The geriatric population is at special risk for AMS for a number of reasons. Several studies have shown that 40% of all geriatric patients over 70 years of age have some degree of AMS. Of these, approximately 25% had alterations in their level of consciousness, 25% had delirium and 50% had cognitive impairment. The most common cause of AMS was multifactorial, followed by medication (22–39% of all cases), infection, metabolic disturbance (such as diabetes mellitus), trauma, neoplasm, cardiovascular disease, pain and dehydration/nutritional abnormalities. Not surprisingly, elderly patients had greater morbidity and mortality compared with younger populations. Acute confusional states are more likely to herald an infectious process in the elderly than classic symptoms of fever, pain and tachycardia seen in younger patients.

Pediatric

In contrast to the elderly, children and infants with AMS usually present with treatable causes that often lead to favorable outcomes. Because psychiatric disease is an unusual cause of AMS in young children, an exhaustive search for an organic cause is typically required. The most common cause of AMS in a pediatric patient is toxicologic in nature. The critical factors in these patients are early detection and prompt treatment with gut decontamination and antidotes. Other causes of AMS in children include infections, trauma, metabolic derangements and child abuse. Complications unique to pediatric populations must be recognized and anticipated. For example, unlike in adults, hypoglycemia and metabolic derangements are commonly seen following beta-blocker ingestions and exposure to alcohols or perfumes. Failure to consider unique causes of hypoglycemia in children could lead the provider to the wrong treatment algorithm. Ruling out central nervous system (CNS) infection with a lumbar puncture is recommended once other causes of AMS have been excluded.

Immune compromised

The immunocompromised patient with AMS can be a medical quagmire. This patient population includes those with malignancies, immunosuppressive therapies and immunocompromising diseases such as acquired immune deficiency syndrome (AIDS). Not only are these patients at greater risk for complications from any given disease, but also at risk for pathology not seen in the usual clinical setting. For instance, toxoplasmosis in the HIV-positive patient is a serious condition that may present with subtle findings, making it difficult to diagnose. Other CNS diseases and organisms threaten this patient population and commonly cause altered levels of consciousness. These include cryptococcus, cytomegalovirus, herpes simplex, bacterial infections, and CNS malignancies such as lymphoma. It is important to remember that significant chronic illness and IV drug use also predispose individuals to opportunistic infections. History taking must include meticulous attention to medication lists (both prescribed and over-the-counter), recent changes in diet (increased protein in renal dialysis patients) and environmental exposures, as these are critical in the evaluation of the altered, immunocompromised patient.

Disposition

AMS is a medical emergency. Most patients have reversible causes for their altered state and will clear their sensorium – usually from the metabolism of substances such as alcohol, recreational drugs and prescription medications, or by recovering from their postictal state. However, physicians must be meticulous because a small but significant number of patients can progress to coma or death unless rapid evaluation and treatment are instituted. Despite the risk of significant morbidity and mortality, young patients more commonly present with benign causes of AMS, and are more likely to be discharged from the ED following an extended period of observation.

Patients who present with an altered sensorium as the result of an intentional ingestion need acute psychiatric evaluation once they have been medically stabilized and observed for toxic side effects (e.g., dysrhythmias, hypoglycemia, or gastrointestinal [GI] bleeding). These patients also need to be stabilized with regard to any coexisting medical conditions.

Diabetics often present to the ED with hypoglycemia. In most cases, the cause is related to a change in diet, dietary noncompliance, a change in activity, illness, or an inadvertent medication error. Once infectious causes of their glucose disturbance have been ruled out, these patients may be safely discharged home after treatment, observed oral intake and education, preferably with a friend or family member who can offer assistance. Patients with hypoglycemia secondary to long-acting oral agents must have further observation and treatment either in the hospital, ED, or observation unit.

Elderly and immunocompromised patients are far more complex and often require hospitalization regardless of the ultimate cause of their AMS. It is not unusual for this population to experience a persistent decline in their baseline level of functioning with a loss of at least one activity of daily living. Not surprisingly, elderly hospitalized patients have longer hospital stays, higher mortality rates and increased rates of institutional care after hospitalization. Patients with underlying dementia often suffer significant deterioration in their sensorium despite seemingly minor medical insults. Hospitalization is often required to deal with social as well as medical concerns in this patient population.

Pearls, pitfalls and myths

The clinical arena that includes altered states of consciousness is complex and high-risk. There are many pitfalls. With meticulous evaluation, however, most can be avoided.

- The enormous list of differential diagnostic possibilities in AMS should be considered, without narrowing the possibilities too early.
- The first critical branch point is to recognize the importance of distinguishing delirium from dementia and psychosis. Traveling down the wrong path at this initial juncture can result in harm to the patient.
- Patients with delirium have a mortality rate of approximately 15%, but the causes are reversible 80% of the time.
- Recognize the difficulty in obtaining a quality history. Do not lose sight of the importance of that history, and aggressively overcome any obstacles to obtaining it.
- Remember the basics: ABCs, hydration, temperature control, and a thorough physical examination that includes the eyes, skin, odor and mental status examination using the CAM. The skin and neck exams are often overlooked when considering causes of AMS.
- An awake, alert patient with a unilateral dilated pupil has a posterior communicating aneurysm until proven otherwise.
- An intracranial mass will be on the same side as the dilated pupil in about 85% of cases.

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15 Bleeding

Jonathan E. Davis, MD

Scope of the problem

Patients with bleeding may present to the emergency department (ED) in a dramatic fashion, leading to anxiety for patients, caregivers and health care providers. In practice, bleeding may span from transient and self-limited (e.g., epistaxis) to life-threatening (e.g., hemorrhage resulting from major trauma).

Hemostasis, the halting of bleeding, results from well-regulated processes that balance two important functions: (1) maintaining blood in a fluid, clot-free state in normal vessels, and (2) inducing a localized hemostatic plug at a site of vascular injury. Hemostasis is a dynamic balance between two competing forces: anticoagulation and procoagulation. Most bleeding occurs in the setting of normal hemostasis. However, this chapter focuses on bleeding in the setting of abnormal hemostasis; that is, abnormalities of endogenous coagulation pathways. Specifically, this chapter covers bleeding resulting from problems with platelets (primary hemostasis) or the plasma coagulation cascade (secondary hemostasis), as well as treatment of such disorders. Frequently encountered hemostatic abnormalities may result from acute or chronic disease states (e.g., thrombocytopenia, sepsis, chronic liver disease) or medication administration (e.g., aspirin, heparin, warfarin).

Anatomic essentials

Normal hemostasis results from a set of well-regulated processes. Three essential components are necessary for normal hemostasis: the vascular endothelium, functional platelets, and the plasma coagulation cascade.

Clot formation at a site of vessel injury results from a series of events that follow a breach in the integrity of the vascular endothelium (Figure 15.1). Damage to vascular endothelial cells causes exposure of subendothelial extracellular matrix components, including von Willebrand Factor (vWF). Circulating platelets adhere to vWF and become activated (change shape and release secretory granules). The secreted products recruit additional platelets, leading to formation of a platelet plug at the site of injury. Platelet plug formation is termed *primary hemostasis*. Disorders of primary hemostasis typically present with mucocutaneous bleeding (i.e., petechiae, epistaxis, hematuria). It is important to note that either an abnormal *quantity* of platelets (low platelet count) or abnormal *quality* (function) of platelets, whether present in adequate numbers or not, may lead to bleeding.

Tissue factor is an endothelial-based procoagulant that is also expressed at the site of vascular injury. It acts in conjunction with secreted platelet factors to activate the plasma coagulation cascade (Figure 15.2). The plasma coagulation cascade has both intrinsic (platelet initiated) and extrinsic (tissue factor initiated) arms. This cascade culminates with conversion of soluble fibrinogen to insoluble *fibrin*. Fibrin acts as cement, stabilizing the initial platelet plug. The formation of a polymerized fibrin cement cap is termed *secondary hemostasis* (Figure 15.3). Disorders of secondary hemostasis manifest clinically by deep and delayed bleeding (i.e., joint, intracranial, retroperitoneal hemorrhage).

As hemostasis is a dynamic balance between pro- and antithrombotic forces, endogenous antithrombotic activity is incited by the formation of a platelet plug. It is useful to highlight the three primary endogenous antithrombotic systems: antithrombin III, proteins C and S, and the plasmin system.

Antithrombin III regulates the coagulation cascade by inactivating other coagulation factors. Its principle site of

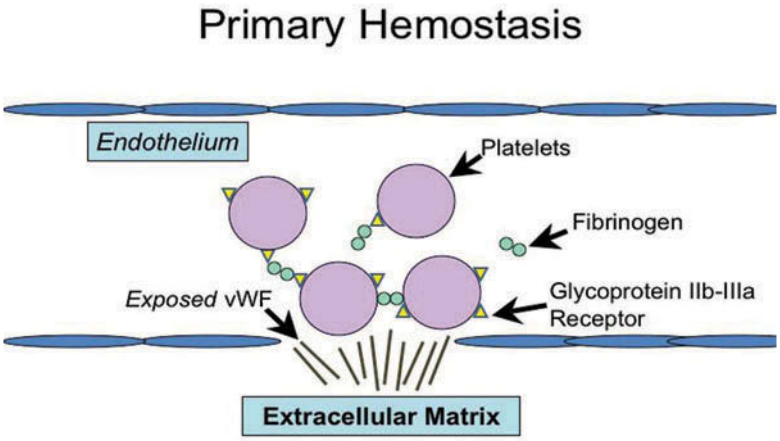

Figure 15.1
Primary hemostasis. Platelets organize into a platelet plug at the site of endothelial injury by binding subendothelial von Willebrand factor (vWF) and circulating fibrinogen.

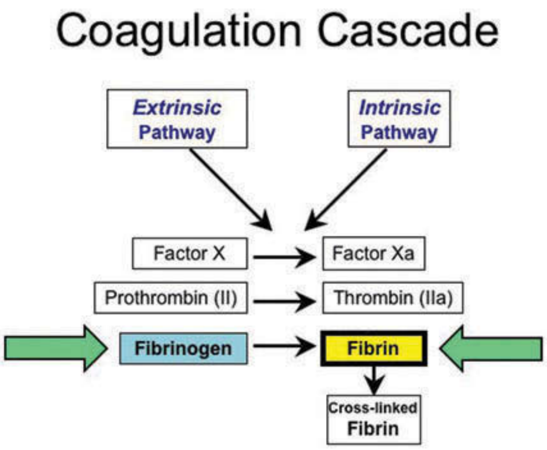

Figure 15.2
Coagulation cascade. The plasma coagulation cascade funnels down to the conversion of soluble fibrinogen to insoluble fibrin.

action is factor X. Heparins exert their anticoagulant effects by facilitating the activity of antithrombin III. Similarly, *Proteins C and S* are important endogenous anticoagulants. Along with procoagulant factors II, VII, IX and X, activation of proteins C and S is dependent on vitamin K. Therefore, initiation of a vitamin K antagonist (i.e., warfarin) results in inactivation of both procoagulant (II, VII, IX, X) as well as anticoagulant (proteins C and S) factors. Antagonism of endogenous anticoagulants (proteins C and S) may paradoxically lead to a transient *pro*coagulant state (warfarin has very rapid effects on proteins C and S, and comparatively slower effects on the other factors). For this reason, alternate anticoagulants (i.e., heparins, such as enoxaparin) are frequently recommended in the initial phase of warfarin therapy as a bridge to warfarin-mediated anticoagulant effect. Finally, *plasmin* is an endogenous fibrinolytic agent.

Red flags

Emergency clinicians must be adept at recognizing “red flags” (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 15.1).

History

A diligent and focused history in a patient presenting with bleeding is the key to formulating an appropriate differential diagnosis and management plan. Key questions for the patient are as follows:

From where are you bleeding?

If a patient presents with the complaint of active bleeding, it is important to determine whether hemorrhage is internal (e.g., gastrointestinal [GI] source), external (e.g., skin laceration), or a combination. However, many patients with disordered hemostasis may not be actively bleeding. The emergency clinician must appropriately diagnose any abnormalities in hemostasis, and initiate appropriate therapies to prevent bleeding or other complications (e.g., an entirely asymptomatic patient presenting with an elevated international normalized ratio [INR] level). Important associated questions include bleeding during brushing teeth (which may be present with platelet disorders) and recent changes in the color or consistency of stool.

When did the bleeding begin?

Distinguishing between new onset symptoms and subacute or chronic symptoms is important. For instance, the patient presenting with new-onset melanotic stools (i.e., acute upper GI bleed) necessitates greater urgency than the stable patient with an incidental, long-standing complaint of blood-tinged tissue paper when straining for bowel movements (i.e., external hemorrhoid).

How much have you bled?

It is important to note that patient and bystander accounts of quantity of blood loss are notoriously unreliable. Additionally, the quantity of hemorrhage has different

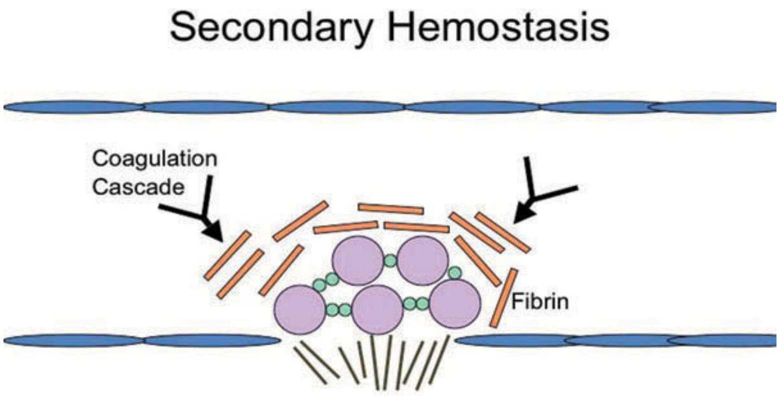

Figure 15.3
Secondary hemostasis. Cross-linked fibrin cements and stabilizes the platelet plug formed at the site of endothelial injury.

Table 15.1 Bleeding red flags

History	**Concerning diagnoses**
Warfarin/other antithrombotic agent use, congenital bleeding diathesis (e.g., hemophilia), or thrombocytopenia/platelet dysfunction (disordered hemostasis)	Include internal bleeding in your differential of any presenting complaint
Disordered hemostasis:	
and altered mental status/confusion	Intracranial hemorrhage Consider TTP if low platelet count
and any form of head trauma or injury	Intracranial hemorrhage
and melena, bloody stools or hematemesis	Acute gastrointestinal hemorrhage
Fatigue, dyspnea, chest pain, presyncope or syncope in a bleeding patient	Critical anemia
Low platelets:	
and otherwise asymptomatic	ITP or medication-associated thrombocytopenia
and recent heparin use	HIT
and neurological symptoms	TTP
and recent afebrile bloody diarrhea	HUS
in setting of sepsis/infection, trauma or malignancy	DIC
and pregnant	HELLP syndrome
Examination finding	**Concerning diagnoses**
Petechiae/purpura	Thrombocytopenia until proven otherwise
Ecchymoses	Elevated INR or elevated aPTT until proven otherwise
Disordered hemostasis:	
and focal neurological findings	Intracranial hemorrhage
and abdominal tenderness	Intra- or retroperitoneal hemorrhage
Low platelets:	
and renal insufficiency/failure	TTP or HUS
and microangiopathic hemolytic anemia seen on peripheral smear	TTP or HUS
and thrombosis	HIT
and elevated INR/aPTT, decreased fibrinogen	DIC

aPTT: activated partial thromboplastin time; DIC: disseminated intravascular coagulation; HELLP: hemolysis, elevated liver function tests, low platelets; HIT: heparin-induced thrombocytopenia; HUS: hemolytic uremic syndrome; INR: international normalized ratio; ITP: idiopathic thrombocytopenic purpura; TTP: thrombotic thrombocytopenic purpura.

meanings in different patients, based largely on comorbid factors and baseline status. An otherwise healthy young adult can generally tolerate blood loss much more effectively that an elderly patient with a history of congestive heart failure or coronary artery disease.

Have you ever had bleeding like this before?

Determining whether the symptoms are new or recurrent may provide important diagnostic clues. Patients with underlying medical conditions or who take particular medications may be prone to recurrent bleeding episodes.

Have you ever had any abnormal bleeding before?

It is important to inquire about bleeding history. For instance, asking “have you ever been told by a health care provider that you had excessive bleeding following surgery or a dental procedure?” may provide clues to the possibility of an undiagnosed underlying congenital bleeding disorder (such as mild or moderate von Willebrand’s disease).

What underlying medical problems do you have?

A thorough investigation of a patient’s medical history, including medication use, has both diagnostic and treatment implications. As for diagnosis, a patient with known colonic diverticular disease is at heightened risk for acute lower GI bleeding. Likewise, an underlying history of congestive heart failure, although not necessarily linked to bleeding, has important implications in bleeding treatment, such as the potential for volume overload when transfusing blood products.

What medications are you taking?

It is critical to elicit a history of antithrombotic medication use, including the use of over-the-counter analgesic agents or herbal remedies that may have antithrombotic properties (Table 15.2).

Table 15.2 Antithrombotic medications

Category	Drug class	Representative medications
Anticoagulants (principle coagulation factor(s) inhibited listed in parenthesis)	Vitamin K antagonists (II, VII, IX, X) Heparins (Xa, others) Pentasaccharides (Xa) Direct thrombin inhibitors (II)	Warfarin UFH, LMWH Fondaparinux, idraparinux Argatroban, dabigatran, bivalirudin, lepirudin
Antiplatelet agents (principle site of action for inhibiting platelet function listed in parenthesis)	NSAIDs (prostaglandin) Thienopyridines (ADP) Pyrimidopyrimidine (cAMP) Glycoprotein IIb/IIIa receptor antagonists	Aspirin Clopidogrel, ticlodipine Dipyridamole Eptifibatide, Tirofiban, Abciximab
Fibrinolytics	Plasminogen activators	Alteplase, reteplase, tenecteplase

ADP: adenosine diphosphate; cAMP: cyclic adenosine monophosphate; LMWH: low-molecular-weight heparin; NSAIDs: nonsteroidal antiinflammatory drugs; UFH: unfractionated heparin.

Have you experienced any recent trauma?

This is a very important consideration when evaluating either internal or external bleeding.

Have you ever received a blood transfusion in the past?

This simple question may elicit important historical information that may not have been reported otherwise. In addition, history of very recent transfusion may affect other hemostatic parameters, including dilution of platelets or coagulation factors with recent packed red blood cell transfusion.

Associated symptoms

Systemic symptoms

It is critical to ask about systemic findings in any patient with bleeding. This is particularly important in the workup of certain platelet disorders, which may present with systemic symptoms (e.g., the presence of fever typically seen with thrombotic thrombocytopenic purpura [TTP]). In addition, complaints such as fatigue, dyspnea, chest pain, lightheadedness or syncope may be suggestive of anemia from blood loss.

Mental status

One of the most devastating consequences of bleeding is the development of intracranial hemorrhage. Therefore, it is crucial to inquire about changes in mental status. This information can often be elicited directly from the patient, whereas at times it can only be obtained indirectly from others (e.g., family members, friends, bystanders, or emergency medical services personnel).

Skin

Always ask patients if they have noticed a new rash or skin lesion(s). Thrombocytopenia may lead to the development of petechiae or purpura (Figure 15.4). Likewise, disorders of primary or secondary hemostasis may lead to the development of skin ecchymoses.

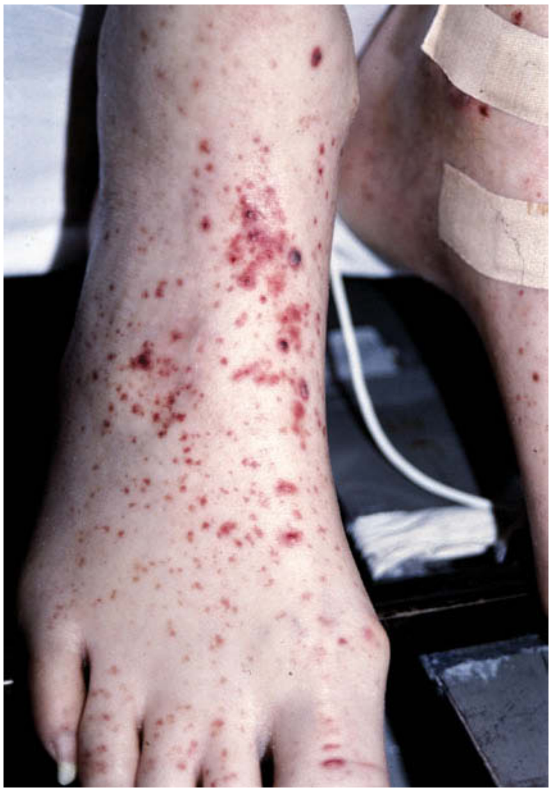

Figure 15.4
Petechiae and purpura. Courtesy: Steven Shpall, MD.

Past medical

Hemostatic abnormalities may result from acute or chronic disease states or medication administration. Chronic liver and renal disease may be associated with bleeding. Liver disease affects both primary (thrombocytopenia, platelet dysfunction) and secondary (deficiency in generation of essential plasma coagulation proteins) hemostasis. Renal disease typically affects primary hemostasis (uremic platelet dysfunction).

In addition, a thorough medication history should include all agents that may be associated with bleeding, including over-the-counter products such as analgesics (aspirin, nonsteroidal antiinflammatory drugs [NSAIDs]), vitamins and herbal remedies, in addition to prescription antithrombotic and antimicrobial agents.

Physical examination

General appearance

General appearance (including skin color, diaphoresis, anxiety and restlessness) provides important diagnostic clues in the evaluation of both external and internal bleeding.

Vital signs

Determination of hemodynamic stability is crucial in a patient with any form of blood loss. Following significant blood loss, tachycardia with or without hypotension may be noted. Importantly, the anticipated tachycardic response to blood loss may be blunted in patients taking beta-adrenergic blocking medications. Also, significant intraperitoneal hemorrhage may at times cause relative bradycardia from vagal stimulation.

Mucous membranes

Assess for the presence of petechiae, which may be a diagnostic clue to a platelet disorder.

Abdomen

Abdominal tenderness may be present in the case of intra-abdominal bleeding. Similarly, flank or costovertebral angle tenderness may be present with retroperitoneal bleeding. Identification of stool positive for the presence of occult blood is important in the evaluation of suspected internal bleeding.

Skin

Identification of petechiae, purpura, or ecchymoses may provide important diagnostic clues to the presence of a hemostatic disorder.

Neurologic

Perform a comprehensive neurologic examination, including assessment of mental status, sensory, motor and cerebellar function. Focal neurologic findings may indicate central nervous system hemorrhage.

Special signs/techniques

There are a few adjuncts to the traditional examination in assessing the bleeding patient. One useful technique involves the use of a glass slide or blood collection tube to distinguish non-blanching petechiae from other blanching skin lesions (Figure 15.5).

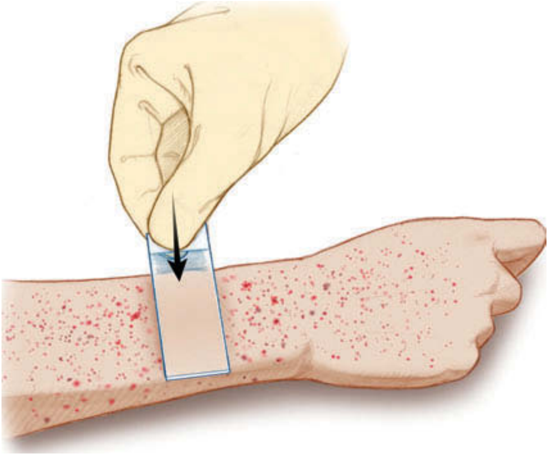

Figure 15.5
Slide test for blanching skin lesions. © Chris Gralapp.

Differential diagnosis

Table 15.3 provides a list of possible bleeding etiologies, classified as involving either primary or secondary hemostasis.

Diagnostic testing

Important initial laboratory studies in the evaluation of the bleeding patient include the complete blood count (CBC) with peripheral smear (assessing for signs of microangiopathic hemolytic anemia [MAHA]) and a plasma coagulation profile. The typical plasma coagulation profile measures the prothrombin time (PT), partial thromboplastin time (PTT) and the INR.

Other critical studies may include a fibrinogen level or measurement of fibrin breakdown products, particularly in the evaluation of disseminated intravascular coagulation (DIC).

Additional laboratory studies may be important depending on the particular condition, including measurement of bleeding time (assesses platelet functional *quality*) or single coagulation factor assays. However, the impact of these additional studies on real-time clinical decisions in the ED setting is often limited.

The importance of additional diagnostic adjuncts (including radiographic imaging) in the evaluation of the bleeding patient varies. The need for additional diagnostic testing is determined on a case-by-case basis.

General treatment principles

As with any ED patient, priorities begin with the ABCs (airway, breathing, circulation). The primary treatment goals in the bleeding patient are physiologic stabilization and hemorrhage control. It is important to note that for many hemostatic disorders, patients may not present

Table 15.3 Differential diagnosis

Diagnosis	Symptoms	Signs	Work-up
Primary hemostasis			
Idiopathic thrombocytopenic purpura (ITP)	Acquired autoimmune disorder (antiplatelet antibodies) Classically presents with otherwise asymptomatic petechiae	Petechiae May present with active bleeding (mucocutaneous most frequent)	Platelet count Exclude other causes, including marrow infiltrative process (such as leukemia)
Drug-induced thrombocytopenia (DIT)	Immune-mediated process May occur with a wide variety of medications	Petechiae May present with active bleeding (mucocutaneous most frequent)	Platelet count Exclude non-drug causes of thrombocytopenia
Heparin-induced thrombocytopenia (HIT)	Platelets paradoxically activated by heparin-antibody complex Paradoxical risk of thrombosis	May present secondary to thrombotic complications	Platelet count: > 50% decline from pre-heparin baseline
Thrombotic thrombocytopenic purpura (TTP)	Microvascular thrombosis May be idiopathic or associated with medication usage	Classic diagnostic pentad: thrombocytopenia, fever, MAHA on peripheral smear, neurologic and renal signs or symptoms (neurologic > renal for TTP)	Platelet count Peripheral smear: look for evidence of MAHA, such as schistocytes or red blood cell fragmentation
Hemolytic uremic syndrome (HUS)	Microvascular thrombosis Classically in the pediatric population Associated with *E. coli* H7:O157 infection	Classic diagnostic pentad: thrombocytopenia, fever, MAHA on peripheral smear, neurologic and renal signs or symptoms (renal > neurologic for HUS)	Platelet count Peripheral smear: look for evidence of MAHA, such as schistocytes or red blood cell fragmentation
Disseminated intravascular coagulation (DIC)	Various triggers, such as infection, trauma, pregnancy, malignancy Incited by liberation of tissue factor (pathologic initiation of plasma coagulation cascade)	Bleeding or thrombosis may predominate, depending on the balance of endogenous anti- or procoagulant activity, respectively	Platelet count Coagulation profile DIC panel: fibrinogen level, fibrin degradation product levels
HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome	Pregnancy-associated complication	Thrombocytopenia Hepatopathy Hemolytic anemia	Platelet count Peripheral smear Liver function tests
von Willebrand's disease	Typically a long-standing history of congenital bleeding disorder for severe forms	Mild, moderate, and severe variants of disease	Bleeding time
Medication-associated	Typically a history of antithrombotic use is relayed or elicited	Bleeding may span from nuisance-level to life-threatening May be entirely asymptomatic in the setting of abnormal laboratory values	Varies depending on the particular antithrombotic medication
Secondary hemostasis			
Congenital			
Hemophilia A, B	Typically a long-standing history of congenital bleeding disorder	Typically associated with "deep" bleeding (intra-articular, retroperitoneal, muscular compartment, intracranial, etc.)	Plasma coagulation profile (PT/PTT/INR) Specific factor assays
Acquired			
Medication-associated	Typically a history of antithrombotic use is relayed or elicited	Bleeding may span from minor to life-threatening May be entirely asymptomatic in the setting of abnormal laboratory values	Varies depending on the particular antithrombotic medication

INR: international normalized ratio; MAHA: microangiopathic hemolytic anemia; PT: prothrombin time; PTT: partial thromboplastin time.

with active bleeding. However, recognition and treatment of the underlying disorder remains critical in preventing associated morbidity and mortality (Table 15.4).

Table 15.4 Treatment of primary and secondary bleeding disorders

Primary hemostasis	**Treatment**
ITP	Steroids, intravenous immunoglobulin; less frequently splenectomy
DIT	Discontinue offending medication
HIT	Discontinue heparin, alternative anticoagulant (direct factor Xa inhibitor or direct thrombin inhibitor)
TTP	Emergent plasma exchange
HUS	Supportive
DIC	As DIC is a "consumptive coagulopathy," treatment involves replenishing deficient hemostatic components (fresh-frozen plasma, cryoprecipitate, platelets)
HELLP syndrome	Delivery
von Willebrand's disease	Desmopressin for mild or moderate; factor replacement for severe disease
Medication-associated	Varies depending on the particular antithrombotic agent (see text and Table 15.2)
Secondary hemostasis	
Congenital	
Hemophilia A, B	Typically factor replacement
Acquired	
Medication-associated	Varies depending on the particular antithrombotic agent (see text and Table 15.2)

DIC: drug-induced thrombocytopenia; DIT: disseminated introvascular coagulation; HELLP: hemolysis, elevated liver function tests, low platelets; HIT: heparin-induced thrombocytopenia; HUS: hemolytic uremic syndrome; ITP: idiopathic thrombocytopenic purpura.

Primary hemostasis

The approach to treatment varies greatly depending on the particular disease process. Platelet transfusion is infrequently utilized for the treatment of many primary hemostatic disorders. Instead, treatment typically targets the specific underlying disease process or condition. A notable exception to this rule is significant bleeding associated with antiplatelet medication use, where platelet transfusion may provide a temporizing bridge to physiologic stabilization and treatment of the underlying bleeding source.

In some conditions, platelet transfusion is a futile endeavor. Such is the case in idiopathic thrombocytopenic purpura (ITP), where transfused platelets are rapidly coated with antiplatelet antibodies and scavenged from the circulation. For other conditions, platelet transfusion may actually exacerbate the underlying disease process. This generally holds true for the thrombotic microangiopathies (TTP, hemolytic uremic syndrome [HUS], and hemolysis, elevated liver enzymes, low platelets [HELLP] syndrome). In these disorders, transfusion of fresh platelets may lead to heightened microvasculatory thrombosis. Similarly, transfused platelets may provide more substrate for thrombosis in heparin-induced thrombocytopenia (HIT), or in cases of DIC where thrombosis predominates the clinical picture. A general exception, however, is the accepted use of platelet transfusion for many of these conditions when there is associated life-threatening hemorrhage (e.g., intracranial). It is advisable to consult a hematologist prior to the routine use of platelet transfusion in many low platelet disorders.

Secondary hemostasis

The approach to treatment differs significantly depending on the underlying cause. Treatment of hemophilia with bleeding is targeted on the specific factor deficiency. The general emergency treatment principle is to replace the deficient factor to 100% factor activity level when there is moderate or significant bleeding. If specific factor concentrates are not readily available, fresh-frozen plasma (FFP) or cryoprecipitate (rich in von Willebrand factor and factor VIII) can be utilized as a temporizing measure.

Antithrombotic medication-associated bleeding

The treatment of antithrombotic medication-associated bleeding is covered in Table 15.5. Specific guidelines for the management of an elevated INR level associated with warfarin therapy are presented in Table 15.6. Regardless of the particular medication involved, allowing the antithrombotic state to resolve on its own over time is the most frequently employed initial approach to antithrombotic reversal, particularly when bleeding is not life-threatening. In addition, because a lack of evidence for improved outcomes and many reports of untoward events exist, recombinant factor VIIa (rFVIIa) should be reserved for patients with serious or life-threatening bleeding (resulting from antithrombotic medications) only after more conventional therapies have failed.

Massive transfusion

Massive transfusion is typically defined as the transfusion of 10 or more units of red blood cells (RBC) in less than 24 hours. Replacement of such a significant proportion of blood volume dilutes circulating platelets and coagulation factors, and can lead to coagulopathy which may be difficult to correct. Infusion of crystalloid further contributes to this dilutional coagulopathy. As such, massive transfusion protocols utilizing fixed ratios (such as 1:1:1 RBC:plasma:platelets) have been adopted by many centers to treat massive ongoing hemorrhage, such as from trauma or GI bleeding.

Table 15.5 Reversing antithrombotic agents

Category	Antithrombotic medication	Reversal agent(s)
Anticoagulants	Warfarin UFH LMWH	Vitamin K, FFP, PCC, possibly rFVIIa Protamine, possibly rFVIIa Possibly protamine or rFVIIa
Antiplatelet agents	Aspirin, NSAIDs, glycoprotein IIb/IIIa receptor antagonists	Desmopressin (DDAVP), platelets, possibly rFVIIa
Fibrinolytics	Fibrinolytic agents (i.e., alteplase)	Cryoprecipitate, FFP, aminocaproic acid, possibly rFVIIa

DDAVP: deamino-D-arginine vasopressin; FFP: fresh-frozen plasma; LMWH: low-molecular-weight heparin; NSAIDs: nonsteroidal antiinflammatory drugs; PCC: prothrombin complex concentrate; rFVIIa: recombinant factor VIIa; UFH: unfractionated heparin.

Table 15.6 Treatment of warfarin over-anticoagulation

INR	No significant bleeding	Serious or life-threatening bleeding
3–5	Omit 1 warfarin dose	Regardless of INR, treat with:
5–9	Omit 1–2 warfarin doses (1–5 mg PO vitamin K)[a]	Hold warfarin 10 mg IV vitamin K FFP, PCC, or rFVIIa
>**9**	Hold warfarin (2.5–5 mg PO vitamin K)[a]	

FFP: fresh-frozen plasma; IV: intravenous; PCC: prothrombin complex concentrate; PO: oral; rFVIIa: recombinant factor VIIa.

[a](treatment option) – consider use of the treatment option when listed in parentheses.

Table adapted from *Chest* 2008;133:6(suppl):160S.

Special patients

Geriatric

Older patients may present with seemingly innocent bleeding, which may be a harbinger of more sinister underlying pathology. As such, maintain a lower threshold for checking the platelet count and plasma coagulation profile (PT/PTT/INR) in patients with seemingly straightforward bleeding presentations such as epistaxis. Ideally, these decisions should be made on a case-by-case basis. In addition, it is prudent to exercise heightened caution when treating bleeding disorders in the setting of underlying comorbid conditions.

Pediatric

ITP and HUS are more likely to be encountered in the pediatric population. It is important to check a platelet count in the evaluation of the child presenting with petechiae. In the asymptomatic thrombocytopenic pediatric patient, ITP is commonly the culprit. However, the finding of a low platelet count necessitates further evaluation for a potentially devastating infiltrative marrow process, such as acute leukemia. One of the principle treatments for ITP is steroid administration. However, even a single dose of steroids may send an undiagnosed aggressive hematologic malignancy into transient remission, rendering diagnosis via bone marrow biopsy difficult. Such a "missed" diagnostic window could be catastrophic. Pediatric hematologists typically prefer to be involved in the evaluation of the thrombocytopenic patient, even those who are symptom-free, prior to initiation of any therapies.

HUS tends to present with systemic symptoms. However, in afebrile children with bloody diarrhea, consider the possibility of *Escherichia coli* H7:O157 infection, which has been associated with the development of HUS in the period immediately following the acute GI infection.

Disposition

The primary goals in the management of the bleeding patient presenting to the ED are hemodynamic stabilization, bleeding control, and identification of serious or life-threatening underlying hemostatic defects.

Many patients found to have underlying disorders of primary or secondary hemostasis are admitted for further work-up and initiation of disease-specific therapies.

Patients whose bleeding has been alleviated or controlled are candidates for discharge from the ED, provided they are hemodynamically stable and serious underlying conditions affecting hemostasis have been excluded. For these patients, follow-up is typically scheduled with a primary care provider and a hematologist.

Pearls, pitfalls and myths

- In life-threatening hemorrhage, immediate treatment takes precedence over precise diagnosis. Pay attention to the ABCs, assess and re-assess vital signs, and apply direct pressure to halt external bleeding.

- A low platelet count should trigger a mental rundown of the "can't-miss" conditions: ITP, HIT, TTP, HUS, DIC and HELLP syndrome.
- A missing platelet count (e.g., the remainder of the complete blood count results are released, with the platelet count still "in process") should be treated as a low platelet count until demonstrated otherwise.
- The American College of Chest Physicians (ACCP) has published guidelines on the recommended approach to the treatment of an elevated INR level associated with warfarin therapy. Treatment is based on the INR level and the degree of bleeding (mild to life-threatening).
- Vitamin K is often recommended for the treatment of an elevated INR level associated with warfarin use. Recommendations include administration of Vitamin K via the oral or intravenous route, as subcutaneous absorption is erratic; intramuscular use is discouraged because of the risk of hematoma formation.
- Recombinant factor VIIa should be considered for bleeding control of carefully selected conditions when conventional therapies have failed. It has been associated with a number of deleterious consequences, including thrombosis.
- Other than extreme cases, patient (or bystander) accounts of the quantity of blood loss (whether at the site of traumatic injury or in the toilet bowl) are notoriously unreliable.

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16 Burns

David A. Wald, DO

Scope of the problem

It is estimated that fire and burn injuries account for over 1 million annual emergency department (ED) visits in the United States. The majority of these injuries are managed on an outpatient basis; however, nearly 60,000 patients are hospitalized in the United States each year. Despite the advances made in health care over the past 20 years, the mortality rate from fire and burns in the United States remains among the highest of all industrialized nations. Even with smoke and carbon monoxide (CO) detectors in homes, fire and burns are the fifth leading cause of death from unintentional injury in the United States, and the third leading cause of injury-related death in the home.

Residential fires are the leading cause of fire-related death and account for approximately 75% of fire-related injuries. Cooking is by far the leading cause of residential fires, whereas smoking is the leading cause of residential fire deaths, accounting for approximately 25% of fatalities. The combination of careless smoking and alcohol abuse accounts for nearly half of all fire-related deaths.

Anatomic essentials

Traditionally, burns have been described as first, second (partial thickness), or third degree (Figure 16.1). Superficial burns (formerly referred to as first-degree burns) injure only the epidermis. Superficial burns do not damage the dermal–epidermal junction (basement membrane), and thus spare deeper skin structures responsible for re-epithelialization (i.e., epidermal appendages, hair follicles, sweat and sebaceous glands). Superficial burns can be painful, and are characterized primarily by erythema and the lack of blisters. Skin peeling may be seen as erythema fades. Superficial burns heal without scar formation in 3–5 days and should not be included in the total body surface area (TBSA) calculation for initial fluid resuscitation requirements.

Second-degree burns are now commonly referred to as either superficial or deep partial-thickness burns. Superficial partial-thickness burns extend through the epidermis into the papillary dermis, injuring pain-sensitive nerve endings. These burns have intact sensation and are painful. Blisters or bullae are common; these burns usually

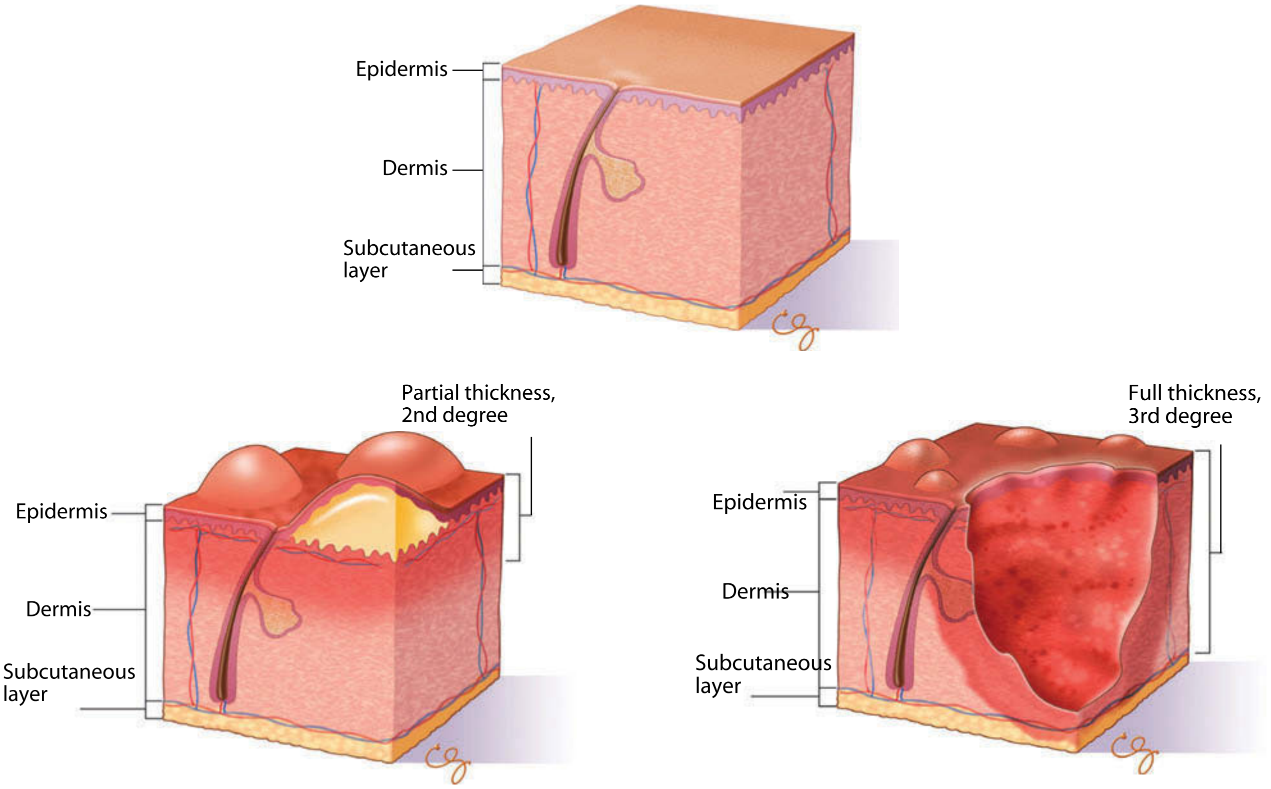


Figure 16.1
A. Anatomy of the skin. **B.** Partial-thickness burn. **C.** Full-thickness burn. © Chris Gralapp.

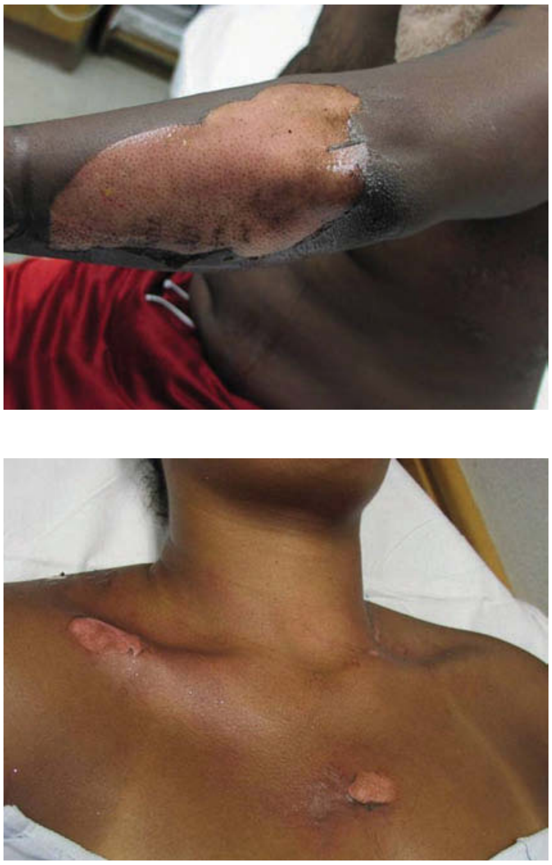


Figure 16.2
A. Superficial partial-thickness scald burn of the arm.
B. Superficial partial-thickness scald burn of the upper chest.

appear pink and moist, and blanch to touch because capillary refill is preserved (Figure 16.2). Because the deeper rooted epidermal appendages are spared, superficial partial-thickness burns typically heal within 2–3 weeks with little or no scarring.

Deep partial-thickness burns extend through the epidermis and damage both the papillary and reticular dermis. These injuries may or may not be painful and often appear white, mottled pink or cherry red (Figure 16.3). Deep partial-thickness burns have impaired sensation and do not blanch to touch. In the immediate post-burn period, deep partial-thickness burns can be difficult to distinguish from full-thickness injury. Because the epidermal appendages located in the reticular dermis are damaged, the skin has a limited ability to re-epithelialize and often takes 3 or more weeks to heal. Burns of this depth often result in hypertrophic scarring if left to heal spontaneously. It is important to keep in mind that without proper care, some deep partial-thickness burns will progress to full-thickness burns in the first few days post injury.

Full-thickness burns (formally referred to as third-degree burns) involve all layers of the epidermis and dermis, and may extend into subcutaneous structures. These burns usually appear white or charred (Figure 16.4). Full-thickness burns are usually insensate due to the destruction of the nerve endings; however, surrounding areas may be painful. All but the smallest full-thickness burns are treated with skin grafting to help limit the development of hypertrophic scarring. Some references in the literature may describe

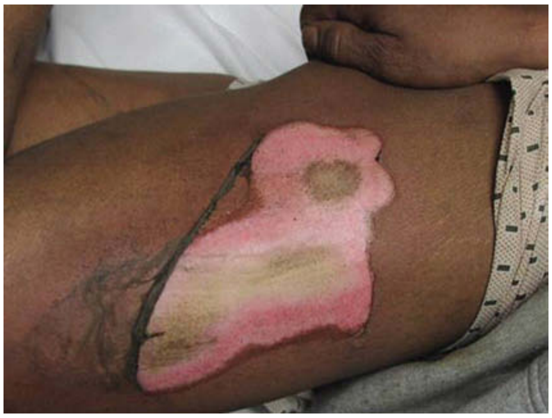


Figure 16.3
Deep partial-thickness burn from contact with a hot radiator. The central area of contact with the radiator is of deeper depth than the periphery of the burn.

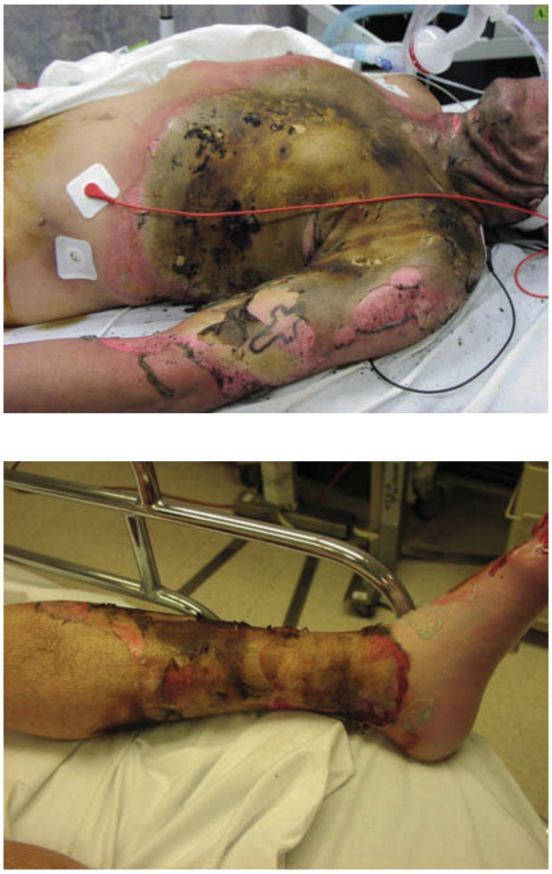


Figure 16.4
A. Full-thickness flame burn of the chest, neck and upper arm.
B. Full-thickness flame burn of the lower leg.

fourth-degree burns which involve deeper structures (i.e., muscle and bone).

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 16.1).

History

A focused history should be performed on all burn patients. Key historical information may heighten the suspicion for associated injuries and can influence management.

When did the burn occur?

Determining the timing of a burn is important for patients requiring IV fluid resuscitation. Delays in initiating fluid resuscitation increase fluid requirements, and delays greater than 2 hours after burn injury are associated with increased mortality. Determining the time of injury is important when evaluating acute or subacute burn injuries, as the depth of certain deep burns will not be evident in the immediate post-burn period.

What were the circumstances surrounding the injury?

Information regarding the circumstances surrounding the injury should be obtained from the patient, family, witnesses, or prehospital care providers. Was the injury caused by hot tap water, grease, or another hot liquid? Was the injury caused by flame or contact with a hot object? Were chemicals or industrial solvents involved? Details regarding the mechanism of injury may suggest the depth of burn injury and alert physicians to potential associated injuries. The patient's condition at the scene and any treatment provided by bystanders or an outside referring hospital should also be sought.

Was the patient confined in a burning environment, or was there a history of an explosion?

Patients involved in a closed space fire or an explosion are at risk for inhalation injury. In addition, a history of an explosion places patients at risk for barotrauma and blast-related injuries.

Did the patient sustain associated traumatic injuries?

Associated trauma may be masked in a patient with a significant burn injury. All "burn" patients should undergo a systematic trauma assessment. If the patient was in a house fire, it is important to find out how the patient got out of

Table 16.1 Burns red flags

History	Concerning diagnosis
Burns in a confined space	Inhalation injury
Explosion	Barotrauma or other associated trauma
Difficulty speaking, swallowing or drooling	Upper airway burns or inhalation injury
Coughing, wheezing or difficulty breathing	Inhalation injury
Headache, dizziness, or history of loss of consciousness at the scene	Carbon monoxide toxicity
Burn wound inconsistent with history	Non-accidental trauma
Preexisting medical conditions (HIV/AIDS, renal disease, liver disease, and metastatic cancer)	Increased morbidity, mortality and length of hospitalization
Examination finding	**Concerning diagnosis**
Tachypnea	Airway burn
Hypotension	Volume depletion or other associated significant trauma
Burns to the face, neck or upper torso; singed eyebrows and nasal vibrissae; carbon deposits and acute inflammatory changes in the oropharynx; carbonaceous sputum	Airway burns or inhalation injury
Burns that are painful and characterized primarily by erythema and lack of blisters	Superficial burns
Burns that are painful and usually appear pink, moist, with blisters or bullae	Superficial partial-thickness burns
Burns that appear white, mottled pink or cherry red with impaired sensation	Deep partial-thickness burns
Burns that appear white or charred with impaired sensation	Full-thickness burns

the building (i.e., did they jump out of a window or were they found in bed). Burns associated with motor vehicle collisions are often associated with traumatic injuries.

Is the burn painful?

Typically, all superficial partial thickness burns are painful. Deeper burns, such as deep partial-thickness and full-thickness burns, are often less painful or painless, respectively.

Were there suspicious circumstances surrounding the burn injury?

It is important to consider that burn injuries might be self-inflicted, perhaps in a suicide attempt or gesture. Additionally, toxic ingestions or potentially lethal overdoses can occur prior to setting oneself or a building on fire.

Is the burn pattern consistent with the explanation?

Burns with a clear line of demarcation, or located on the buttocks, between the child's legs or other areas that would be difficult for the child to reach should be very concerning for burns inflicted by another individual (abuse). Concurrent injuries or bruises at different stages of healing are also suspicious for child abuse, which is far more common than most physicians believe. Additional information suggestive of non-accidental burn injury includes delays in seeking care, a pattern of burn injury inconsistent with the child's motor abilities, or witness stories that do not correlate or seem possible. Abuse should be considered when caregivers appear angry, resentful toward the child, or even overly protective or afraid of letting their child speak to physicians alone. In other words, the possibility of abuse should be considered in all traumatic injuries in children, especially burns.

Associated symptoms

Head, eyes, ears, nose and throat (HEENT)

Ask about difficulty speaking or swallowing? Difficulty speaking or swallowing, or pain during these activities suggests an upper airway burn or inhalation injury, which may portend future airway compromise. Any voice change (including hoarseness) may indicate injury to or edema of the larynx and vocal cords.

Ask about difficulty with vision, such as blurred vision, photophobia, or pain? Visual complaints suggest ocular involvement, including burns, abrasions, or edema of the cornea. Foreign bodies (including penetration of the globe or orbit from flying debris) may accompany some burn injuries or explosions.

Pulmonary

Ask about coughing, wheezing, or trouble breathing? These respiratory complaints suggest the possibility of lung inhalation injury.

Neurologic

Ask about headache, dizziness, or a history of loss of consciousness at the scene? In the context of a burn injury or fire exposure, these symptoms suggest concomitant CO toxicity. Complaints of CO toxicity are often subtle, so a high index of suspicion must be maintained.

Past medical

After controlling for patient age, sex, TBSA burned and the presence of inhalation injury, several preexisting medical conditions are associated with increased mortality risk and length of hospital stay in patients with an acute burn injury. Preexisting medical conditions with the highest mortality risk include HIV/AIDS, renal disease, liver disease and metastatic cancer. Other conditions independently associated with an increased mortality risk in the burn patient are pulmonary circulation disorders, congestive heart failure, obesity, non-metastatic malignancies, alcohol abuse, peripheral vascular disorders, cardiac dysrhythmias and patients with neurologic disorders. Several conditions are associated with prolonged hospital stays but no increase in mortality. These include paralysis, dementia, peptic ulcer disease, psychiatric illness, cerebrovascular disease, valvular disease, diabetes, drug abuse and hypertension. The presence of one or more preexisting comorbidities may warrant hospital admission or transfer to a regional burn center for patients with otherwise minor or moderate size burns.

Physical examination

When evaluating a burn patient, the physical examination should be performed in a systematic fashion (as for all trauma victims). After life-threatening conditions are identified and addressed, the emergency physician should determine the depth and TBSA of the burn.

Vital signs

Major burns lead to a hyperdynamic state commonly associated with tachycardia. For this reason, the heart rate should not be used in isolation as a reliable indicator of volume status. Tachypnea may also indicate a hyperdynamic state or airway involvement. If hypotension is present, volume depletion resulting from third spacing of fluids or associated trauma should be given high priority.

Primary and secondary surveys

The physical examination begins with the primary survey. Immediate life-threats should be addressed first, which may be difficult given the dramatic nature and overpowering odor of burns. Additional attention should be given to clinical findings associated with inhalation injury, such as burns to the face, neck or upper torso; singed eyebrows and nasal vibrissae; carbon deposits

Table 16.2 A bedside assessment of burn depth

	Superficial burn	Superficial partial-thickness burn	Deep partial-thickness burn	Full-thickness burn
Bleeding on pin prick testing	Brisk	Brisk	Delayed	None
Sensation	Painful	Painful	Dull	None
Appearance	Light red, dry	Moist, pink	Mottled pink-red or waxy white	White, charred, dry
Blanching to pressure (capillary refill)	Brisk	Slow return	None	None

Testing for bleeding and sensation can be performed with a 21-gauge needle. Testing for blanching can be performed with a sterile cotton tip swab.

Adapted from Hettiaratchy S, Papini R. Initial management of a major burn: I – Overview. *BMJ* 2004;328(7455):1555–7.

and acute inflammatory changes in the oropharynx; and carbonaceous sputum. In addition, any change in voice quality, stridorous respirations, wheezing, hoarseness, or drooling should alert physicians to the probability of airway involvement. While performing the secondary survey, emergency physicians should closely examine the entire patient to determine the depth and TBSA of the burn, in addition to identifying associated traumatic injuries.

Determination of burn depth and TBSA burned

Burn depth and TBSA determination will guide initial fluid resuscitation volume and the need for hospitalization or transfer to a regional burn center. In addition, burn wound classification includes identification of preexisting medical conditions, associated trauma, inhalation injury, and unusual circumstances such as consequences or location of the burn. For the purpose of fluid resuscitation, no distinction is made between partial-thickness and full-thickness burns.

Even experienced clinicians and burn specialists are not always able to differentiate between deep partial-thickness and full-thickness burns at the time of injury. First of all, many burns are not uniform in depth. Furthermore, burns of similar depth may not look alike due to differences in underlying skin pigmentation. When evaluating the depth of a burn, the age of the patient also needs to be considered, as children less than 2 years of age and the elderly have thin skin (dermis). As a result, patients at the extremes of age may have full-thickness injury following an exposure (e.g., hot tap water) that might only cause a partial-thickness injury in an older child, adolescent or adult.

Traditionally, burn wound classification has emphasized the distinction between partial- and full-thickness injury (previously referred to as second- and third-degree burns). In the immediate post-burn period, it may be more clinically relevant to distinguish between superficial partial thickness and all deeper (deep partial-thickness and full-thickness) burns. For patients with moderate to major burns, this distinction does not affect initial fluid resuscitation requirements or the need for hospitalization. However, distinguishing between superficial partial-thickness and all deeper burns may help reduce hospital transfers of some patients with minor burns. Bedside testing may help emergency physicians differentiate between burns of different depth, specifically between superficial partial-thickness and all deeper burns. A clinical approach to evaluating burn depth at the bedside is provided (Table 16.2).

In adults, the TBSA estimation of the burn is commonly based on the *rule of nines* (Figure 16.5). Although adequate for adults, application of this rule can lead to inaccurate burn size estimations in infants and small children, who have larger surface body area-to-weight ratios than older children, adolescents, or adults. A more accurate estimation of TBSA burned can be obtained using the Berkow or Lund-Browder burn size chart (Figure 16.6). These formulas estimate burn size based on age and

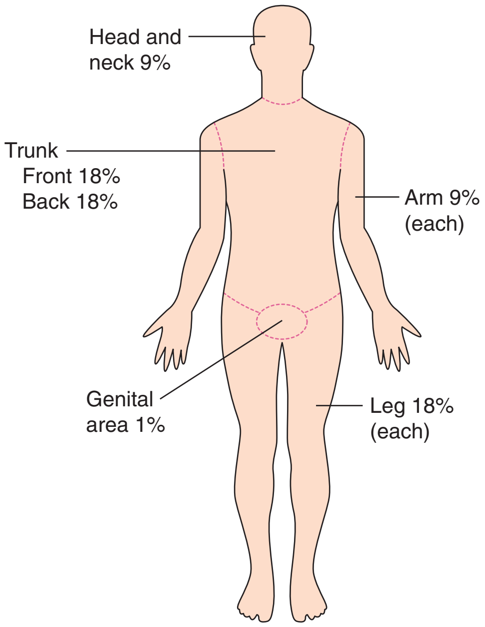
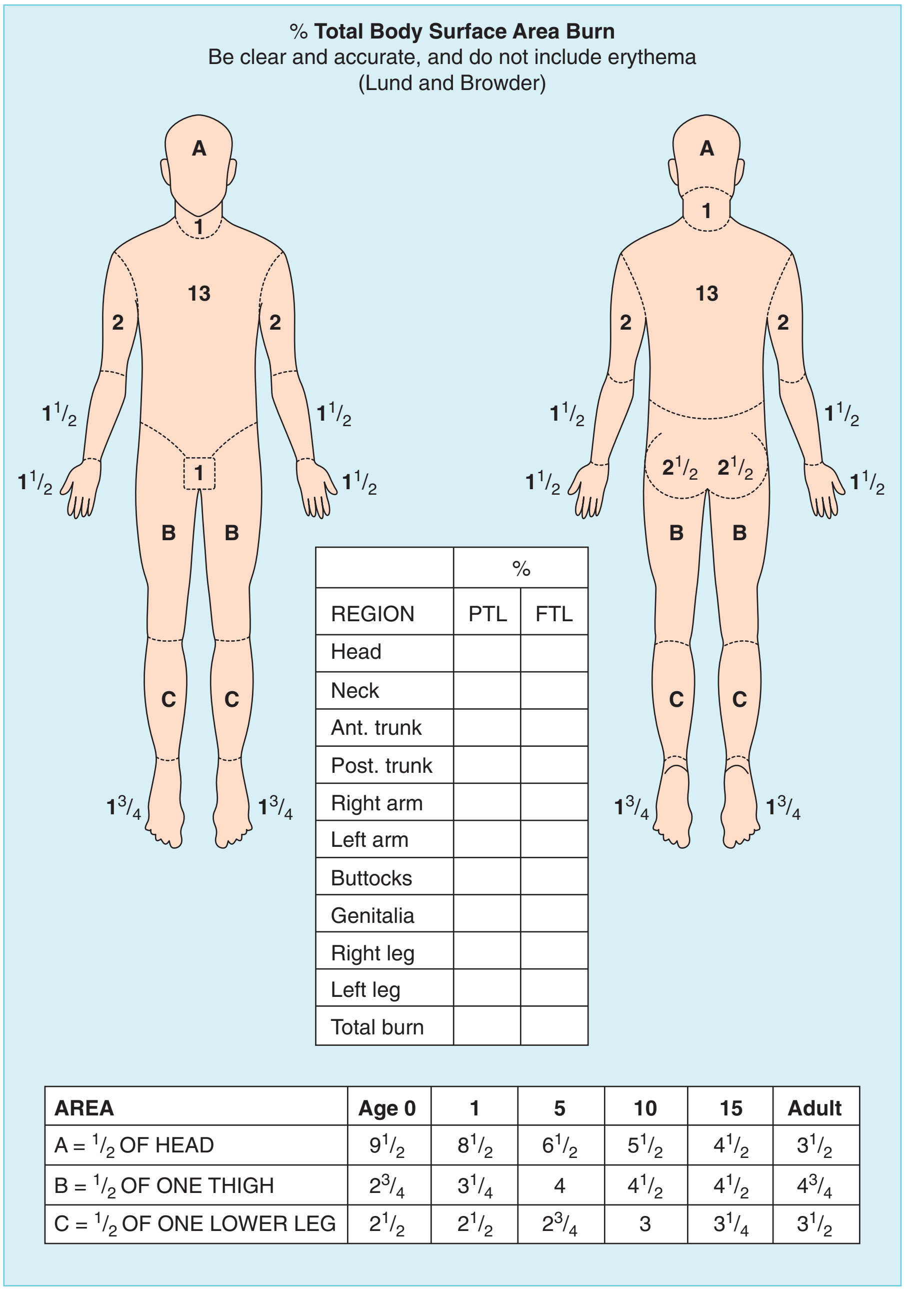


Figure 16.5
Rule of nines.


% **Total Body Surface Area Burn**
Be clear and accurate, and do not include erythema
(Lund and Browder)
A
1
13
2
2
$1\frac{1}{2}$
$1\frac{1}{2}$
$1\frac{1}{2}$
$1\frac{1}{2}$
1
B
B
C
C
$1\frac{3}{4}$
$1\frac{3}{4}$
A
1
13
2
2
$1\frac{1}{2}$
$1\frac{1}{2}$
$2\frac{1}{2}$
$2\frac{1}{2}$
$1\frac{1}{2}$
$1\frac{1}{2}$
B
B
C
C
$1\frac{3}{4}$
$1\frac{3}{4}$

	%	
REGION	PTL	FTL
Head		
Neck		
Ant. trunk		
Post. trunk		
Right arm		
Left arm		
Buttocks		
Genitalia		
Right leg		
Left leg		
Total burn		

AREA	**Age 0**	**1**	**5**	**10**	**15**	**Adult**
A = $\frac{1}{2}$ OF HEAD	$9\frac{1}{2}$	$8\frac{1}{2}$	$6\frac{1}{2}$	$5\frac{1}{2}$	$4\frac{1}{2}$	$3\frac{1}{2}$
B = $\frac{1}{2}$ OF ONE THIGH	$2\frac{3}{4}$	$3\frac{1}{4}$	4	$4\frac{1}{2}$	$4\frac{1}{2}$	$4\frac{3}{4}$
C = $\frac{1}{2}$ OF ONE LOWER LEG	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{3}{4}$	3	$3\frac{1}{4}$	$3\frac{1}{2}$

Figure 16.6
Burn size estimation based on age and body location.

body location, and take into account the different ratios of the head and lower extremities in childhood compared with adulthood . The size of small burns can also be estimated using the *rule of palms*, which assumes that a burn the size of the patient's palm accounts for roughly 1% of their TBSA. With children, the entire volar surface of the hand more closely approximates 1% TBSA than the palm itself.

Inaccurate assessment of burn depth and size can adversely impact the calculation of the initial fluid resuscitation volume. This can lead to over-resuscitation of small to moderate size burns, and under-resuscitation of larger burns. In addition, overestimation of the size of small to moderate burns may result in unnecessary hospital transfers.

Differential diagnosis

A number of dermatologic conditions may present with skin findings that appear similar to a burn wound (Table 16.3). Some of these conditions are cared for in burn centers, because treatment of skin manifestations, patient handling and associated complications are similar to those of burn victims.

Table 16.3 Dermatologic conditions that appear similar to burns

Diagnosis	Symptoms	Signs	Workup
Pemphigus vulgaris	Mucous membrane lesions are typical and often precede other skins lesions by months	Mucosal erosions in the mouth are common Fluid-filled blisters / bullae can be seen Positive Nikolsky's sign	Diagnosis confirmed by histopathology or direct immunofluorescence
Staphylococcal scalded skin syndrome	Usually begins with fever and generalized erythematous rash	Rash progresses from maculopapular scarlatiniform to desquamating bullous Positive Nikolsky's sign	Diagnosis confirmed by culture and biopsy
Stevens-Johnson syndrome	Typical nonspecific prodrome	Fever is common Skin rash can be maculopapular or vesicular Erythema and bullae can develop Ruptured bullae / skin sloughing leave the patient susceptible to secondary infection	Diagnosis confirmed by biopsy
Toxic epidermal necrolysis	Prodrome typical of fever, cough, sore throat and malaise	Erythematous rash with a purpuric center Lesions coalesce forming bullae Skin sloughing at dermal-epidermal junction Positive Nikolsky's sign	Diagnosis confirmed by biopsy

Diagnostic testing

Diagnostic testing should be used selectively in patients with burns. Cases must be handled on an individual basis, as many minor or moderate burn patients require limited or no diagnostic testing. Routine laboratory studies, such as complete blood count (CBC), basic metabolic profile, and coagulation studies are typically obtained for all burn patients requiring hospital admission or transfer to a regional burn center. Additional laboratory studies may be indicated.

Laboratory studies

Arterial blood gas analysis

Arterial blood gas analysis is typically indicated in patients with suspected inhalation injury or those requiring intubation and mechanical ventilation. Carboxyhemoglobin (COHb) levels help guide therapy in cases of suspected or positive CO toxicity. A venous blood gas sample can determine the COHb level.

Type and screen

Type and screening of blood is recommended in patients requiring burn unit admission, as well as other patients who may require blood products or need an operative intervention.

Toxicology testing

Serum ethanol level or urine toxicology screening may be indicated in certain situations.

Miscellaneous

As clinically indicated, additional laboratory studies such as hepatic function, calcium, phosphorous, magnesium, myoglobin, and creatine phosphokinase should be obtained, especially in large TBSA burns.

Electrocardiogram (ECG)

Cardiac monitoring should be initiated and a baseline ECG should be obtained in a patient with a history of high-voltage electrical injury, those with known heart disease or at risk for cardiac complications, and all patients admitted to an intensive care unit. Adult patients transferred to a burn center should have an ECG reviewed prior to transfer.

Radiologic studies

Imaging studies should be obtained as clinically indicated. Although commonly obtained, chest radiography is not sensitive in detecting inhalation injury, and should not be relied on to determine its presence or guide initial therapy.

Laryngoscopy

In cases of suspected inhalation injury or burns of the airway, the upper airway should be visualized by direct, nasopharyngeal, or fiberoptic laryngoscopy. Additionally, the tracheobronchial tree can be visualized by fiberoptic bronchoscopy, if necessary.

General treatment principles

Prehospital care

Burn management should begin at the scene. Prehospital care providers should carefully remove the patient from the site of injury and perform a primary and focused secondary survey to address life-threatening conditions.

Prehospital care providers should determine the need for any immediate interventions, such as airway control, breathing or circulatory support, and cervical spine immobilization. Large burns should be covered with dry sterile dressings; small or moderate size burns can be covered with cool wet dressings to help relieve pain. Supplemental oxygen should be administered if smoke inhalation is suspected. Intravenous fluid administration should be initiated, preferably at a site that does not involve the burn. Intravenous narcotics should be administered (according to protocol). Whenever possible, individuals with extensive burns should be transported directly to a trauma or regional burn center.

Emergency department care

The resuscitation area should be prepared with dry sterile sheets, appropriate protective covering for medical personnel, and airway equipment for the anticipated arrival of a major burn victim. The ED evaluation should be systematic and be performed as any other trauma scenario, starting with the ABCs (airway, breathing, circulation). The airway is the most important initial consideration in the severely burned patient. Assessment for possible airway involvement in a patient with suspected inhalation injury is vital. After the primary survey and treatment of any immediate life-threats, the emergency physician should perform a detailed trauma–burn secondary survey. This specifically focuses on evaluating for possible smoke inhalation and identifying associated traumatic injuries, followed by an estimation of burn depth and size. This critical step will determine the initial fluid resuscitation volume and influence patient disposition. In the patient requiring transfer to a regional burn referral center, early communication with the burn specialist is recommended.

Inhalation injury

Inhalation injury has been reported in 7% of cases reported to the National Burn Registry, and in approximately one-quarter of patients with burns ≥20% TBSA. The incidence of inhalation injury increases with larger burns, presenting in approximately two-thirds of patients with TBSA burns that exceed 70%. Smoke inhalation accounts for >50% of fire-related deaths; when present, it increases the morbidity and mortality associated with all burn injuries. It also increases the initial fluid resuscitation requirements after thermal injury.

Smoke inhalation can cause both airway injury and systemic toxicity. Injury to the upper airway structures (lips to glottis) occurs from inhalation of superheated gases from flame, smoke, or steam. Thermal injury to the lower airway (subglottic structures) is rare because of reflex closure of the glottis and heat dissipation that occurs throughout the tracheobronchial tree. An exception is inhalation injury resulting from steam, because of its ability to carry approximately 4,000 times as much heat as dry air.

Injury to the lower airway usually occurs as a result of inhalation of toxic gases and particulate matter. This can lead to airflow obstruction from the production of mucosal edema, intraluminal debris, inspissated secretions and bronchospasm, subsequently resulting in a chemical tracheobronchitis. If severe, the clinical picture can resemble adult respiratory distress syndrome (ARDS) and further manifest with decreasing pulmonary compliance, increasing airway resistance, hypoxemia and hypercarbia.

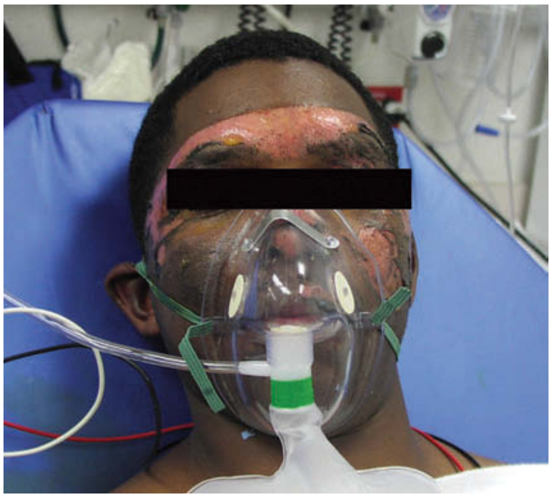


Figure 16.7
Flash burns to the face represent a high risk for an inhalation injury.

Patients suffering from smoke inhalation may also exhibit toxicity from systemic absorption of products of combustion, possibly leading to CO or cyanide toxicity. On arrival, all spontaneously breathing patients with suspected smoke inhalation should immediately be placed on high-flow humidified oxygen. Early intubation should be considered if airway compromise is supported by history and bedside examination. It is crucial to identify which patients with smoke inhalation require early endotracheal intubation. The presence of classic indicators of smoke inhalation (i.e., facial burns, carbonaceous sputum, wheezing, voice change) does not necessarily mandate emergent endotracheal intubation (Figure 16.7). Unfortunately, no group of signs or symptoms can substitute for sound bedside clinical judgment. Furthermore, intubation may become more difficult as edema of the upper airway increases with time. When emergent intubation is not necessary, close observation with frequent serial examinations must be performed, and airway equipment (including equipment for managing the difficult airway) must be available at the bedside.

Carbon monoxide toxicity

CO has an affinity for hemoglobin (Hb) approximately 230 times that of oxygen. COHb decreases the amount of hemoglobin available for oxygen binding and reduces the oxygen-carrying capacity of the blood, leading to impaired tissue oxygenation.

Pulse oximetry is a noninvasive tool that measures functional oxygen saturation. The pulse oximeter cannot distinguish between COHb and oxyhemoglobin, and the presence of COHb produces falsely elevated oxygen saturation readings. This overestimation of oxygen saturation (known as the "pulse oximetry gap") approaches the measured COHb level.

Alternatively, a co-oximeter is a device that analyzes a small blood sample to measure concentrations of oxyhemoglobin, deoxyhemoglobin, COHb, and methemoglobin. Therefore, it can confirm CO toxicity by identifying elevated COHb levels. Traditionally, an arterial blood sample has been used to determine the COHb level; however, a venous blood sample provides a reliable, often less painful alternative. The Masimo Rainbow SET Pulse CO-Oximeter is now available as a noninvasive alternative to screen for CO toxicity.

All patients with known or suspected CO toxicity should receive high-flow oxygen. This can reduce the elimination half-life of COHb (COHb $T_{½}$) from 240–320 minutes at room air to 60–90 minutes. The COHb $T_{½}$ of patients treated with high-flow oxygen by face mask or 100% oxygen if intubated does not appear to be influenced by patient age, gender, history of loss of consciousness, concurrent tobacco use, or initial COHb level.

Hyperbaric oxygen (HBO) therapy has been shown to further reduce the COHb $T_{½}$ to approximately 23 minutes (at three atmospheres with 100% oxygen). Specific selection criteria can identify candidates appropriate for HBO therapy. Patients with myocardial ischemia, cardiac dysrhythmias, neuropsychiatric abnormalities, syncope or persistent neurologic findings in the face of CO toxicity should be considered for HBO therapy. All patients with COHb levels >25% , and pregnant women and young children with levels ≥15% are also candidates for HBO therapy.

It is also important to recognize that patients with similar COHb levels may exhibit varying systemic toxicity. Cyanide toxicity can also complicate severe cases of CO toxicity and should be considered in victims of smoke inhalation with persistent hypotension and acidemia despite adequate arterial oxygenation.

Fluid resuscitation

Fluid resuscitation in the early post-burn period is crucial and should be the top management priority once the airway and other life-threats have been addressed. Over the past 50 years, aggressive volume replacement in the hours immediately following a severe burn has decreased the morbidity and mortality associated with these injuries. The goal of initial fluid resuscitation is to restore and maintain vital organ perfusion and prevent burn shock.

Intravenous fluid resuscitation should be initiated in adults with partial- or full-thickness TBSA burns >20%, in older children with burns ≥15% TBSA, and in infants with burns ≥10% TBSA. Peripheral intravenous access is sufficient for the majority of patients requiring fluid resuscitation. However, in patients with severe burns (>40% TBSA), central venous access using the internal jugular or subclavian vein is the preferred route for fluid resuscitation.

Lactated Ringer's solution is most frequently used for fluid resuscitation of burn patients. In comparison with normal saline solution, which contains sodium 154 mEq/L and chloride 154 mEq/L, Lactated Ringer's solution contains sodium 130 mEq/L, chloride 109 mEq/L, calcium 3 mEq/L, potassium 4 mEq/L and lactate 28 mEq/L. In addition, Lactated Ringer's solution has a higher pH compared with normal saline and more closely resembles physiologic pH (6.5 vs. 5.0, respectively).

The Parkland formula is most commonly used to guide initial fluid resuscitation during the first 24 hours after burn injury. This formula has gained almost universal acceptance, not necessarily for its demonstrated superiority, but more likely because it is easy to remember and use. The Parkland formula calls for the administration of 4 mL/kg of body weight per %TBSA burn (partial- or full-thickness) of intravenous crystalloid fluid over the first 24 hours. Half of the calculated fluid requirement should be administered over the first 8 hours post burn, and the remaining volume over the next 16 hours. Front loading the burn resuscitation fluids over the first 8 hours is required because the early post-burn period is highlighted by increased capillary permeability, protein leak, edema formation and loss of plasma volume.

No resuscitation formulas can accurately predict volume requirements for an individual patient. Therefore, continuous monitoring and reassessment of resuscitation targets such as blood pressure, mental status and urine output is necessary. In adults, a common goal is urine output of at least 0.5 to 1 mL/kg/hr, a reasonable indicator of renal perfusion. However, urine output can be affected by the use of diuretics or the presence of glycosuria (resulting in an osmotic diuresis). Greater urine output may be needed in the presence of rhabdomyolysis to prevent pigment-induced nephropathy. A bladder catheter and urometer should be used to monitor urine output in all critically ill patients.

Escharotomy

An escharotomy may be indicated to relieve restricted ventilation (from circumferential thorax burns) or impaired extremity circulation (from eschar formation in circumferential deep partial-thickness or full-thickness extremity burns). In either of these cases, the eschar should be incised through the dermis down to the level of the subcutaneous fat. If a chest wall escharotomy is required, a vertical incision should be made from the clavicles to the costal margin along the anterior axillary line. This incision may be joined by a transverse incision along the superior, anterior abdominal wall. If a neck escharotomy is required, incisions should be made posterolaterally to avoid vascular structures. On the extremities, incisions are made on the medial and lateral surfaces, with special attention when crossing joints to avoid injuring neurovascular structures.

Table 16.4 American Burn Association grading system for burn severity and disposition

	Minor burn	Moderate burn	Major burn
Criteria	<10% TBSA in adult	10–20% TBSA in adult	>20% TBSA in adult
	<5% TBSA in young (<10 years) or old (>50 years)	5–10% TBSA in young or old	>10% TBSA in young or old
	<2% full-thickness burn	2–5% full-thickness burn	>5% full-thickness burn
		High-voltage injury	High-voltage burn
		Suspected inhalation injury	Known inhalation injury
		Circumferential burn	Any significant burn to face, eyes, ears, genitalia, hands, feet, or major joints
		Concomitant medical problem predisposing to infection (e.g., diabetes, sickle cell disease)	Significant associated injuries (e.g., major trauma)
Disposition	Outpatient management	Hospital admission	Referral to burn center

From American Burn Association. Hospital and Prehospital Resources for Optimal Care of Patients with Burn Injury: Guidelines for Development and Operation of Burn Centers. *J Burn Care Rehabil* 1990;11:98–104.

Outpatient care of burns

Proper patient selection is necessary to ensure optimal burn care in an outpatient setting. The American Burn Association (ABA) has proposed a grading system for estimating burn severity and disposition (Table 16.4). Under ideal conditions, adults with superficial partial-thickness burns <10% TBSA and children with TSBA burns <5% may be considered candidates for outpatient management, as these burns fall into the "minor category" according to the ABA grading system.

The wound care principles for minor burns are the same as for other minor wounds. Minor burns should be cleansed with gentle soap and water, and hair around the burn should not be shaved. Devitalized skin or ruptured blisters should be debrided using aseptic technique. In general, blisters should be left intact. Large or tense blisters can be decompressed by needle aspiration. Tetanus status should be updated according to current Centers for Disease Control and Prevention (CDC) guidelines.

Most burns managed in the outpatient setting are covered with closed dressings. The first layer should be non-adherent, porous, dry sterile gauze. This is covered with a layer of bulky gauze to absorb wound exudate, and subsequently covered with a semi-elastic wrap. Silver sulfadiazine cream (Silvadene) is commonly used for superficial partial-thickness burns. A thin layer of Silvadene can be applied to non-adherent, porous sterile gauze using a tongue blade. The gauze is then applied directly to the burn (Figure 16.8). In general, these dressings are changed once or twice daily, and can be removed in the shower or under running water. The burn is gently washed with mild soap and water and the old cream removed. The wound is then patted dry and re-dressed as above.

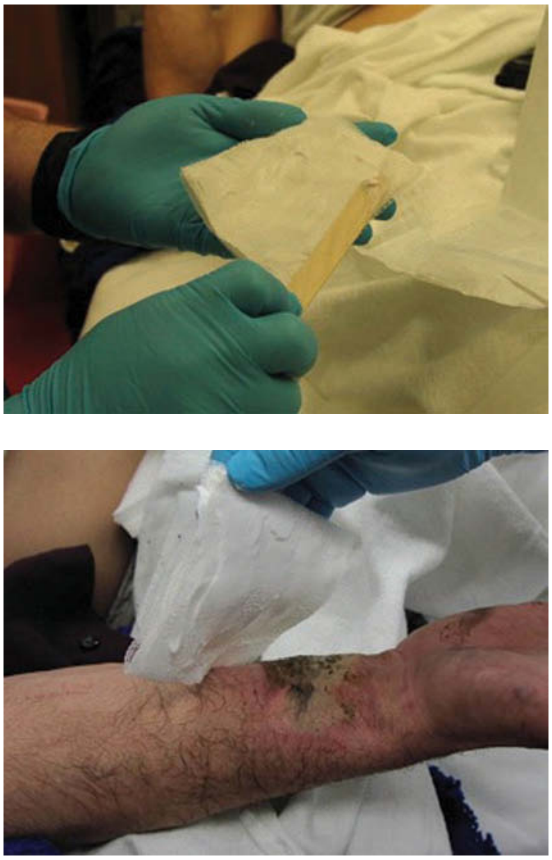


Figure 16.8
A. Application of a thin layer of Silvadene to sterile gauze.
B. Silvadene-covered sterile gauze is applied to a burn wound.

Topical antibiotic ointments (Bacitracin, Polymyxin B sulfate, Neomycin, Polysporin, Neosporin) may be applied to partial-thickness burns when Silvadene is contraindicated, such as in patients allergic to sulfonamides, pregnant women approaching or at term, newborn infants during the first 2 months of life, or patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency. In the outpatient setting, topical antimicrobial agents are most effective for small- to moderate-sized superficial partial-thickness burns that are expected to heal within 2–3 weeks. When applied to deeper burns, topical antimicrobials can prevent wound infections in anticipation of a skin graft. Unfortunately, no consensus opinion or high-quality research presently exists to support one antimicrobial agent over another. Topical neomycin or neomycin-containing agents (e.g., Neosporin) may cause an allergic dermatitis and should be avoided whenever possible. In recent years, newer long-acting silver impregnated and other synthetic dressings have become available.

Select outpatient burns can be managed in an "open" fashion without occlusive dressings. Wounds most commonly treated in this fashion are superficial partial-thickness burns on the neck and face. These burns should be gently washed twice daily with soap and water, followed by application of a topical antibiotic ointment, and then left open to air. Silvadene can cause permanent silver staining of the skin and should not be used on the face.

Patients should be instructed to avoid sun exposure during wound maturation, as this may lead to permanent hyperpigmentation of newly re-epithelialized skin. After re-epithelialization occurs, an unscented moisturizing cream (such as Vaseline Intensive Care, Eucerin, or cocoa butter) should be applied to the wound until the natural lubricating mechanisms of the skin return.

Patients managed in the outpatient setting should first be reevaluated in 24–48 hours, and then every few days until the burn wounds have healed. Minor burns initially managed as an outpatient should be promptly referred to a burn specialist if re-epithelialization has not occurred within 2–3 weeks or if any complications occur.

Analgesia

The majority of burns are painful. Most outpatients can be managed with nonsteroidal antiinflammatory medication with or without the addition of oral narcotic agents. Patients admitted to the hospital often require intravenous narcotic agents. Intramuscular (IM) analgesics should be administered with caution (if at all) in burn victims, as absorption is less reliable and titration is more difficult.

Antibiotics

Routine use of systemic antibiotics is not recommended for patients with acute burn injuries.

Nasogastric tube

Patients with burns ≥20% TBSA may develop a paralytic ileus; a nasogastric tube may prevent gastric distention and emesis.

Bladder catheterization

In critically ill patients, bladder catheterization and urometer placement is recommended to monitor urine output.

Special burns

Tar burns

Hot tar burns, while uncommon, remain a distinct and problematic injury. Tar burns typically affect a small TBSA (mean 4%); however, they can be associated with significant morbidity, as they often occur in critical areas such as the face, hands and feet. Occupational injuries that occur during roofing and road paving account for the majority of tar burns.

Used commonly as a protective coating, roofing tar is generally heated to higher temperatures (232°C) than road paving tar (140°C) to achieve the desired viscosity. As a result, roofing tar burns tend to injure deeper anatomic structures. When tar splatters, it cools to a temperature of 93°–104°C, forming a hard, water-resistant residue. Removing adherent tar without causing further damage to the underlying skin is challenging. A number of non-toxic preparations are efficacious for removing adherent tar, including Neosporin, Tween 80, moist exposed burn ointment (MEBO), De-Solv-it, NISA baby oil, butter, mayonnaise and sunflower oil.

Scald burns

Scald burns from hot liquid or steam account for more than half of all burn-related hospitalizations in the United States. Scald burns are the most common type of injury in children less than 3 years of age, accounting for approximately 70% of all pediatric burns requiring hospitalization. In the elderly (age >65 years), non-fatal scald burns from hot food most commonly affect the arm and hand. Scald burns typically occur in the home, specifically the kitchen. In addition, scald burns are a common occupational injury in restaurant workers. Preventive measures include avoiding mixing water with hot oil when frying, turning pot handles away from the stove's edge, and using dry oven mitts or pot holders.

Despite being somewhat under-recognized, hot tap water is a preventable cause of scald burns (Figure 16.9). Patients at greatest risk for tap water scald burns include the elderly, children <5 years of age, and those with disabilities. Scald burns from hot tap water are directly related to the duration of exposure. Many home water heaters are set at between 60°C and 70°C (140°F and 158°F). Exposure at these temperatures can cause full-thickness burns in less than 5 seconds. Lowering the temperature of a home water heater to 49°C (120°F) would drastically reduce the number and severity of burns from hot tap water. At this temperature, it would take between 5 and 9 minutes to cause a full-thickness injury.

Chemical burns

Chemical burns can be especially challenging to treat. Initial treatment at the scene consists of copious irrigation with water and removal of any particles. Burns due to elemental chemicals (e.g., lithium, sodium, magnesium and potassium) are important exceptions to irrigation with water because the resulting exothermic reaction increases the amount of burn.

Burns caused by hydrofluoric acid (a strong inorganic acid commonly found in rust removers, etching solutions, metal cleaners and electronics manufacturing) are extremely painful due to the corrosive effect of the hydrogen

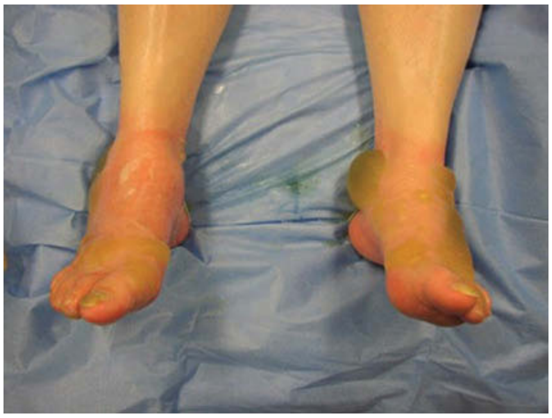


Figure 16.9
Bilateral lower-extremity scald burns from bath water.

ions and penetrating effect of the fluoride ions. Skin injury may appear mild yet cause severe pain. Copious irrigation followed by topical application of calcium gluconate gel may control pain. The gel can be applied using a latex glove if the fingers or hands are burned. If this measure plus narcotics does not control the pain, regional or intra-arterial infiltration of calcium gluconate may be necessary. Life-threatening hypocalcemia and its associated complications have been reported from hydrofluoric acid burns.

Electrical burns

Electrical injuries include those due to high voltage (>1,000 volts), low voltage (<1,000 volts), lightning strikes and arc flash burns. Although these injuries represent a small percentage of burn unit admissions, they are associated with significant morbidity and mortality. High-voltage injuries (typically males, mean age 35 years) most commonly result from contact with power lines. Electrical arc injuries have the lowest morbidity (approximately 1%) but are associated with the largest TBSA. Low-voltage injuries often occur in younger patients (mean age 23 years), and are most likely to involve burns of the upper extremity. Lightning injuries represent the smallest subgroup (approximately 2% of burn unit admissions) but have the highest mortality (17%). In general, survivors of high-voltage electrical injury have the greatest potential for debilitating complications, including traumatic orthopedic injury, fasciotomy and extremity amputation.

Hospital admission with cardiac monitoring is recommended for any electrical injury patient with loss of consciousness, an abnormal initial ECG, or an associated condition necessitating admission. The majority of adult patients sustaining low-voltage electrical injury who have a normal initial ECG can be discharged from the ED.

Special patients

Pediatrics

Children are a challenging subgroup of burn victims and comprise one-third of all burn unit admissions and deaths. Children 4 years of age or younger are at the greatest risk of burn-related injury, accounting for nearly twice the number of burn-related victims as all other pediatric age groups combined. Despite various prevention strategies initiated over the past two decades, burns remain the fourth leading cause of unintentional injury-related death in children between 1 and 14 years of age.

Young children have an increased body surface area-to-weight ratio compared with adults, and therefore have greater fluid requirements than estimated by standard weight-based resuscitation formulas. Failure to include maintenance fluids when resuscitating a young child with a moderate or major burn can result in significant under-resuscitation and vital organ hypoperfusion. Maintenance fluids may be administered as 5% dextrose in ½ normal saline. Daily maintenance fluids are based on the child's weight: 100 mL/kg for the first 10 kg, then 50 mL/kg up to 20 kg, then 20 mL/kg >20 kg.

It is also important to closely monitor blood glucose levels and temperatures in young children, particularly those who weigh <20 kg. Because of limited hepatic glycogen stores, small children have increased susceptibility to hypoglycemia; adding 5% dextrose to Lactated Ringer's solution can prevent hypoglycemia in young children. Small children are also at risk for hypothermia from large burns, as they have smaller muscle mass (limiting their heat generation from shivering) and increased insensible fluid losses (reducing their capacity for adequate thermoregulation).

It is estimated that approximately 10% of pediatric burns are non-accidental, the peak incidence occurring between 13 and 24 months of age. Non-accidental burns should be suspected with any of the following injury patterns: inconsistent history, suspicious-appearing injuries, delay in seeking medical care, "stocking" or "glove" burns (suggesting immersion injury), or a doughnut pattern burn (central sparing) on the buttocks.

Elderly

Elderly patients are at increased risk of burn wounds, with greater complications from burns of similar depth and surface area. Elderly patients typically have thinner skin, multiple comorbidities, less ability to avoid burns, and more physical and psychosocial needs. Elderly patients are also more likely to require a tetanus immunization update.

Disposition

The overwhelming majority of patients with burn injuries who seek emergency medical treatment will be amenable to outpatient treatment. However, approximately 5% of patients with burns will require hospitalization, many requiring transfer to a regional burn center. Other factors to consider when determining suitability for outpatient care include the patient's general state of health, need for ongoing parenteral analgesia, social and family support, ability to follow instructions and perform dressing changes, and access to follow-up health care.

Several questions should be asked when considering a patient for outpatient care:

1. Does the patient have an adequate home environment suitable for outpatient care?
2. Is the patient capable of caring for the burn at home?
3. Are friends or family members available to assist with burn care or activities of daily living?
4. Does the patient have the financial means to be cared for as an outpatient?
5. Is the patient able to follow up as instructed, including transportation to and from follow-up appointments?
6. Do any psychosocial factors exist that may decrease suitability for outpatient burn care?

Pearls, pitfalls and myths

- Residential fires are the leading cause of fire-related deaths, accounting for approximately 75% of fire-related injuries.
- Patients involved in a closed-space fire or explosion are at risk of inhalation injury.
- Complaints of headache or dizziness in the context of a fire or burn injury suggest concomitant CO toxicity.
- Even experienced clinicians are not always able to accurately determine the depth of the burn, including differentiating between a deep partial-thickness and full-thickness burn at the time of injury.
- Intravenous fluid resuscitation should be initiated in adults with partial- or full-thickness TBSA burns >20%, in older children with burns ≥15% TBSA, and in infants with burns ≥10% TBSA.
- Lactated Ringer's solution is the most commonly used fluid for burn resuscitation.
- The Parkland formula calls for the administration of 4 mL/kg of body weight per %TBSA burn (partial- or full-thickness burns) of intravenous fluid over the first 24 hours (half over the initial 8 hours and the remainder over the next 16 hours).
- Direct thermal injury predominantly affects upper airway structures. Injury to the lower airway typically occurs due to inhalation of toxic gases and particulate matter.
- Wound care principles for minor wounds can be applied to patients with minor burns.
- Silver sulfadiazine cream (Silvadene) is most commonly used with closed burn dressings for the management of superficial partial-thickness burns. It should be avoided on the face, in infants, in late-term pregnancy, or in those with sulfonamide allergy or G6PD deficiency.
- Young children have an increased body surface area to weight ratio compared with adults, therefore requiring greater fluid than determined with the use of standard weight-based resuscitation formulas.

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17 Chest pain

Jeffrey A. Tabas, MD and Susan B. Promes, MD

Scope of the problem

Acute chest pain is the presenting complaint in roughly 3% of emergency department (ED) patients. The diagnostic possibilities range from the immediately life-threatening (acute coronary syndrome [ACS], comprised of acute myocardial infarction [AMI] and unstable angina [USA], aortic dissection, pulmonary embolism [PE] and ruptured esophagus) to the self-limiting (chest wall strain), and the common (gastroesophageal reflux disease) to the unusual (herpes zoster). Although the etiology of the chest pain may remain unidentified in a significant proportion of patients, which can be frustrating to both the patient and provider, it is imperative that the clinician recognizes and treats life-threatening causes.

Anatomic essentials

When considering the differential diagnosis of the patient with chest pain, it is helpful to consider the five organ systems in the thorax: cardiac (heart and pericardium), pulmonary (lungs and pleura), gastrointestinal (esophagus and upper abdominal contents), vascular (aorta and great vessels) and musculoskeletal (chest wall). *Visceral pain* from internal structures such as the heart, lungs, esophagus and aorta may be difficult for the patient to define. Pain may be described as a discomfort or strange sensation, and it is often challenging for the patient to discern an exact location. *Somatic pain* from chest wall structures is often more localizable and easier for the patient to characterize. Pain may be sharp or stabbing, brought on by movement or position, and can often be pinpointed. *Referred pain* from irritation or inflammation of the upper abdominal contents may be perceived as pain in the chest or upper back. A differential diagnosis based on these structures is given in Table 17.1.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 17.2).

History

A careful and focused history may uncover important clues to the etiology of chest pain. To avoid a common pitfall, ask open-ended questions when possible. "Why did you come to the emergency department today?" may yield more initial information than "How often do you get this chest pain?" Additionally, ask about chest discomfort rather than pain, since a patient may deny pain and admit only to chest pressure. The mnemonic *LMNOPQRST* (location, medical history, new, other symptoms, provoking/palliative, quality, radiation, severity, timing) may be helpful in obtaining a complete history. Whether or not you use this mnemonic, it is important to obtain a picture of the patient's symptoms that includes the following aspects.

Location

The location of pain may increase or decrease the likelihood of different diagnoses. A specific spot that is extremely tender to palpation and worsens with position change may be consistent with a musculoskeletal etiology. As mentioned previously, visceral pain may be difficult to localize.

Table 17.1 Differential diagnosis of chest pain

Heart
Myocardial infarction
Cardiac angina
Pericarditis
Myocarditis
Valvular diseases (especially aortic stenosis)
Lungs
Pneumonia/other infections
Pneumothorax
Pulmonary embolism
Chronic obstructive pulmonary disease exacerbation
Esophagus
Esophagitis (e.g., candidal)
Gastroesophageal reflux disease
Spasm (Nutcracker esophagus)
Foreign body
Rupture (Boerhaave's)
Aorta
Dissection
Aneurysm
Aortitis
Upper abdomen
Gallbladder disease (cholecystitis or cholelithiasis)
Pancreatitis
Duodenal or peptic ulcer
Hepatic disease
Chest wall
Costochondritis (Tietze's disease)
Contusion
Rib fracture
Muscle strain or tear
Herpes zoster

Table 17.2 Chest pain red flags

History	Concerning diagnosis
Elderly (older)	Serious disease
Diaphoresis	Serious disease
Exertional, relieved by rest	Unstable angina
Radiation to arm, shoulder, neck, jaw	Acute coronary syndrome, aortic dissection
Following vomiting	Esophageal rupture
Neurologic deficit	Aortic dissection
Sudden onset, maximal intensity, tearing	Aortic dissection
Sudden onset	PE, AMI, pneumothorax
Radiation to back	Aortic dissection
Pleuritic	PE, pericarditis, pneumothorax
Dyspnea	ACS, PE, COPD exacerbation, PNA, PTX
History of trauma with dyspnea	Pulmonary contusion, flail chest, cardiac contusion, hemothorax/ pneumothorax/hemopneumothorax
Examination finding	**Concerning diagnosis**
Hypotension	Tamponade, CHF, sepsis, aortic dissection, PE, tension PTX
Diaphoresis	Serious disease
Asymmetric pulse or BP	Aortic dissection
Systolic murmur	Aortic stenosis
Diastolic murmur	Aortic dissection causing aortic insufficiency
Subcutaneous air	Pneumothorax, ruptured esophagus
Neurologic deficit	Aortic dissection
Bilateral rales	Congestive heart failure
Focal rales, egophony	Pneumonia

ACS: acute coronary syndrome; AMI: acute myocardial infarction; BP: blood pressure; CHF: congestive heart failure; COPD: chronic obstructive pulmonary disease; PE: pulmonary embolism; PNA: pneumonia; PTX: pneumothorax.

Medical history

Although classic risk factors for coronary artery disease (CAD) have not been shown to be predictive in the acute setting, many clinicians extrapolate their importance from long-term studies. These include hypertension, smoking, diabetes, increased cholesterol, obesity, gout and family history of premature heart disease. Cocaine use is a risk factor for both cardiac ischemia and aortic dissection. A history of rheumatic fever or a murmur can suggest valvular disease. Patients with a history of cerebral or peripheral vascular disease (including erectile dysfunction) or CAD are at risk for ACS. Patients with a history of hypertension or Marfan's disease are at risk for aortic dissection. Patients with vomiting or a recent esophageal procedure may be at risk for esophageal perforation. A heavy smoking history suggests underlying chronic obstructive pulmonary disease (COPD). Risk factors for PE include recent surgery, family history, cancer, estrogen use or pregnancy. Patients with PE have no identifiable risk factor in 20% of cases.

New

It is important to discover whether the patient has had similar episodes of chest pain in the past, if they have been of similar severity, and what medical diagnosis and treatment, if any, they received. A history of multiple similar episodes of pain associated with previous COPD exacerbations is an important clue that this may be a similar exacerbation.

Other (associated) symptoms

Nausea, vomiting, shortness of breath, syncope, near-syncope and palpitations may increase the clinician's suspicion for serious illness, such as cardiac ischemia or dysrhythmia. Shortness of breath, productive cough and fever may suggest a respiratory infection. Any neurologic deficit in the setting of pain should immediately suggest aortic dissection.

Provoking/palliative

Pain that is worse with a deep breath is termed pleuritic. Pleuritic pain is associated with pulmonary etiologies such as pneumonia, PE, or COPD exacerbation. Pain that is worse lying flat and improves sitting up suggests pericarditis. Chest discomfort due to angina may be associated with exertion and often improves with rest or nitroglycerin (NTG). Burning pain that is associated with meals may suggest a gastrointestinal (GI) etiology.

Quality

Chest discomfort may be described in numerous ways, such as sharp, burning, stabbing, pinching, squeezing, heaviness or pressure. It is important to begin with open-ended questions, such as "Tell me about this discomfort in your chest," rather than "How long have you had this discomfort?" If a patient cannot provide a description of the character, you can use prompts such as "Is it sharp, burning, stabbing, heavy, squeezing or pinching?" Pressure, heavy or squeezing pain is often consistent with cardiac ischemia. Sharp or stabbing pain suggests a non-cardiac cause, although cardiac ischemia can present in a multitude of ways. A patient's demonstration of pain by a clenched fist against his or her chest is termed *Levine's sign*. One small study showed a high correlation with this description and acute cardiac ischemia.

Radiation

Radiation of pain to the neck, jaw, shoulder or arm significantly increases the likelihood that the pain is of cardiac origin. Pain that radiates to the back may be associated with aortic dissection.

Severity

"On a scale of 0 to 10, how severe is the pain?" Severe chest pain should always raise concern for a life-threatening emergency. A rating of 1 is almost undetectable, whereas a rating of 10 is the worst pain ever experienced. Severity of pain is not predictive of disease, but should be followed over time to document the effect of interventions. The goal of the practitioner is to relieve pain as rapidly as possible, especially in a patient with a high suspicion of acute cardiac ischemia.

Timing (duration and onset)

"How long have you had this discomfort? Is it constant, waxing/waning, or intermittent?" Pain lasting seconds or more than 24 hours is less likely to be cardiac in origin. However, use caution when questioning about symptom duration. Discomfort that has been intermittent for several days and recently became severe differs from pain that has remained constant and unchanged for the last 72 hours. Be certain to ask questions about today's episode of pain, distinguishing it from previous episodes. "Do you get this pain with exertion?" would receive a different response depending on whether you are referring to prior anginal-type pain or today's non-exertional chest pain due to AMI. Sudden or abrupt onset of chest pain may indicate a serious underlying disorder, such as MI, PE, or aortic dissection.

Physical examination

The physical examination in patients with chest pain may be unrevealing. However, a thorough examination is essential to identify important diagnostic clues.

General appearance

The general appearance of a patient is an important clinical observation. Patients who appear markedly uncomfortable or present with pallor, diaphoresis, or respiratory distress should be considered acutely ill. Evaluation and treatment should proceed in parallel rather than sequentially.

Vital signs

Vital signs are vital, and should be verified. Hypotension is a sign of shock and may be due to decreased cardiac output, intravascular volume depletion, or sepsis. Note any difference in blood pressure between arms, or between arms and legs. This may suggest aortic dissection. Tachycardia may suggest systemic illness, dysrhythmia, or pain. In addition to reviewing the recorded vital signs, observe the patient's respiratory pattern and rate. Abnormal respirations may be a clue to a pneumothorax, congestive heart failure (CHF), PE, COPD exacerbation, or other pulmonary abnormalities. Fever or hypothermia may suggest an infectious process. Think of pulse oximetry as the "fifth vital sign," and measure it in all patients with chest pain. A low measurement may suggest a pulmonary disorder or poor perfusion from a cardiac or vascular event, especially if it is decreased from the patient's baseline.

Pulsus paradoxus is a loss of the pulse during inspiration. It represents the fall in systolic pressure from expiration to inspiration that can be felt or auscultated. As the cuff is slowly deflated, note the pressure at which any pulse is first detected, and then the pressure at which every beat is detected. Normally, the fall in systolic arterial pressure is less than 10 mmHg during inspiration. Presence of a "pulsus" classically suggests cardiac tamponade, although it may also be present in pulmonary conditions such as emphysema or asthma.

Skin

Note the degree of perfusion, pallor, or diaphoresis. Visual examination of the chest may identify the cause for the pain, such as contusion or ecchymosis suggesting traumatic injury, or vesicular rash with a dermatomal distribution suggesting herpes zoster. It is important to note that pain associated with herpes zoster may occur prior to the development of the rash.

Pulmonary

Inspection of the chest and surrounding structures may reveal increased respiratory effort or accessory muscle use. Auscultation may reveal normal, abnormal, or diminished breath sounds. Bilaterally decreased breath sounds with poor air movement suggest severe reactive airway disease or emphysema. Unilaterally decreased breath sounds suggest consolidation, pneumothorax, or pleural effusion. If there are decreased breath sounds unilaterally, the position of the trachea should be examined for signs of tracheal deviation suggestive of tension pneumothorax. Check for "E to A" changes (egophony) throughout the lungs; their presence indicates consolidation. Increased inspiratory to expiratory (I:E) ratio should be noted (normal is 2:1). Prolongation of the expiratory phase suggests significant obstructive airway disease. Adventitial sounds such as wheezes suggest reactive airway disease. Rales or crackles may be consistent with atelectasis, infiltrate, or edema. The location and extent of these should be documented (e.g., one-third of the way up bilaterally, or right lower lung field). "Velcro-like" rales are consistent with chronic interstitial fibrosis. Percussion may be useful for localizing dullness (suggesting infiltrate, mass, or fluid) or hyperresonance (suggesting pneumothorax). Palpation of the chest wall may be helpful to identify crepitus, or to localize tenderness when a musculoskeletal source is suspected.

Cardiac

Inspection of the chest may reveal previous surgical scars, implanted devices such as pacemakers or cardioverter defibrillators (ICDs), and hyperdynamic states. Palpation should assess for location and quality of the left ventricular systolic impulse. Normal location of this impulse is in the fifth intercostal space at the mid-clavicular line. Placing the fingers of the right hand at the left sternal border in each rib space allows appreciation of a right-sided heave. Auscultation of heart sounds should proceed over all four cardiac listening areas, first with the diaphragm and then with the bell. The regularity of the heart sounds and any murmurs, rubs, or gallops should be noted. If a murmur is noted, determination of any radiation will assist in diagnosis. The most commonly heard murmurs and methods to distinguish them are listed in Table 17.3.

Carotid arteries

Auscultation of the carotid arteries should be performed using the bell of the stethoscope to assess for bruits (often unilateral) or transmitted murmurs (bilateral). Pressing too firmly on the carotids may create a false bruit. If there is confusion as to the source of the bruit (carotid vs. cardiac), auscultation in the region of the sternal notch will either confirm the presence or absence of a transmitted murmur. Palpation of the carotid pulses should also be performed to confirm normal strength and upstroke.

Jugular venous pressure (JVP)

Findings of right heart failure include jugular venous distension, hepatic congestion and peripheral edema. Patients may have right-sided heart failure from left-sided failure (most common etiology), pulmonary hypertension (COPD, PE), or impaired right-sided filling (pericardial tamponade, tension pneumothorax). The jugular venous pressure (JVP) is noted in the anterior triangle of the neck (Figure 17.1). The patient should rest with the head of the bed at 30° and the chin rotated left of midline by 30°. The pulsation is most often visible just above the clavicle. The jugular pulse is distinguished from the carotid pulse by its double wave and lack of palpability. It can further be confirmed by noting a rise in the height of the JVP by lowering the bed or by compressing the liver (hepatojugular reflux).

Table 17.3 Most common cardiac murmurs and methods to distinguish them

Three common systolic murmurs

1. Systolic ejection (flow)
    - (a) Heard across the precordium with the diaphragm of the stethoscope
2. Aortic stenosis
    - (a) Harsh, crescendo/decrescendo, heard with diaphragm
    - (b) Radiates to carotids (heard with bell of the stethoscope)
3. Mitral regurgitation
    - (a) Heard at apex with the bell, radiating to the axilla
    - (b) Blowing, holosystolic
    - (c) Heard best with patient turned slightly into left lateral decubitus position
    - (d) Increased with Valsalva maneuver

Two common diastolic murmurs

1. Mitral stenosis
    - (a) Low, rumbling
2. Aortic regurgitation
    - (a) Blowing, decrescendo or holodiastolic

Other cardiac sounds

1. Idiopathic hypertrophic subaortic stenosis (IHSS)
    - (a) Late systolic murmur without radiation
2. Pericardial rub
    - (a) Triple phase (mid-systole, mid-diastole, pre-systole)
    - (b) Scratchy

3. S3
    - (a) Heard best with bell at apex in left lateral decubitus position
    - (b) Sounds like *Kentucky* (Ken = S1, tu = S2, cky = S3)
    - (c) Represents heart failure in an adult
    - (d) Normal finding in small children

4. S4
    - (a) Heard best with bell at apex
    - (b) Sounds like *Tennessee* (Te = S4, nne = S1, ssee = S2)
    - (c) Represents atrial filling of stiff ventricle in an adult
    - (d) Always pathologic in a child

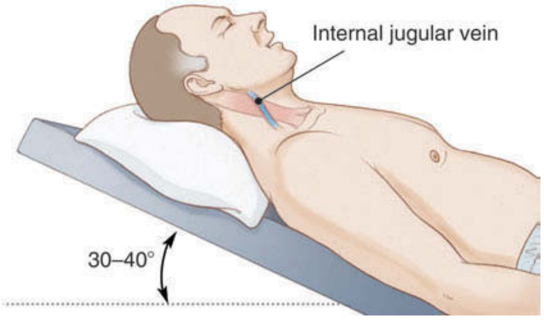


Figure 17.1
Jugular venous pressure assessment. © Chris Gralapp.

Extremities

Pulses should be assessed including symmetry between sides and between upper and lower extremities. Changes of peripheral vascular disease, such as decreased pulses, hair loss, or shiny reddened skin, may provide evidence of underlying atherosclerotic disease. Bilateral lower extremity edema may represent right heart failure (usually secondary to left heart failure or pulmonary disease), especially in the presence of elevated JVP. Another common cause of bilateral lower extremity edema is venous insufficiency. Liver failure, hypoalbuminemia and nephrotic syndrome should also be considered as causes of edema. Asymmetric edema should raise concern for deep venous thrombosis (DVT), especially in the presence of cords or venous distension. When asymmetry is present, the size of each leg should be measured and recorded.

Abdomen

Always perform the abdominal exam with the head of the bed flat (so the patient is completely supine), both knees bent (to relax the abdominal musculature), and both arms down by the sides. This allows the most accurate and reproducible exam. The examination of the abdomen should progress sequentially with observation, auscultation, percussion and palpation. It is particularly important to evaluate for non-thoracic causes of chest pain, such as diseases of the gallbladder (cholecystitis or cholelithiasis). Note the presence of bruits or pulsatile masses suggesting abdominal aortic aneurysm, a potential life-threatening emergency.

Rectal

Rectal examination should be performed to assess for gross blood, melena, or occult blood. The presence of GI bleeding may impact imminent therapy (anticoagulation or fibrinolysis), or be the source of significant blood loss resulting in cardiac ischemia.

Neurologic

A complaint-directed neurologic examination should be performed. Any new neurologic deficits in the setting of chest pain should be presumed due to aortic dissection and considered an emergency unless proven otherwise.

Differential diagnosis

Table 17.4 provides an extensive list of possible chest pain causes.

Table 17.4 Differential diagnosis of chest pain

Diagnosis	Classic symptoms	Signs	Work-up
Acute myocardial infarction	*High-risk features include:* 1. Advanced age 2. Known CAD 3. Diabetes 4. Pain like prior AMI or worse than usual angina 5. Pain that is pressure-like or squeezing 6. Radiation to neck, left shoulder, or left arm *Low-risk features include*: 1. Pleuritic, sharp, or stabbing pain 2. Pain reproducible with palpation or movement 3. Younger age 4. Pain lasting for seconds or constant for >24 hours However, 22% of patients with AMI have pain that is sharp or stabbing, 13% have partially pleuritic pain, and 7% have pain completely reproduced by palpation.	Physical examination is most helpful when there are findings of decreased cardiac output: rales, hypotension, an S3, new or worsening mitral regurgitation murmur. Otherwise, it is often unremarkable.	Diagnosed by an elevation of serum cardiac markers and one of the following: 1. Clinical history of ischemic-type chest discomfort *or* 2. Serial changes on ECG *or* 3. Results from urgent angiography or provocative testing
Aortic dissection	Presentation: pain (95%), abrupt onset (85%), severe or worst ever (90%), tearing or ripping (50%), chest (75%) and/or back location (50%), syncope (10%), hypertension history (70%).	Hypertension (50%), hypotension (5%), aortic insufficiency murmur (30%), pulse deficit (15%), neurologic deficit.	CXR may reveal abnormalities (Table 17.6). Helical CT and echocardiogram are sensitive and specific.
Aortic stenosis	Classic progression of symptoms over time from chest pain to syncope to CHF.	Harsh, systolic, crescendo–decrescendo murmur radiating to carotids. Weak, delayed pulses, narrow pulse pressure.	ECG may show left ventricular hypertrophy. Diagnosis by echocardiography or cardiac catheterization.
COPD exacerbation	Patients may complain of dyspnea and pleuritic chest pain. Symptoms of respiratory infection may be present.	Vital signs may show tachypnea, tachycardia, and hypoxia. Breath sounds are typically decreased. Wheezing is variable depending on the amount of air movement.	Obtain CXR to exclude pneumonia or pneumothorax as exacerbating factor. Diagnosed when symptoms respond to appropriate therapy (such as beta-agonists).

(*continued*)

Table 17.4 Differential diagnosis of chest pain (*cont.*)

Diagnosis	Classic symptoms	Signs	Work-up
Esophageal rupture	Chest pain in the setting of vomiting or recent esophageal procedure. Progressively increasing symptoms with diagnostic delays.	Early physical examination can be remarkably benign. As disease progresses, infectious mediastinitis develops.	Laboratory analysis may be unremarkable. CXR may reveal abnormal air in the mediastinum (pneumomediastinum) or may be normal. Definitive diagnosis by CT scan, Gastrografin esophagram (avoid barium given risk of extravasation), or endoscopy.
Pericardial tamponade	Often presents with shortness of breath or weakness rather than chest pain.	Tachycardia is an early presentation. Pulsus paradoxus is present. With progression, distended jugular veins and hypotension develop. The classic presentation of Beck's triad (muffled heart tones, distended neck veins and hypotension) is actually uncommon.	ECG usually reveals low voltage. Electrical alternans (alternating size of the QRS complex) is highly suggestive. Definitive diagnosis by ultrasound demonstrates impaired relaxation of the right atrium and ventricle during diastole.
Pericarditis	Sharp or burning pain, often of several days duration, pleuritic component, worse lying down, better sitting forward, may have prodrome of fever and malaise. Uremia from renal failure is a common predisposing factor.	Scratchy or squeaky pericardial friction rub heard best in left lower sternal border using the diaphragm – usually triphasic, but may have just two components. Varying degree of fever. An increased pulsus paradoxus is concerning for tamponade.	Four stages on ECG: 1. Diffuse ST-segment elevation, PR depression, and peaked T waves most common 2. Normalization 3. Deep, symmetric, diffuse T-wave inversion 4. Normalization Diagnosis is suggested by pericardial effusion on echocardiography, although an effusion may be absent in the presence of pericarditis.
Pneumonia	Productive cough, fever, shortness of breath. Symptoms may be less impressive in immunocompromised states (diabetes, HIV, chronic alcoholism).	Fever, tachypnea, hypoxia, and/or findings of consolidation, such as rales or E to A changes (egophony).	Leukocytosis on CBC. CXR demonstrates an infiltrate. PA and lateral films are more sensitive and specific than a portable AP film. Consider tuberculosis, pneumocystis in the HIV patient.
Pneumothorax	Often associated with history of trauma. Spontaneous pneumothorax typically occurs in tall, thin individuals, 20–40 years old, male >female. Secondary pneumothorax may occur in smokers, patients with emphysema or asthma, or patients with pneumocystis. Symptoms include pleuritic chest pain and shortness of breath.	Decreased breath sounds, tachypnea, hypoxia may or may not be present. Tracheal deviation may be noted with tension pneumothorax.	CXR, CT, or ultrasound can identify pneumothorax. A diagnosis of tension pneumothorax should never be made radiographically, since it should be diagnosed clinically and treated immediately.
Pulmonary embolism	Risk factors include recent pelvic or low abdominal surgery, family or patient history of thromboembolism, cancer, paralysis, LE casting or immobility, CHF, estrogen use or pregnancy, LE extremity or pelvic trauma, age >40 years. Twenty percent of patients with PE have no risk factors.	Respirations >20/min (70%), rales (51%), tachycardia (30%), leg swelling (28%), loud P2 (23%), fever >38.5 °C (13%), wheezing (5%).	V/Q scan, helical CT, or pulmonary angiography are diagnostic tests of choice. A negative result of a high sensitivity D-dimer test in a low-risk patient may be adequate to exclude disease.
Unstable angina	Angina is discomfort, induced by exercise, relieved by rest or NTG. USA is either: 1. Angina at rest (usually >20 min) *or* 2. New-onset exertional angina (<2 months) with walking 1–2 blocks or 1 flight of stairs *or* 3. Increased severity within 2 months at above exertion level.	Often absent	Exclusion of this condition requires noninvasive stress testing or cardiac catheterization. Dynamic ECG abnormalities or elevated cardiac markers define a high-risk group.

AMI: acute myocardial infarction; AP: anteroposterior; CAD: coronary artery disease; CBC: complete blood count; CHF: congestive heart failure; COPD: chronic obstructive pulmonary disease; CT: computed tomography; CXR: chest X-ray; ECG: electrocardiogram; HIV: human immunodeficiency virus; LE: lower extremity; NTG: nitroglycerin; PA: posteroanterior; PE: pulmonary embolism; USA: unstable angina; V/Q: ventilation–perfusion.

Diagnostic testing

Laboratory studies

Complete blood count

A complete blood count (CBC) is frequently ordered in patients with chest pain. A low hematocrit may indicate a reason for symptoms of cardiac ischemia, or may be due to bleeding associated with the source of pain (gastric ulcer). Most authorities recommend maintenance of the hematocrit in a patient with cardiac ischemia above 30 mg/dL to maximize O_2 delivery. A high white blood cell count may represent demargination due to stress, pain, or a catastrophic event (e.g., sepsis from delayed diagnosis of esophageal rupture). However, this test is rarely illustrative.

Chemistry panel

Chemistry panels generally provide little help in the evaluation of the patient with chest pain. They may suggest acidosis (low bicarbonate), especially in the presence of an anion gap. If intravenous (IV) contrast imaging is to be performed to evaluate the aorta, it is important to obtain a creatinine level prior to contrast administration. Renal insufficiency, suggested by an elevated serum creatinine, is a relative contraindication to contrast injection, as in computed tomography (CT) or cardiac catheterization. Elevated glucose may reveal previously unsuspected diabetes, a risk factor for CAD.

Cardiac markers

Serial serum cardiac marker measurements can be used to rule out AMI at an appropriate interval from symptom onset (Table 17.5). However, serial cardiac markers are only elevated in 10–30% of cases of USA, and therefore cannot be used to exclude this condition.

Liver function tests

Liver function tests may be elevated in patients with biliary or hepatic disease, or due to passive congestion of the liver in heart failure.

Amylase/lipase

When an abdominal source of pain is suspected, or tenderness is elicited in the mid-epigastrium, pancreatitis should be considered. This is especially true in the presence of risk factors (e.g., alcohol use, biliary disease and diabetes).

Urinalysis

Evaluation of the urine is rarely helpful in the chest pain patient, unless glucosuria (possible screen for diabetes) or bilirubinuria (possible screen for biliary duct obstruction or hepatic disease) is present.

Table 17.5 Measurements of serial cardiac markers to rule out AMI

Cardiac marker characteristics	**Rise (hours)**	**Peak (hours)**	**Return to baseline**
Myoglobin	<3	4–9	<24 hrs
CK-MB mass	3–8	9–30	1–3 days
CK-MB subforms	1–3	4–6	18–24 hrs
cTnT	2–6	10–24	10–15 days
cTnI	2–6	10–24	7–10 days
Timing of testing to exclude myocardial infarction – hours post pain *onset*			
Myoglobin	Does not exclude		
CK-MB mass	6–10		
CK-MB subforms	6–10		
cTnT	8–12		
cTnI	8–12		

Serial marker testing for rapid exclusion of AMI in low-risk patients – ACEP policy.

Obtain an initial marker on arrival. Obtain a second marker at least 6 hours from chest pain onset for CK and 8 hours from onset for Troponin. Note that serial marker testing excludes AMI but does not exclude USA.

ACEP: American College of Emergency Physicians; AMI: acute myocardial infarction; CK-MB: creatine kinase; cTnI: cardiac troponin I; cTnT: cardiac troponin T; USA: unstable angina.

Pregnancy test

Consider a pregnancy test in all female patients of childbearing age, especially if they may undergo radiologic imaging.

Urine toxicology screen

Cocaine has been associated with ACS, AMI and aortic dissection, especially in the first hour after use.

D-dimer

D-dimers are degradation products of circulating cross-linked fibrin. They are elevated when the coagulation pathway is activated, as with thromboembolism. Sensitivity and specificity for thromboembolism vary, depending on the type of test. The characteristics of the test available at your institution should be determined – most are appropriate to exclude DVT or PE in low-suspicion patients. There are some data that the D-dimer has similar utility to exclude aortic dissection in low-suspicion patients.

Arterial blood gas

Arterial blood gas sampling is useful to assess ventilatory status (CO_2 level) and serum pH, and to confirm a low pulse oximetry reading. In the assessment of a patient with suspected PE, it has been shown to lack significant predictive value. In one study, 26% of patients with PE had a partial pressure of O_2 (PO_2) >80 mm. A low oxygen saturation, either from pulse oximetry or ABG, that lacks an adequate explanation (i.e., pneumonia, heart failure or COPD) should raise suspicion for pulmonary embolism.

Electrocardiogram

An attempt should be made to perform an electrocardiogram (ECG) within 10 minutes of arrival for all patients with unexplained chest pain (recommendation of the American College of Emergency Physicians and the American College of Cardiology). In studies of patients with AMI, ECGs are diagnostic in 30–50%, nonspecific in 40–70%, and normal in up to 10%. Findings of acute ischemia include new or presumed new ST-segment elevation, ST-segment depression, or inverted T waves (Figure 17.2). Known findings on ECG that make the assessment of ischemia challenging include bundle branch block (especially left bundle branch block) and left ventricular hypertrophy with repolarization abnormality (strain pattern). American Heart Association guidelines recommend right-sided ECG leads when there are findings of ischemia in the inferior leads (II, III and aVF) (Figure 17.3), and posterior ECG leads when there is ST-segment depression in the septal leads (V1 and V2).

The ECG may also reveal evidence of pericarditis (Figure 17.4) or pericardial effusion.

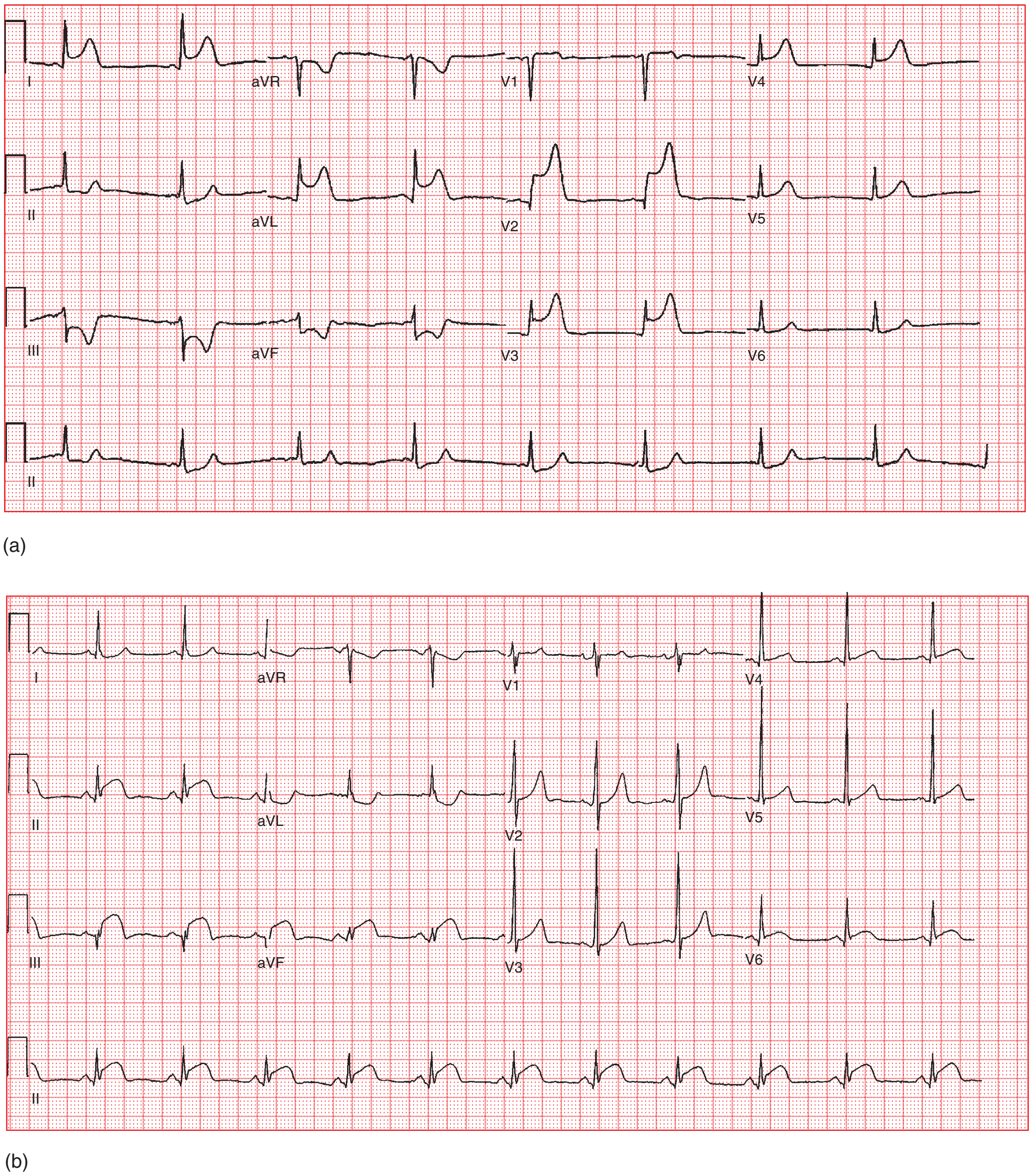
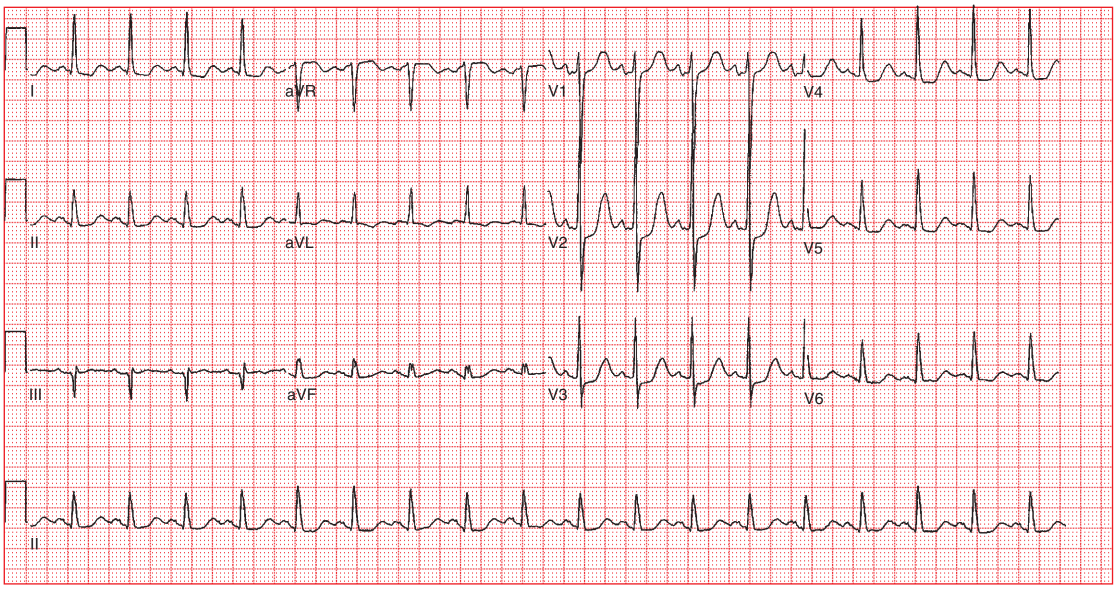


Figure 17.2
(a) ECG in a patient with anterolateral MI demonstrating ST-segment elevations in leads I, aVL and V2–V4. Note the reciprocal ST-segment depressions in leads II, III and aVF. (b) ECG in a patient with inferoposterior MI demonstrating ST-segment elevations in leads II, II and aVF, and prominent R waves in leads V1–V3. Courtesy: Amal Mattu, MD.



(c)

Figure 17.2 (*cont.*)
(c) ECG in a patient with posterior MI demonstrating prominent R waves in leads V1–V3 and ST-segment depression in leads V2–V3. Courtesy: Amal Mattu, MD.

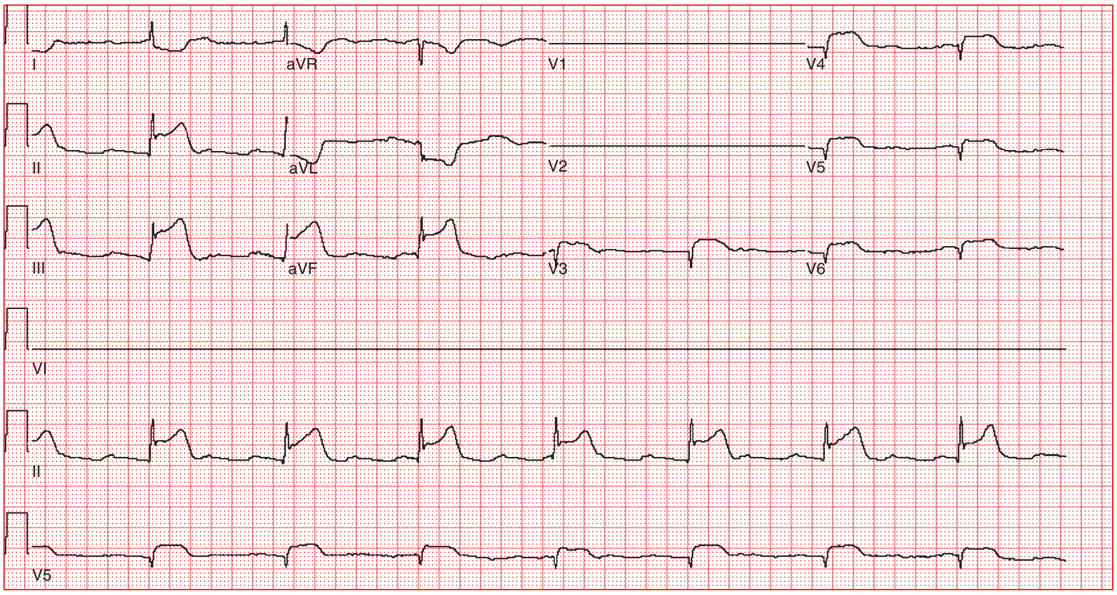


Figure 17.3
Right-sided ECG in a patient with right ventricular infarct demonstrating ST-segment elevation in lead V4R. Courtesy: S.V. Gurudevan, MD.

Risk stratification scores

Using clinical features, ECG findings and troponin results measured over 6–12 hours, the TIMI (Thrombolysis In Myocardial Infarction) risk score was derived in patients with proven ACS to predict risk of subsequent adverse events. Subsequent attempts have been made to apply the score to patients with suspected or possible ACS to guide disposition or treatment. However, no scoring system has been shown to be sensitive enough to guide discharge from the ED, and compelling evidence to guide treatment selection is lacking.

Radiologic studies

Chest radiography

Chest radiography is most helpful when it points to a definitive diagnosis such as pneumothorax or pneumonia. Although chest radiography is often normal

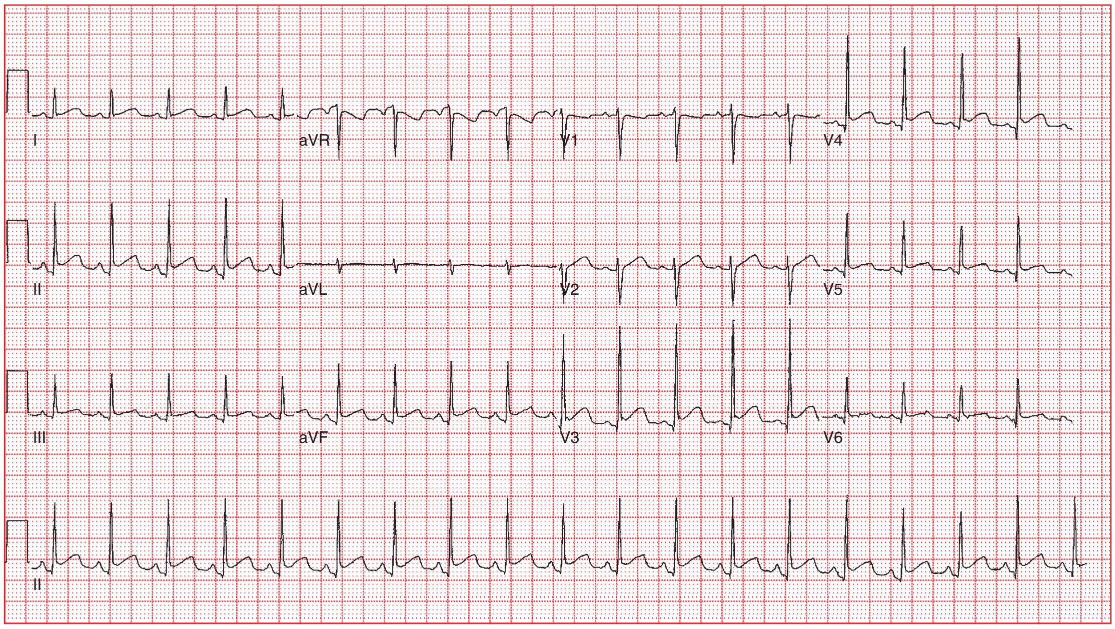


Figure 17.4
ECG in a patient with pericarditis demonstrating PR-segment depression, PR-segment elevation in aVR and diffuse ST-segment elevations. Courtesy: Amal Mattu, MD.

or nonspecific in conditions such as AMI, PE and aortic dissection, it may also suggest the diagnosis (Figures 17.5 and 17.6). Tables 17.6 and 17.7 describe chest X-ray (CXR) findings in aortic dissection and PE, respectively.

Helical computed tomography

Helical CT may be extremely helpful in the evaluation of a stable patient with chest pain. New generation scanners are reasonably sensitive (80% to >90%) and specific (90% to >95%) for PE depending on scanner technology and the expertise of the radiologist. CT often provides additional information either suggestive or supportive of a final diagnosis in patients without PE. It is 95–100% sensitive and specific for aortic dissection (Figure 17.5b). In the rare occasion that helical CT is inconclusive for aortic dissection or PE and the pretest probability for the diagnosis is high, conventional angiography should be performed. Some institutions are using CT angiography and calcium scores of the coronary vessels to aid in the diagnosis of CAD. However, this modality for identifying patients with CAD is not universally available.

Echocardiography

This test can prove helpful in the evaluation of chest pain, especially in the unstable patient. Transthoracic echocardiography can evaluate the cardiac chamber sizes, wall motion, systolic function, valvular function and aortic integrity. Remarkable findings include valvular disease, pericardial effusion with tamponade physiology, regional wall motion abnormalities suggesting ischemic cardiac disease, right heart failure suggesting acute PE, and aortic dissection. Transesophageal echocardiography is more sensitive than transthoracic echocardiography in detecting aortic dissection.

Table 17.6 Chest X-ray findings in aortic dissection

Normal (10–30%)
Wide mediastinum or abnormal aorta (70–80%)
Wide paraspinal shadow
Pleural effusion
Tracheal shift
Aortic calcification displacement
“Lump” distal to vessels

Table 17.7 Chest X-ray findings in pulmonary embolism

Classic presentation is normal X-ray in patient with dyspnea and hypoxia
Atelectasis or parenchymal abnormality (68%)
Elevated hemidiaphragm
Pleural effusion
Hampton’s hump is a wedge-shaped pleural-based density (Figure 17.6)
Westermark sign is distension of pulmonary vasculature proximal to embolism with loss of vascular markings distally (rare)

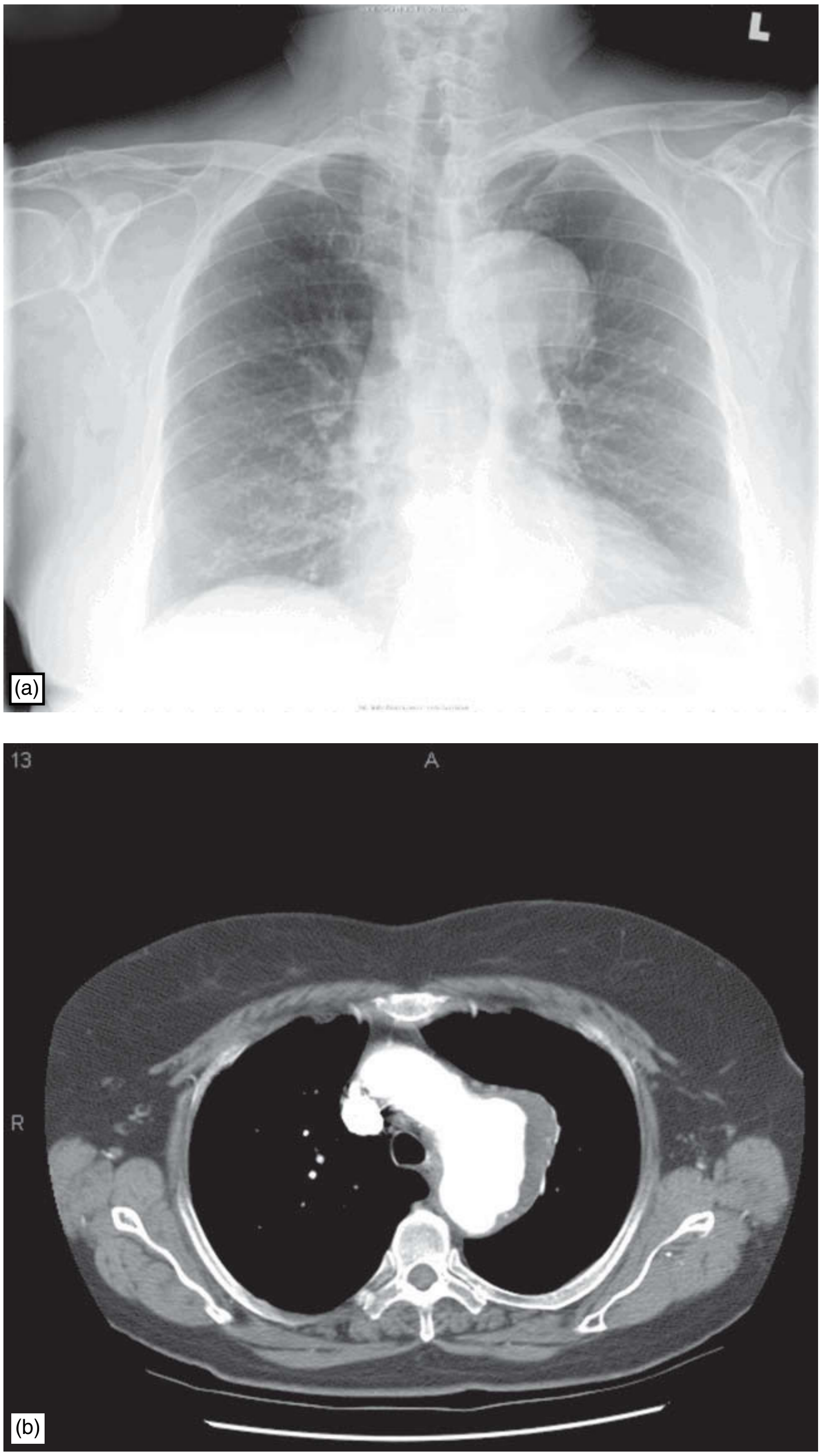


Figure 17.5
(a) Abnormal chest X-ray and (b) chest CT revealing aortic dissection. Courtesy: Gus M. Garmel, MD.

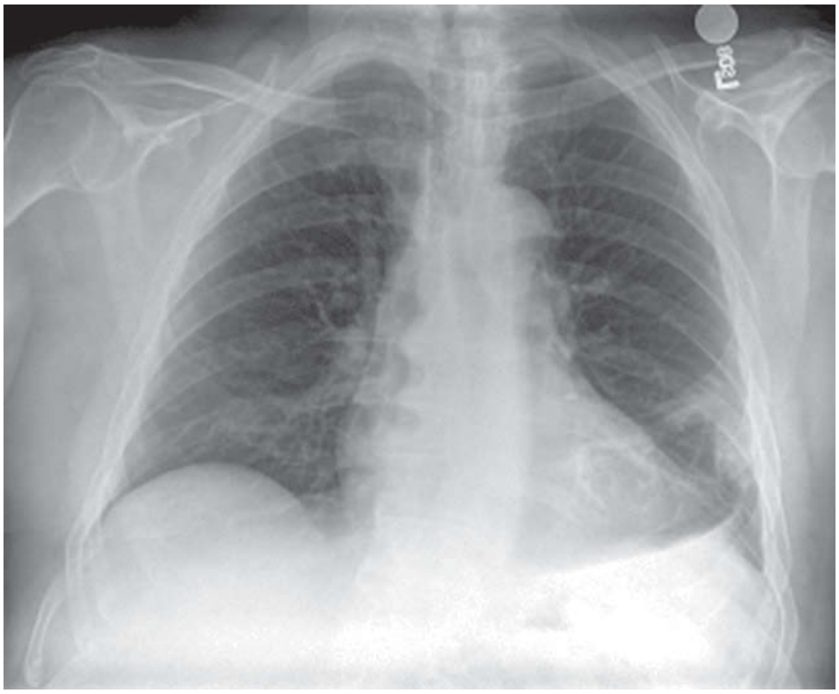


Figure 17.6
Hampton's hump. Reprinted from Tarleton GP, Manthey DE. The elusive Hampton's hump. *J Emerg Med* 2003;24(3):329–30, with permission from Elsevier.

General treatment principles

As with all ED patients, treatment begins with the ABCs (airway, breathing, circulation). The goals of treatment are stabilization, symptom relief, and limitation of morbidity and mortality due to the disease entity.

Patients with chest pain should receive a high triage level, indicating that they have a potentially life-threatening medical problem. They should be placed in a room expeditiously. The initial assessment of the chest pain patient should focus on the patient's stability. If the patient has unstable vital signs or appears ill, an accelerated assessment and treatment plan should be used. Performance and interpretation of an ECG within 10 minutes of arrival to the ED is recommended.

Initial assessment and interventions

- ABCs
- Patient appearance
- Vital signs including O_2 saturation
- Place IV line, administer O_2 and place on cardiac monitor
- ECG within 10 minutes of arrival
- Directed history and physical (H&P); includes pulmonary and cardiovascular examination.

If immediate life-threatening disease is found or suggested, initiate rapid and directed treatment. Otherwise perform a secondary assessment and treatment.

Secondary assessment and interventions

- Complete H&P
- Aspirin 325 mg PO (unless patient allergy, appropriate dose already taken, or ischemia excluded)
- Provide pain relief
- Consider additional (serial) ECGs, radiologic and laboratory evaluation as indicated.

Acute coronary syndrome (ACS)

Aspirin

Aspirin should be given to everyone with suspected ACS who is not allergic. Its efficacy is equivalent to that of costly thrombolytics, and contraindications are infrequent. There is a 23% reduction in 30-day mortality in patients with AMI. In patients with USA, there is a 50% reduction in the rate of progression to AMI.

Dosing: 325 mg oral (or rectal).

Nitrates

Nitrates are recommended in AMI, although a clear benefit on morbidity or mortality has not been proven. Nitrates act to vasodilate the coronary arteries, and reduce both preload and afterload. Hypotension, a frequent and unacceptable adverse effect, should be avoided at all costs; therefore, blood pressure should be monitored before each additional dosage. Sublingual NTG is recommended in patients with suspected ACS, except those with contraindications such as allergy, bradycardia <50 beats per minute (bpm), tachycardia, or hypotension. The use of agents for erectile dysfunction (sildenafil or vardenafil within 24 hours, or tadalafil within 48 hours) is an absolute contraindication to use of nitrates because of the risk of prolonged and exaggerated hypotension. NTG should be used with caution in patients with right ventricular infarct who are often sensitive to preload reduction. IV NTG is indicated in patients with persistent ischemia, CHF, hypertension, or a large anterior AMI.

Dosing: Treatment should begin with sublingual tablet or spray dosing of 0.4 mg every 5 minutes until pain-free. Three doses are commonly recommended but are not a limit. Check blood pressure before each additional dose. If symptoms are relieved with sublingual therapy, apply 1–2 inches of nitropaste to the anterior chest wall. Indications for IV therapy include the first 24–48 hours for patients with definite USA or AMI who experience ongoing or recurrent ischemic discomfort, hypertension, or signs of congestive heart failure, or for controlled titration of therapy. Start an infusion at 10–20 mcg/min and titrate by 10–20 mcg/min every 3–5 minutes until symptom relief.

Morphine sulfate

Morphine is used as an analgesic for the relief of ischemic chest pain. Any patient with significant discomfort should receive treatment with analgesics, although the benefit of narcotics for pain relief in patients with AMI is inferred rather than clearly supported by literature.

Dosing: Depending on the patient's previous exposure to narcotics, an initial IV dose of 2–4 mg is recommended with titration to effect.

Beta-blockers

A large study of 50,000 AMI patients failed to show benefit from immediate beta-blocker treatment in the ED. As a result, immediate treatment with beta-blockers is only recommended in patients with systolic blood pressure (SBP) >140 mmHg without other contraindications, such as signs of heart failure, evidence of a low output state, increased risk for cardiogenic shock, bradycardia or heart block, active asthma, or reactive airway disease. Although any one of the following is not a contraindication to treatment, the greater number of factors present represent a greater risk for cardiogenic shock: (1) age greater than 70 years, (2) systolic blood pressure >120 mmHg, (3) heart rate >110 bpm or <60 bpm, or (4) increased time since onset of symptoms of ST-segment elevation myocardial infarction (STEMI).

Dosing: 5 mg metoprolol is given IV three times at 5-minute intervals. Vital signs should be checked before each dose. If this is tolerated, then 25–50 mg metoprolol is given orally.

Heparin

The significant benefit shown from heparin use in patients with USA was largely from the pre-aspirin era. A meta-analysis comparing heparin plus aspirin to aspirin alone revealed a 2.4% reduction in death or MI that did not reach statistical significance. Heparin is recommended for patients with USA or AMI. It is part of the treatment protocol for most thrombolytic regimens (except streptokinase). Low-molecular-weight heparin can be given subcutaneously without laboratory monitoring, whereas intravenous unfractionated heparin requires adjustment by monitoring of the partial thromboplastin time (PTT). Some authors argue that low-molecular-weight heparin results in lower rates of heparin-induced thrombocytopenia. Bleeding rates and efficacy are equivalent, and when nursing and laboratory costs are included, overall cost of therapy is equivalent. Precautions include extremes of weight (<45 or >100 kg) and renal insufficiency. *Subcutaneous* unfractionated heparin without laboratory monitoring has been shown to be equivalent in safety and efficacy to low-molecular-weight heparin at a fraction of the cost, but this has not become widely accepted or studied.

Dosing: IV unfractionated heparin is given as 60 U/kg (maximum 4,000 U) and an initial infusion of 12 U/kg/hr (maximum 1,000 U/hr). A nomogram should be used for dose adjustment. For low-molecular-weight heparin, Enoxaparin is given 1 mg/kg SQ BID or dalteparin 120 IU/kg SQ BID.

Thrombolysis

Thrombolysis is indicated in patients with AMI with ST-segment elevation (1 mm in two or more contiguous leads) or presumed new left bundle branch block (LBBB) and symptoms <12 hours when timely percutaneous coronary intervention (PCI) is not available (Table 17.8). Relative mortality is reduced by 21%, with the greatest reduction occurring in patients with bundle branch block. There is no evidence of benefit in patients with AMI who lack ST-segment elevation or LBBB; in fact, outcomes may be worse. Every hour of delay to thrombolytics increases death by 1.6 per 1,000 patients treated. Complications, however, are not benign. These include intracranial hemorrhage in 0.5–1.0% of treated patients. Blood transfusions are required in 5–15%. The GUSTO (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) trial of 40,000 patients

Table 17.8 Thrombolysis indications and contraindications

Indications for thrombolysis
- ST-segment elevation (≥1 mm in ≥2 contiguous leads) or *new* left bundle branch block (not known to be old) *and*
- Symptoms <12 hours which are continuing

Contraindications to thrombolysis
- Active internal bleeding (not including menses)
- Suspected aortic dissection
- Uncontrollable hypertension (>180/110 mmHg)
- History of hemorrhagic CVA
- History of non-hemorrhagic CVA within 1 year

Relative contraindications
- Presenting blood pressure >180/110 mmHg
- History of chronic severe hypertension
- Active peptic ulcer
- Pregnancy
- Internal bleeding within 4 weeks
- Noncompressible vascular puncture(s)
- Trauma/surgery or CPR within 2–4 weeks
- Current use of anticoagulants in therapeutic doses (INR ≥ 2–3) or known bleeding diathesis
- History of prior CVA or known intracerebral pathology not mentioned in contraindications

CPR: cardiopulmonary resuscitation; CVA: cerebrovascular accident; INR: international normalized ratio.

provides the main support for the use of alteplase (t-PA) over streptokinase. Thirty-day mortality of alteplase plus heparin was 6.3%, whereas that for streptokinase was 7.3%. Tenecteplase (TNKase) is a modified form of alteplase that can be delivered by weight-based single bolus dosing. Comparison with front-loaded alteplase in the ASSENT-2 (Assessment of the Safety and Efficacy of a New Thrombolytic) trial showed equivalent mortality and complication outcomes.

Dosing: Streptokinase: 1.5 million units IV over 60 minutes.

Alteplase: 15 mg IV bolus, then 0.75 mg/kg (50 mg maximum) over 30 minutes, then 0.5 mg/kg (35 mg maximum) over the next 60 minutes. Concurrent heparin infusion.

Tenecteplase: Single IV bolus over 5 seconds based on body weight: ≤60 kg = 30 mg, 60–69 kg = 35 mg, 70–79 kg = 40 mg, 80–89 kg = 45 mg, ≥90 kg = 50 mg.

Glycoprotein IIB/IIIA inhibitors

Glycoprotein IIB/IIIA (GP IIB/IIIA) inhibitors block platelet aggregation by inhibiting binding of fibrinogen at the GP IIB/IIIA platelet receptor. They have been shown to be of significant benefit when given to patients receiving PCI. It remains controversial whether patients with USA and non–ST-segment elevation MI (NSTEMI) benefit from IIB/IIIA receptor antagonism. Recommended indications for their use include elevated cardiac markers, continuing ischemia, or ongoing ST-segment changes >0.5 mm despite aspirin and heparin therapy.

Percutaneous coronary intervention

PCI is preferred over thrombolysis if performed within 90 minutes of presentation. Operator experience has been shown to have a significant impact on outcome, and high-volume centers have been shown to produce significantly better results. Indications for PCI are the same as for thrombolysis. Additional indications include failure to reperfuse after thrombolytic therapy, cardiogenic shock, persistent electrical instability, severe CHF, and when thrombolysis is contraindicated.

Angiotensin-converting enzyme inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are recommended in patients with AMI (especially with CHF and systolic blood pressure >100 mmHg) based on the ISIS-4 (Fourth International Study of Infarct Survival) and GISSI-3 (Third Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico) trials. Treatment should begin within the first 24 hours but not necessarily in the ED.

Dosing: Begin at the lowest starting dosage for the chosen ACE inhibitor.

Clopidogrel

Clopidogrel is an adenosine diphosphate receptor antagonist that acts to inhibit platelet aggregation. It is indicated instead of aspirin when a patient is aspirin-allergic. It is also recommended as early as possible in patients with ACS (ECG changes or elevated cardiac markers) when urgent catheterization is not planned. When urgent catheterization is planned, clopidogrel can be given in the catheterization lab after the need for coronary artery bypass grafting has been excluded. However, many centers prefer to give clopidogrel in the ED. Bleeding is significantly increased in patients who receive clopidogrel within 3–5 days of coronary artery bypass grafting.

Dosing: 600 mg oral load, then 75 mg/day.

Aortic dissection

The goal of aortic dissection treatment in the ED is to decrease shearing stress on the aorta by decreasing cardiac inotropy and lowering blood pressure. Any patient with a high suspicion for dissection should be started immediately on a beta-blocker, achieving a desired heart rate of 50–60 bpm. Options include metoprolol or labetalol, which has the additional benefit of some alpha-blockade, or esmolol, which has the benefit of an ultra-short acting effect (seconds to minutes). If beta-blockers are contraindicated, as in patients with cocaine use, hypotension, or significant conduction blocks, calcium channel blockers with negative inotropic effects (i.e., diltiazem) should be given. For additional control of SBP, nitroprusside is often recommended.

Management depends on the location of involvement. Dissection that involves any portion of the ascending aorta (Type A) requires emergent surgical repair. If involvement is limited to portions of the aorta distal to the right brachiocephalic takeoff (Type B), attempts at medical management are warranted.

Pulmonary embolism

Initial treatment of PE is with heparin. If IV unfractionated heparin is used, weight-based dosing and treatment algorithms improve the rate of therapeutic heparinization. Patients with subtherapeutic heparinization in the first 24 hours experience up to 15 times the rate of recurrent thromboembolism compared with patients who reach therapeutic anticoagulation. Low-molecular-weight heparins (enoxaparin and tinzaparin) are approved for patients with PE who have documented DVTs. Clinical trials in PE, although limited, show equivalence between heparins in complications and efficacy. Coumadin should be started in the first 24 hours. Coumadin is contraindicated in pregnancy. Weight-based dosing for IV unfractionated heparin is 80 units/kg bolus, followed by an 18 units/kg/hr infusion. The goal is a PTT of 46–70 seconds. For low-molecular-weight heparins, dosing of enoxaparin is 1 mg/kg SQ BID and dosing of tinzaparin is 175 anti-Xa IU/kg SQ daily.

Indications for a *vena caval filter* include recurrent thromboembolism despite adequate anticoagulation, active bleeding or high risk for bleeding, or history of heparin-induced thrombotic thrombocytopenia. Indications for *thrombolysis* include hemodynamic instability due to PE or massive iliofemoral venous thrombosis (phlegmasia cerulea dolens). Indications for *thrombectomy* include chronic thromboembolic pulmonary hypertension or massive PE in patients with contraindication to thrombolysis.

Disposition

Admission versus discharge

Admission rates are high for patients with chest pain, since it is difficult to exclude life-threatening disease without an extended period of observation. Admission rates vary in studies from 30–70%. Any patient with chest pain who has concerning findings, such as abnormal vital signs, an ischemic ECG, or elevated cardiac enzymes requires admission. In addition, any patient with a potentially life-threatening cause for symptoms who is awaiting definitive testing to exclude disease should be admitted (or transferred to a hospital where the study is available) if testing cannot be performed in a reasonable time period. In a patient with possible ACS, evaluation with serial examinations, ECGs, and cardiac marker testing is required. In addition, a noninvasive evaluation such as exercise treadmill testing is needed to exclude USA. If it is possible to obtain these in the setting of a chest pain observation unit, it is not necessary to admit these patients. In a patient with suspected aortic dissection, a normal CT scan of the chest is reassuring for safe discharge if other concerning etiologies have been excluded. In the patient with suspected PE, negative D-dimer results or low-probability ventilation/perfusion scanning excludes disease in the low-risk patient, and negative multi-detector CT pulmonary angiography excludes disease in moderate- to high-suspicion patients. Any patient who is discharged with chest pain should have close follow-up arranged, with clear instructions to return for concerning symptoms such as recurrent or increasing pain, shortness of breath, lightheadedness, neurologic symptoms, or other concerns.

Pearls, pitfalls and myths

- Given the range of potentially life-threatening conditions associated with the complaint of chest pain, the history, physical examination, diagnostic testing and treatment of such patients should proceed in parallel.
- Consider other diagnostic possibilities in addition to cardiac ischemia in patients with chest pain.
- Do not exclude diseases such as PE or ACS simply on the lack of risk factors.
- Recognize the limitation of emergency testing (laboratories, ECG, CXR) to exclude the presence of life-threatening diseases such as ACS, PE and aortic dissection.
- Do not ignore high-risk findings, even in a patient with many low-risk findings.
- Beware of using a single negative cardiac marker or single nondiagnostic ECG to exclude AMI.
- Negative cardiac markers do not exclude USA.

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18 Constipation

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Scope of the problem

Expert consensus defines constipation as at least two of the following in any 12-week period in the previous 12 months (Rome Criteria): fewer than three bowel movements (BMs) per week; hard stool, a sense of incomplete evacuation, or excessive straining in more than 25% of BMs; a need for digital manipulation to facilitate evacuation. Patients perceive being constipated somewhat differently. They use the term to mean straining (81%), hard, pellet-like stools (72%), an inability to defecate when desired (34%), or infrequent defecation (33%). Whatever patients mean by constipation, it should be recognized as a symptom, not a diagnosis.

The prevalence of constipation in the young adult population of industrialized nations is as high as 20%, rising to 30–40% in those over 65 years of age. Women are twice as likely to report constipation than men (18.3% vs. 9.2%), and much more likely to seek care for it (35.6% vs. 19.5%). At least 20% of the population habitually uses over-the-counter laxative preparations.

Approximately 2.7 million ambulatory physician visits in the United States in 2001 had a primary diagnosis of constipation; 20% of these were to an emergency department (ED). Constipation is particularly common in the elderly and in those with multiple medical problems. Constipation is a surprisingly frequent chief complaint in the ED, despite the medical community's perception that it is a "minor" problem. It is important to recognize that constipation may be an adverse outcome of an ED visit, resulting from discharge medications prescribed.

Anatomic essentials

Normal bowel function has two components – colonic transit and defecation. *Colonic transit* is maintained by smooth muscle function via bowel wall myenteric plexuses regulating motility and submucosal plexuses regulating absorption, with overall control by the parasympathetic nervous system. Transit time is also affected by bowel contents, specifically fiber and water.

Defecation is a complex series of events in which rectal distension triggers a series of reflexes to relax sphincters and pelvic floor muscles. This is coordinated with an increase in intra-abdominal pressure to facilitate expulsion of rectal contents. In infants, defecation is entirely a reflex act. Voluntary control of the external anal sphincter is physiologically possible from the second year of life, after which children generally become "toilet trained." Neurologic disease (including spinal cord injury) may obliterate voluntary control of this reflex.

Abnormal bowel function causing constipation may be functional, idiopathic, or primary, occurring in one of three distinct pathophysiologic groups: normal transit constipation with difficulty evacuating (the most common subtype), slow-transit constipation with infrequent BMs and limited urgency, or pelvic floor dysfunction with straining and feelings of incomplete evacuation. Secondary constipation is related to various medical conditions and/or drug side effects.

Complications that may arise from constipation include hemorrhoids, anal fissure (cause or effect), fecal impaction, incontinence ("spurious" diarrhea), rectal prolapse and stercoral (fecal) ulceration.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 18.1).

History

It is essential to establish exactly what the patient means when he/she complains of constipation. Key questions should include:

How often do you have a bowel movement? When was your last bowel movement?

The answers to these questions help establish the exact nature and significance of the patient's complaint. There is a wide variation in the natural frequency of bowel movements. Adults normally have anywhere from three bowel movements per day to one every 3 days. Children may be even more "irregular."

What is the consistency of the stool? Do you have difficulty or pain passing stool?

Patients with normal stool frequency may present due to a change in their stool's consistency, commonly hard and pellet-like. Others strain to pass a BM, or have pain, bloating, or a sensation of incomplete evacuation on defecation. These symptoms are as important as stool frequency in suggesting abnormal bowel function. A history of gradually diminishing or changing stool caliber may indicate an obstruction or mass, possibly due to an anal fissure or colon cancer.

Table 18.1 Constipation red flags

History	Concerning diagnosis
Acute-onset constipation (particularly age over 50 years), obstipation	Colon cancer, diverticulitis, bowel obstruction, inflammatory bowel disease, drug side effect (see Table 18.2), diabetes, hypercalcemia
Significant abdominal pain	Bowel obstruction, diverticulitis, colon cancer
Rectal bleeding	Colon cancer, ischemic bowel, diverticulitis, inflammatory bowel disease
Weight loss (unintentional)	Colon cancer, other malignancy, diabetes
Fatigue	Anemia from gastrointestinal blood loss, hypothyroidism
Vomiting, abdominal distension	Bowel obstruction
Tenesmus	Anal fissure, perianal abscess
Fecal incontinence, diarrhea	Fecal impaction with overflow ("spurious") diarrhea
Change in stool caliber	Colon cancer, anal fissure
Alternating diarrhea and constipation	Colon cancer, irritable bowel syndrome
Medications	Drug side effect (see Table 18.2)
Sweating, headache, flushing in spinal cord injury patient	Autonomic dysreflexia
Newborn, irregular stool pattern	Hirschsprung's, meconium ileus, imperforate anus
History of rectal foreign body insertion	Mechanical obstruction due to rectal foreign body
Examination finding	**Concerning diagnosis**
Abnormal vital signs	Peritonitis, ischemic bowel, stercoral ulceration, bowel obstruction, Ogilvie syndrome (acute colonic pseudo-obstruction)
Cachexia	Malignancy
Dehydration, dry mucous membranes, poor skin turgor, hypotension	Bowel obstruction, hypercalcemia, increased risk of side effect from phosphate or magnesium enema
Focal tenderness, guarding, rebound	Perforation, ischemic bowel, diverticulitis
Abdominal distension, tinkling bowel sounds (borborygmi)	Bowel obstruction
Blood on digital rectal examination	Colon cancer, ischemic bowel, diverticulitis, inflammatory bowel disease
Abnormal neurologic signs	Neurologic etiology (see Table 18.3)
Confusion, hyporeflexia, hypotonia, coma, short QT interval on ECG, dysrhythmias	Hypercalcemia, hyperparathyroidism, malignancy
Anal stenosis, blind pouch or fistula (newborn)	Imperforate anus

How long have you had a problem with constipation?

This information helps identify the acuity of the problem. A new problem or sudden change must prompt rigorous focus on diagnostic evaluation, as acute constipation may indicate an underlying intestinal obstruction due to tumor, stricture, volvulus, drug side effect, or endocrine condition. A problem that is more chronic in nature or has slowly worsened should focus on therapy to relieve symptoms, with attention to diet and lifestyle issues.

Associated symptoms

Although constipation may be the sole presenting complaint, it is frequently part of a complex of symptoms. Clinicians should initially ask open-ended questions about associated symptoms, with subsequent specific inquiry made regarding sentinel associated symptoms.

Abdominal pain

Because constipation is a symptom, it should not be attributed as the cause of abdominal pain without a thorough search for more sinister etiologies, such as obstruction caused by colon cancer or diverticular disease. This is especially important in elderly patients.

Weight loss or fatigue

Weight loss may occur in conjunction with constipation due to malignancy, hypercalcemia, diabetes or hypothyroidism. Fatigue may indicate underlying anemia. Iron deficiency anemia mandates exclusion of gastrointestinal blood loss, particularly from malignancy.

Rectal bleeding or blood-stained stools

Hemorrhoids and minor anal trauma typically cause hematochezia (bright red blood per rectum), and may be common in patients with constipation. However, attributing rectal bleeding or blood-stained stools to either of these is inappropriate. Rather, these findings should prompt endoscopy or other arrangements for urgent evaluation to exclude malignancy, inflammatory bowel disease, or diverticulitis, particularly with increasing age.

Diarrhea, fecal incontinence or flatus

Diarrhea with soiling may suggest underlying constipation with overflow, therefore mandating a rectal examination. Diarrhea alternating with constipation suggests an obstructing colonic lesion or irritable bowel syndrome. Inconsistent bowel habits require investigation for tumor or malabsorption. Inability to pass flatus should raise concern for a bowel obstruction.

Vomiting

Vomiting is not a feature of simple or functional constipation, but may suggest bowel obstruction or an endocrine abnormality that must be excluded.

Tenesmus

Tenesmus is defined as straining, typically ineffective and painful, during attempts at moving the bowels. This may be a symptom of constipation, although it is more common with inflammation and infection, preventing patients from passing stool despite their efforts.

Past medical

Specific inquiry should be made concerning diabetes, renal failure, neurologic disorders, spinal cord lesions, thyroid disease and depression, as constipation is commonly associated with these conditions.

Medications

Patients should be asked what medications they take regularly, both prescribed and over-the-counter (OTC), including herbal medications and treatments. This may identify medications responsible for constipation (Table 18.2), and what treatments have been attempted. Medications responsible for constipation should be discontinued or modified.

Dietary habits

Inadequate dietary intake of fiber and water is responsible for chronic constipation in the majority of patients. Lack of availability and cost may inadvertently start a cycle of constipation. Reduced fluid intake in the elderly may be related to concerns of bladder control. Decreased fluid intake in infants may be related to parental education, breast milk production, or vomiting.

Table 18.2 Medications commonly associated with constipation

Analgesics: Morphine, codeine, tramadol, hydrocodone, other opiates NSAIDs
Medications with anticholinergic properties: Antihistamines Tricyclic antidepressants Phenothiazines (e.g., antipsychotics) Antispasmodics (e.g., hyoscyamine, baclofen, atropine) Antiparkinsonian agents (e.g., dopa)
Antacids (aluminium- or calcium-containing)
Cardiac medications: Diuretics, calcium channel blockers, ACE inhibitors, lipid-lowering agents, clonidine
Miscellaneous: Iron, phenytoin, barium, bismuth, cholestyramine
Laxative abuse (colonic stimulants)

ACE: angiotensin converting enzyme; NSAIDs: nonsteroidal antiinflammatory drugs.

Additional lifestyle factors

Constipation is more likely when immobility due to illness or injury or a sedentary lifestyle exist. Irregular routines such as traveling or shift work also affect bowel function, as will a lack of privacy or inaccessible toilets. Neurovegetative features such as sleep disturbance or anhedonia may suggest depression, which is associated with constipation.

Physical examination

General appearance and vital signs

An individual with uncomplicated constipation should look well, other than general discomfort. Abnormal vital signs or a patient with cachexia, dehydration or significant pain suggests a more serious problem (e.g., bowel obstruction, perforation of a colonic diverticulum, ischemic bowel). Signs of sepsis in a patient with constipation require urgent investigation.

Abdomen

A careful and thorough abdominal examination should be performed. There may be mild distension and tympany, and stool may be palpated in a thin patient. Specific abdominal tenderness, rebound, significant distension, abnormal bowel sounds or signs of localized peritoneal inflammation with guarding and rigidity must prompt an urgent search for underlying pathology. Abdominal scars from prior surgeries should be noted.

Rectal examination

Inspection of the anus may identify a fissure with a sentinel tag or rectal prolapse. Digital rectal examination (DRE) should be performed in every patient, and may reveal blood, tumor, stricture, or fissure. Significant discomfort on DRE suggests an anal fissure from trauma due to hard feces. Anal fissures are a common cause of constipation in young children. Impacted feces may be diagnosed on DRE, which may indicate mechanical obstruction requiring manual disimpaction. With severe impaction due to anal pain, analgesia or sedation may be required.

Head to toe

In the ED, the physical examination should be thorough but focused. Patients should be evaluated for anemia, lymphadenopathy, and signs of endocrine disease, such as diabetes or hypothyroidism. Hypothyroidism may cause coarse facies, fine or brittle hair, hoarse voice, bradycardia, and reflexes with a slow relaxation phase. A focused neurologic examination must also be performed, looking for abnormal motor, sensory, or reflex findings in the legs and perianal region (Table 18.3). Examination of other systems should be made according to historical information as suggested by the abdominal findings. Any ill-appearing patient presenting with constipation, especially if elderly or newborn, deserves a complete physical examination.

Table 18.3 Clinical features suggesting a neurologic cause of constipation

Paraplegia – previous ischemia, trauma, tumor, surgery or congenital cause
Acute spinal pathology – abnormal tone, motor, reflexes or sensation in lower limbs, particularly if bilateral and symmetrical
Autonomic dysfunction – lability of heart rate or blood pressure, orthostatic hypotension, urinary retention, or incontinence
Parkinson's disease – fine tremor, cogwheel rigidity, shuffling gait
Demyelination, polyneuropathy – focal neurologic deficits in any upper motor neuron, spinal or peripheral nerve distribution

Differential diagnosis

The most common causes of constipation are related to diet and lifestyle factors, medications, painful perianal conditions and psychogenic illness (Table 18.4). Less common but important causes include metabolic conditions such as diabetes, hypothyroidism, or hypercalcemia; intrinsic bowel lesions (e.g., tumor or stricture); diverticulitis; and neurogenic disorders. Pediatric causes are largely functional, but include Hirschsprung's, imperforate anus and meconium ileus.

A careful history including search for "alarm symptoms" such as acute onset, significant pain, rectal bleeding, weight loss and anemia should direct physicians to look for underlying causes, especially in patients over 50 years of age.

Table 18.4 Differential diagnosis of the cause of constipation

Most common (etiology frequently multifactorial):
- Inadequate fiber and fluid in the diet
- Lifestyle factors – immobility, ignoring urge to defecate
- Medications (Table 18.2)
- Painful perianal region – hemorrhoids, fissure, abscess
- Irritable bowel syndrome
- Psychogenic – depression, anxiety, eating disorder
- Pregnancy
- Chronic laxative abuse

Less common:
- Metabolic – diabetes, hypothyroidism, hypokalemia, hypercalcemia, renal failure
- Intrinsic bowel lesion – tumor, stricture, ischemia
- Diverticulitis
- Inflammatory bowel disease – Crohn's or ulcerative colitis
- Volvulus, hernia, adhesions, pelvic or abdominal mass
- Neurogenic disorder – autonomic dysfunction including diabetes, multiple sclerosis, Parkinson's disease, spinal cord lesion, amyotrophic lateral sclerosis, cerebral palsy

Uncommon/rare:
- Scleroderma
- Hyperparathyroidism, amyloidosis
- Lead, arsenic poisoning

Pediatric:
- Functional – coercive toilet training, diet, social phobia (common)
- Imperforate anus, colonic or rectal atresia
- Meconium ileus
- Hirschsprung's disease
- Cystic fibrosis
- Intussusception

Diagnostic testing

The history and physical examination allow emergency physicians to determine the urgency of diagnostic testing needed in a patient presenting with constipation. Patients previously investigated who present with an exacerbation of a chronic problem may not need diagnostic testing.

Laboratory studies

Limited data exist to support which blood tests should be performed. A complete blood count (CBC), electrolytes and renal function, liver function tests (LFTs) and thyroid function tests (TFTs) should be ordered as directed by the history and physical examination. This is particularly true in an acute, first-time presentation of an adult patient. Conditions such as hypokalemia and hypercalcemia may cause constipation. Tests of thyroid and renal function are helpful as thyroid disease, renal disease and dehydration may cause or contribute to constipation. Iron deficiency anemia may be a presenting feature of a patient with colon cancer.

Radiologic studies

No studies have addressed the clinical utility of plain abdominal radiography in the routine investigation of constipation. No radiologic tests are indicated unless there is suspicion of an underlying secondary cause, as visualization of "fecal loading" on plain abdominal radiograph rarely changes management and is usually not justified as a diagnostic test in the absence of other indications.

Erect and supine abdominal radiographs may be performed to evaluate for possible bowel obstruction, particularly in a patient with prior abdominal surgery, vomiting, significant abdominal distension, abdominal pain, or an acute or subacute history of constipation. An upright chest film is useful to look for free air under the diaphragm associated with bowel perforation.

Abdominal computed tomography (CT) has a low-yield for the evaluation of constipation, but is often useful for the diagnostic evaluation of abdominal pain, suspected malignancy, obstruction or complicated diverticulitis. A lower intra-abdominal or pelvic abscess may cause a sensation of rectal fullness and constipation.

Outpatient studies

A patient referred to his or her primary care physician from the ED may ask what investigations might be performed as an outpatient. These may include sigmoidoscopy, colonoscopy (particularly if over 50 years of age), colon transit time studies with radio-opaque markers, anorectal manometry, balloon expulsion testing, or defecography.

General treatment principles

The symptom of constipation may be part of a life-threatening emergency with acute abdominal pain, shock, distension or guarding, or a more benign condition without sequelae. Immediate life-threats must be identified and treated first. This might occur in a patient with peritonitis from a perforated colon due to carcinoma or stercoral ulceration, ischemic bowel, or volvulus.

Red flags for serious underlying conditions include abnormal vital signs, an ill-appearing patient, guarding or rebound on abdominal examination, and comorbidities such as advanced age, previous abdominal surgery or chronic steroid treatment. Initial investigations include evaluation of the ABCs (airway, breathing, circulation), resuscitation as indicated, and, when appropriate, consultation with a general or colorectal surgeon.

In a patient in whom constipation is a chronic problem, it is necessary to determine whether this presentation represents an acute crisis or urgent complication. An acute crisis may occur in a patient who develops a bowel obstruction or becomes completely impacted, or in patients with new medical problems, medications or drug dosing. The focus on these patients in the ED is on diagnostic evaluation.

Specific therapy

Treatment must be tailored to the individual, starting with attention to dietary and lifestyle factors. This may be sufficient for mild cases of constipation, and will likely improve the success rate of other treatments. Medications known to cause constipation should be stopped or replaced. Fiber and laxatives increase BM frequency, but there is little evidence to suggest which class of laxative is superior. Laxatives may be considered in broad groups according to their actions, including *bulk laxatives* (e.g., psyllium, methylcellulose, sterculia); *fecal softeners* (e.g., docusate, liquid paraffin); *emollient lubricants* (e.g., mineral oil); *stimulant laxatives* (e.g., sennosides, bisacodyl); and *osmotic laxatives* (e.g., macrogol, lactulose, polyethylene glycol). Regimens for the treatment of constipation are provided in Table 18.5.

Table 18.5 Common regimens for the treatment of constipation

Mild constipation • Senna and docusate (Senokot-S): 2 tablets PO QD for 3–4 days until relief *or* • Psyllium (Metamucil): up to 30 g PO daily in 2–3 divided doses *or* • Magnesium hydroxide (milk of magnesia): 30–60 mL regular strength liquid PO QD
Moderate constipation As above, plus: • Lactulose 15–30 mL (syrup) or 10–20 g (powder for oral solution) PO QD *and/or* • Glycerin: One adult or infant suppository PR as needed *or* • Sodium phosphate (Fleet enema): 1 adult or pediatric enema PR (caution in renal failure or dehydration) *or* • Magnesium citrate 150–300 mL PO divided QD/BID (caution in renal failure or dehydration)
Severe constipation As above, plus: • Polyethylene glycol with electrolytes (GoLYTELY): 2–4 L PO over 4 hours *and/or* • Soap suds enema

BID: two times a day; PO: per os (orally); PR: per rectum; QD: once daily.

Therapeutic manual disimpaction may be required for fecal impaction. Some patients may require sedation, analgesia, or anesthesia for this procedure. Enemas, a form of manual disimpaction, are often successful in the treatment of constipation. Many patients try these at home prior to ED presentation. Often, with the assistance of a nurse, these may be successful. Soap suds enemas or enemas containing warm water, glycerol and magnesium citrate may have positive results when other enema preparations fail. Patients must be somewhat cooperative and be able to hold the enema for success. When enemas fail, they often soften the stool, making manual disimpaction more likely to be successful. Laxative preparations are recommended following such procedures to evacuate the rectum, re-establish normal bowel habits and prevent subsequent constipation.

Special patients

Elderly

Constipation in the elderly population is more common, and more frequently represents serious pathology. It may also be caused by comorbid conditions, medications, polypharmacy, or drug–drug interactions, and is more difficult to treat. Elderly patients may have decreased fluid intake from either access or avoidance in an attempt at bladder control, and are more likely to be sedentary, all contributing to constipation. Elderly patients are less likely able to manage treatment at home, and more likely to develop complications from constipation or its therapy. However, the general principles and approach to constipation are unchanged.

Neurologic disease

Patients with a neuromuscular disorder or spinal cord lesion generally have recurrent problems with constipation. Spinal cord patients can often train defecation reflexes to come under "voluntary" control, such as by stroking their inner thigh.

Spinal cord patients, usually with a high cord lesion, may present with sweating, headache, flushing and a "feeling of doom," and may have a high and/or labile blood pressure. This constellation of symptoms in an ill-appearing patient may be due to autonomic dysreflexia, the result of overwhelming autonomic nervous system stimulation. Fecal impaction with rectal distension is a recognized precipitant of this critical condition and must be treated urgently. Antihypertensive medications are often necessary.

Pediatric

Bowel habits commonly vary in children, so the problem of constipation is more difficult to define. It is typically seen in three age groups: infants weaning (>50% of pediatric patients in some case series), toddlers during toilet training, and school-aged children. In children, the etiology of constipation is more frequently functional or behavioral rather than organic. However, this should not preclude a thorough evaluation and investigation for an underlying cause, particularly if prior treatment has failed.

Presentation of an organic etiology may be nonspecific, including poor feeding, irritability, or even dyspnea. Referral to a pediatrician or family practitioner is essential for ongoing care. Neonates are a special group of patients presenting with constipation, as diagnostic possibilities include imperforate anus, meconium ileus, or Hirschsprung's disease.

Disposition

Review of systems, history, physical examination and social situation should all be considered when determining whether a patient presenting with constipation requires an extensive investigation. If so, it is necessary to decide when (how timely) and where (ED, clinic, or hospital) it should be performed. Inpatient evaluation may be required for a patient with severe symptoms such as pain, or for those with new significant anemia, hypothyroidism, or neurologic deficit on physical examination. Referral to an inpatient or GI specialist should occur after reversible causes of constipation have been treated in the ED.

Outpatient referral to a gastroenterologist should be arranged for a patient with:

- Chronic constipation associated with weight loss, anemia, or change in stool caliber
- Refractory constipation
- Constipation despite appropriate laxative use

Home therapy mandates exclusion of serious medical conditions, such as bowel obstruction, and that comorbidities and social considerations have been taken into account. The ability for self-care, to follow-up with a primary care physician, to administer treatment, and to return to the ED if the problem worsens are extremely important to disposition and symptom resolution.

Detailed discharge instructions are essential, and should include a description of proper diet, fluid intake, important behavioral modifications such as exercise to maintain mobility, and "normalizing" daily routines.

Pearls, pitfalls and myths

- "Constipation is a symptom not a diagnosis." This aphorism encourages thorough evaluation of every patient, and averts misdiagnosis from ascribing an inappropriate label.
- "Alarm" features, particularly in a patient over 50 years of age, include acute onset, significant pain, rectal bleeding, weight loss and anemia. An underlying cause must be sought in these circumstances.
- Abdominal pain should not be attributed to constipation without careful consideration. Both of these are symptoms, not diagnoses.
- Abnormal vital signs and signs of peritonitis on abdominal examination should never be considered due to fecal loading.
- Feces identified on plain radiography is a normal finding – imaging is not an appropriate diagnostic modality for constipation, but should be used to exclude alternate significant pathology that may present with constipation.
- Constipation is most often related to dietary factors, medications and lifestyle, all generally amenable to modification.
- Although a thorough evaluation of a patient presenting with constipation is warranted in all age groups, most patients may be investigated in the outpatient setting in the absence of "alarm" features on history or physical examination. Additional caution should be exercised in patients with significant comorbidities and in the elderly.
- Dehydrated patients or those with renal impairment (creatinine clearance less than 30 mL/min) are at potential risk of serious side effects from sodium phosphate enemas, which may cause

hyperphosphatemia, hypocalcemia, hypernatremia and edema, or magnesium-containing enemas, which may cause severe hypermagnesemia, hypotension, bradycardia, heart block, respiratory depression and coma.

- Constipation may be an adverse outcome of an ED visit, so all discharge medications should be carefully considered, particularly narcotic analgesics. Patients (especially the elderly) should be given advice on preventing constipation, including recommendations for an appropriate bowel regimen.

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19 Crying and irritability

Lee W. Shockley, MD **and Katherine Bakes,** MD

Scope of the problem

Small children cry a lot. They cry and cry and cry. You'd cry too if your repertoire of communication skills were as limited as an infant's. They cry because crying is remarkably effective; there is no other infant behavior that elicits an adult's attention and response more reliably.

Inconsolable crying can be a very challenging presentation for several reasons: the child (usually <2 years of age) may have nonspecific symptoms (or no symptoms at all except for the crying), and the associated diseases can range from benign to life-threatening. The primary focus of the emergency practitioner should be to search for and rule out serious causes of crying and irritability. The benign, more common etiologies should only be established after first eliminating serious etiologies.

Anatomic essentials

Crying is one of the only ways by which an infant can communicate discomfort or distress. In that sense, it is a nonspecific form of communication. However, an infant's cry is more than just a simple distress signal. Studies of the acoustic qualities of an infant's cry indicate that it probably contains "encoded" messages about the state of early neurologic development. The acoustic characteristics of the cry are the result of various muscular factors of the vocal anatomy combined with autonomic influences and central nervous system (CNS) control. The cries of infants who are small for gestational age (SGA) and lack neurologic maturity correlate with their ability to modulate their state and quality of alertness. In such infants, high-pitched cries are associated with a poor ability to modulate alertness. Infants suffering from meningitis, birth asphyxia, or hyperbilirubinemia are classically described as having high-pitched harsh cries delivered in short bursts. The distinctive cri-du-chat ("cry of the cat") is associated with trisomy 18.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 19.1).

History

Given the limited (nonexistent) history that can be provided by an infant, one must rely on the parents and care providers to provide a history. Parents often report a distinctive cry for various situations; the infant may have one cry that communicates hunger and another that communicates fatigue. Abnormal characteristics of an infant's cry often elicit a parental response of concern, prompting a visit to the pediatrician or the emergency department (ED). The complaint of an abnormal cry may be the only clue to a serious condition. However, a careful history can provide clues to the etiology of abnormal crying in about 20% of cases.

The infant's medical history (including birth history, pregnancy complications, hospitalizations, illnesses, surgeries and allergies) should be reviewed. Clinicians should ask about recent medication use and recent illnesses. The parents can provide valuable clues about feeding, urination and bowel habits (including any changes), fever or sick contacts, level of activity, and the ability to be consoled.

How often and how long has your child been crying?

The answer to this question allows a clinician to determine if the duration of crying is abnormal. At 2 weeks of age, a normal infant averages crying for 2 hours/day. By age 6 weeks, it increases to nearly 3 hours/day. Fortunately, crying decreases to about 1 hour per day by 12 weeks of age.

What was happening when the crying began? Who was with the child when it began?

A child who awakens crying may have been bitten by an insect or acquired a hair tourniquet. A child who was alone with an older sibling may have sustained trauma or been forced to ingest something that the sibling is unwilling to divulge.

How is this cry abnormal (duration, pitch, volume)? What activity makes it better or worse? Is your child made worse by rocking ("paradoxically inconsolable")?

Parents are unlikely to bring an infant to an ED for a cry that they believe is normal. They are usually concerned about the character or duration of crying, or if their child is less consolable than usual. The words that the parents use to describe the crying may be very helpful in determining

Table 19.1 Crying and irritability red flags

History	Concerning diagnosis
Crying onset during sleep	Insect bite, hair tourniquet
Crying during urination or diaper change	Meatal ulceration, balanitis
Crying onset in presence of sibling	Trauma, foreign body ingestion
Colicky, episodic pain	Intussusception, volvulus
Other sick contacts, particularly with febrile illnesses	Viral or (less often) bacterial infections
Paradoxical inconsolability (crying worse with lifting or rocking)	Meningitis, peritonitis, fractures, arthritis
Fever	Infection
Vomiting	GI pathology, CNS pathology, infections, swallowed air during vigorous crying
Refusing to eat or drink	Stomatitis, oral trauma, GI pathology, hypoxia
Examination finding	**Concerning diagnosis**
Hypothermia	Exposure, sepsis
Bruising about the ears	Nonaccidental trauma
Frenulum tears	Forcible bottle feeding, nonaccidental trauma
Vertical sausage-shaped mass in the RUQ (although uncommon)	Intussusception
"Steamy" corneal appearance, elevated IOP	Glaucoma
Male: elevated testicle, abnormal position, absent cremasteric reflex	Testicular torsion
Female: tender adnexa, possible palpable mass	Ovarian torsion
Tachypnea, hypoxia	Pneumonia
Redness, warmth, swelling at vaccination site within 24–48 hours	Immunization reaction
Fluorescein uptake, long fingernails	Corneal abrasion

CNS: central nervous system; GI: gastrointestinal; IOP: intraocular pressure; RUQ: right upper quadrant.

the cause. Although there is no single acoustic characteristic that can differentiate a normal from abnormal cry, several characteristic cries that may be associated with neurologic impairment include:

- Very high or low pitch
- Extreme changes in pitch, little or no change in pitch, or rapidly fluctuating sounds
- Extreme variation in length of individual cry bursts
- Very short to very long latency to cry (very high or very low threshold to cry after a stimulus)
- Flat, atonal, or stark cry with no harmonic quality, fullness, or overtones
- Non-harmonic sounds that interrupt the cry sound

Crying with infantile colic characteristically begins in the evening, is not quelled for long by feeding, is often associated with relief accompanied with the passage of flatus or feces, and terminates from apparent exhaustion. Paradoxical inconsolability (an increase in crying associated with efforts at consolation, such as lifting or rocking) is associated with meningitis, peritonitis, fractures and arthritis.

Are your other children ill?

Sick contacts, particularly with febrile illnesses, increase the risk for viral or (less often) bacterial infections.

What is your social situation? Have there been any recent changes in that situation?

Changes in caregivers may elicit distress in children, resulting in crying. Furthermore, the correlation of inconsolable crying with a new caregiver should prompt suspicion of nonaccidental trauma.

Associated symptoms

Fever

Fever provides a strong clue to an infectious etiology. A parent's assessment of whether their child has had a fever has been shown to be highly accurate.

Vomiting

Vomiting is a nonspecific symptom that may be associated with GI pathology, CNS pathology, infections, or as a response to gastric distention brought on by swallowed air during vigorous crying.

Refusing to eat or drink

Refusing to eat or drink may be associated with stomatitis, oral trauma, GI pathology, or hypoxia.

Failure to thrive

Children with infant colic often show accelerated growth. Failure to thrive is inconsistent with the diagnosis of infant colic.

Past medical

Birth history

It is recognized that infants with neurologic immaturity have abnormal cries. Premature and SGA infants may exhibit abnormal or prolonged crying. Infants who were premature are at special risk for infectious etiologies for their inconsolable crying.

Previous illnesses

A history of chronic or recurrent diseases can be valuable. Infants with congenital, anatomic, genetic or metabolic abnormalities may present with inconsolable crying.

Medications, recreational drugs, herbal preparations, access to medications, maternal medications if breastfeeding

It is important to identify the drugs or medications the child may have been exposed to (prescribed, over-the-counter, accidental ingestion, or ingestion through breast milk). Many cultural remedies for various conditions may be dangerous for infants, resulting in inconsolability or excessive crying.

Trauma

Known or occult injuries must be considered in a crying child. A history of how an injury occurred is important and its likelihood should be considered. Injuries inconsistent with the history or the infant's age should heighten the clinician's suspicion for nonaccidental trauma. Similarly, an injury mechanism that is inconsistent with an infant's developmental age should be considered suspicious.

Physical examination

The primary goal of the physical exam is to identify painful conditions that could result in excessive crying. While history alone can identify the diagnosis in 20% of crying infants, a careful physical examination can find the cause in an additional 40% of cases. Although it is important to conduct a thorough physical exam, "high yield" procedures include:

- Inspection underneath clothing
- Palpation of the anterior fontanelle
- Retinal examination
- Eversion of the eyelids
- Fluorescein staining of both cornea
- Otoscopy
- Laryngoscopic examination of the hypopharynx
- Oral examination
- Auscultation of the heart
- Palpation of bones
- Neurologic examination
- Rectal examination

General appearance

The general appearance of an infant should be noted, with particular attention to the child's crying and consolability. Will the child take a bottle? Does feeding calm the child? What is the child's tone and posture? How are the parents reacting to the child? Is the child interactive with the examiner? Does the child's cry intensify with rocking or motion?

Vital signs

Special attention should be paid to the vital signs, especially the heart rate and respiratory rate when crying and when calm. A rectal temperature (particularly in infants younger than 90 days) should be used to assess the child's core temperature. Hypothermia (<36 °C) as well as hyperthermia can be a sign of sepsis. Pulse oximetry should be performed to assess for hypoxia. The child's weight should be measured. This can be helpful in comparison with previous weights to assess hydration and nutritional status. It is also important for assessing the child's growth curve and calculating appropriate medication and fluid doses.

Skin

Undress the child completely to examine for rashes, lesions, or bruising. Evaluating capillary refill and examining the skin for signs of tenting are important assessments of perfusion and hydration status.

Head, ears, eyes, nose and throat

Examine the head, ears, eyes, nose and throat for fullness of the anterior fontanelle, pupillary activity, retinal hemorrhages, otitis media or externa, and foreign bodies. Evert the upper eyelids to identify foreign bodies or lashes under the lid. Apply fluorescein and examine the corneas under ultraviolet light to exclude corneal abrasions. Examine the neck for masses, lymphadenopathy, tenderness or rigidity. Nuchal rigidity, however, is not a reliable finding in infants less than 18 months of age with bacterial meningitis. Pay particular attention to bruising about the ears, a hallmark of nonaccidental trauma, that may indicate more serious but less visible internal injuries.

Dental

Examine the mouth and hypopharynx for new tooth eruptions, trauma, infections, aphthous ulcers and foreign bodies. A tongue depressor or laryngoscope (used as a tongue depressor) may be necessary to visualize the hypopharynx. Inspect the lips for frenulum tears, classically caused by forcible bottle feeding, which may provide a subtle clue to nonaccidental trauma.

Chest

Auscultate the chest for abnormal breath sounds. Assess for tachypnea, grunting, nasal flaring, intercostal retractions, and a prolonged expiratory phase.

Abdomen

The abdomen should be evaluated for bowel activity, distention, tone, tenderness and masses. A mass in the left upper quadrant is suggestive of constipation. A vertical sausage-shaped mass in the right upper quadrant is the classic (although uncommon) finding in intussusception, which often manifests as bouts of crying followed by lethargy in an infant. An olive-shaped mass may be palpated in the epigastrium with pyloric stenosis.

Genitourinary

Examine the genitourinary region in both male and female infants for hernias and masses (Figure 19.1). In boys, examine the genitalia for testicular torsion, paraphimosis and penile strangulation from a hair tourniquet. In girls, examine for trauma as well as clitoral hair tourniquets.

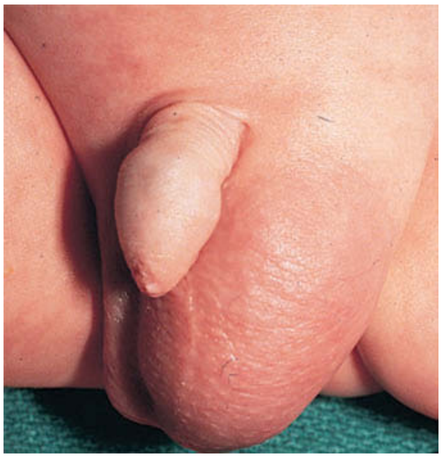

Figure19.1
Left inguinal hernia with erythema of the overlying hemiscrotum. Reprinted from Atlas of Pediatric Physical Diagnosis, 4th ed., Eds Zitelli BJ, Davis HW. Copyright 2002, with permission from Elsevier.

Rectal

Perform a visual inspection of the perineum, looking for blood or fissures. Using a lubricated Q-tip, perform a guaiac exam. Although "currant jelly" stool is the classic finding described in intussusception, it is only evident in 20% of patients and indicates advanced disease (i.e., bowel necrosis). An earlier finding is occult blood present on guaiac testing.

Musculoskeletal

Inspect and palpate all extremities over their entire range of motion. Specifically, examine for signs of arthritis, focal tenderness along bones, edema, erythema, and hair tourniquets on digits (Figure 19.2).

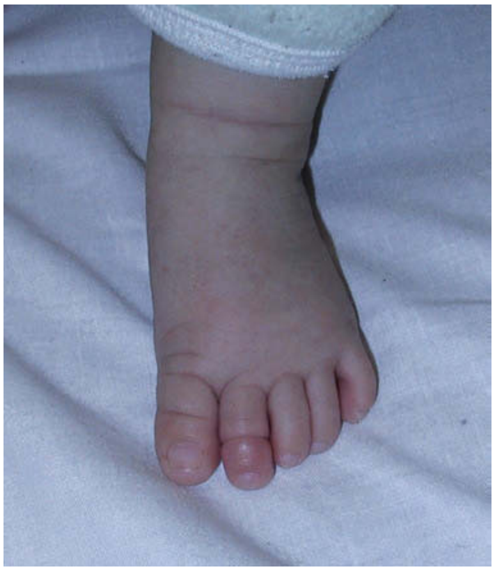

Figure 19.2
Hair tourniquet of 2nd toe. Courtesy: S.V. Mahadevan, MD.

Neurologic

The neurologic examination provides an assessment of the infant's overall level of activity, responsiveness, and ability to be consoled. It should assess movement of all of the extremities, muscle tone, and reflexes appropriate for age.

Differential diagnosis

Table 19.2 provides an extensive list of possible causes of crying and irritability in children.

The most common etiologies identified in a classic study of 56 infants with acute, unexplained, excessive crying are listed in Table 19.3.

Diagnostic testing

In general, diagnostic studies should be ordered based on suspicions raised from the history and physical examination.

Laboratory studies

CBC, blood culture

A complete blood count and blood culture(s) are included in the work-up of a suspected septic infant (Chapter 28).

Table 19.2 Differential diagnosis for crying and irritability

Diagnosis	Symptoms	Signs	Work-up
Congenital/Anatomic			
Anal fissure	Constipation/diarrhea	Blood	Careful H&P, Hgb/Hct
Constipation	Crampy abdominal pain	Hard stool	H&P
Gastric distention	Vomiting	Distended abdomen	Persistent vomiting and weight loss should prompt UGI series or US to rule out pyloric stenosis
Gastroesophageal reflux	Vomiting, difficulty sleeping, respiratory complaints	May be none	Response to treatment
Glaucoma	Crying associated with changes in lighting	May have a "steamy" cornea; elevated IOP	Measure IOP
Incarcerated hernia	Abdominal pain, mass	Hernia on examination	H&P, surgical consultation
Intussusception	Colicky, episodic pain	Stool mixed with blood and mucous is a late finding	Imaging: US, air or barium contrast enema, CT
Meatal ulceration	Crying associated with urination or diaper changes	Ulceration at the urethral meatus	H&P
Peritonitis	Worse crying with movement (rocking, bouncing, car ride, etc.)	May or may not have spasm and rebound	H&P, surgical consultation
Testicular/ovarian torsion	Severe pain, sudden onset, nausea/vomiting	**Males:** Elevated testicle, abnormal position, absent cremasteric reflex **Females:** Tender adnexa, mass may not be palpable	H&P, US, if equivocal examination in males US with Doppler for females
Volvulus	Colicky, episodic pain	Stool mixed with blood and mucous is a late finding	Imaging: plain radiographs, US, air or barium contrast enema, CT
Infectious			
Arthritis	Joint pain, stiffness	Joint swelling, warmth, tenderness	Consider arthrocentesis, inflammatory markers
Balanitis	Crying associated with urination or diaper changes	Uncircumcised male with glans inflammation	H&P, blood sugar
Dermatitis	Pain, worse with wet diaper	Skin irritation, satellite lesions indicate candida	Blood glucose
Gastroenteritis	Vomiting and diarrhea	May be dehydrated	Maintain volume by ORT or IV hydration, if necessary
Meningitis	Vomiting, paradoxical inconsolability, fever (or hypothermia)	Nuchal rigidity is not reliable in young children	LP
Osteomyelitis	Often without a history of penetrating trauma	Tender bones	Lab: inflammatory markers Imaging: plain films, CT, MRI (all may lag clinical picture)
Otitis media/ externa	Ear pain, nasal congestion, pulling at ears	Externa: exudate in auditory canal Media: red, retracted, immobile TM	H&P only
Pneumonia	Cough, fever, poor feeding	Tachypnea, hypoxia	CXR
Sepsis	Fever, chills, altered mental status, apparent life-threatening event (ALTE)	Hypotension, tachycardia, tachypnea, evidence of infection	Age-dependent work-up
Skin infections	Fever, rash	Rash, warmth, redness, tenderness	Glucose, search for trauma
Stomatitis	Refusal to eat or drink	Mouth ulcers or exudates	H&P
Urinary tract infection	Fever, vomiting, abdominal pain	None specific	UA/urine culture

(*continued*)

Table 19.2 Differential diagnosis for crying and irritability (*cont.*)

Toxic, Environmental, Drugs			
Drug reactions	Vomiting, itching	Rash	Diagnosis based primarily on history
Emotional/ physical neglect	Failure to thrive, depression, social withdrawal	Unkempt, unexplained injuries	Consider NAT work-up
Immunization reactions	Tender injection site, fever	Redness, warmth, swelling at vaccination site within 24–48 hours	Diagnosis based primarily on history
Milk intolerance	Nausea, cramping, diarrhea, following milk meal	Bloating, flatulence	Referral for work-up
Neonatal drug withdrawal	Failure to thrive, poor feeding, jitteriness, irritability, vomiting	Piloerection, hyperreflexia	Diagnosis based primarily on history
Prenatal/perinatal cocaine exposure	Failure to thrive, poor feeding, jitteriness, irritability	SGA, hyperreflexia	Diagnosis based primarily on history
Vitamin A toxicity	Nausea and vomiting, headache, dizziness, blurred vision	Loss of muscular coordination	Refer for work-up
Trauma			
Bites	Animal, insect, or human contact	Bite marks	Adult human bites: NAT work-up
Burns	Inciting agent	Burns	Consider NAT
Contusions	Trauma history	Hematoma, ecchymosis	Consider NAT, consider hematologic work-up
Corneal abrasion	May or may not have a history of eye trauma	Fluorescein uptake, long fingernails	Careful H&P
Foreign body (eye, ear, hypopharynx)	May be history of exploratory play	Mucinous, purulent discharge; foreign body seen	Possibly radiographs
Fractures	Trauma history	Point tenderness	Radiographs, consider NAT
Hair tourniquet syndrome	Awakening crying	Examine fingers, toes, penis, clitoris	H&P
Intracranial hemorrhage	Trauma history, vomiting	Altered mental status, signs of head trauma, retinal hemorrhages	CT brain, consider NAT
Open diaper pin	Crying onset after diaper change, worse with movement	Puncture wound, finding the pin	H&P
Genetic/Metabolic			
Electrolyte abnormalities	Irregular heart beat, weakness	Conduction abnormalities, dysrhythmias, volume overload	ECG, lab
Hypocalcemia	Paresthesias, extremity spasm	Petechia, tetany, carpal spasm, Trousseau sign, Chvostek's sign, hyperactive reflexes, laryngospasm, cardiac dysrhythmias	Electrolytes, ECG
Hypoglycemia	Shakiness, sweating, vomiting, headache	Pallor, hypothermia, diaphoresis, lethargy, ataxia, seizure	Search for cause (endogenous or exogenous)
Inborn errors of metabolism	Failure to thrive, neurologic impairment, vomiting, diarrhea	Hypotension, hepatomegaly, jaundice, hyperventilation, unusual facial features	Referral for testing
Phenylketonuria	Impaired growth, impaired neurologic development, seizures	Microcephaly, musty odor	Referral for testing
Sickle cell crisis	Impaired growth	Anemia, hypoxia, dactylitis	Hematologic work-up
Allergic/Inflammatory			
Celiac disease	Vomiting, diarrhea, stunted growth, fatigue	Abdominal distension	Referral for GI work-up
Cow milk allergy	Loose stools (possibly containing blood), vomiting, gagging, refusing food, irritability or colic	Rash	Referral for allergy testing

Table 19.2 Differential diagnosis for crying and irritability (*cont.*)

Functional			
Parental expectations/ responses	Often special-needs child; often new parents	None specific	Diagnosis of exclusion
Miscellaneous			
Aphthous ulcers	Refusal to eat or drink	Mouth ulcers	H&P
Autism	Impaired social interaction and communication, restricted and repetitive behavior	None specific	Refer for neuropsychiatric testing
Caffey disease (infantile cortical hyperostosis)	Refusal to eat, failure to thrive	Soft tissue swelling	Radiographs
Colic	Usually onset < 3 months of age	Flushed face, occasionally circumoral pallor, abdomen distended and tense, legs drawn up, feet often cold, legs may extend periodically, fingers clinched, relief with the passage of flatus or feces	Diagnosis of exclusion
Congestive heart failure	Poor feeding	Cyanosis, tachypnea, hypoxia	CXR, ECG, echocardiogram
Discomfort (cold, heat, itching, hunger)	Other family members may experience discomfort, altered routine, new environment	May be none	Diagnosis of exclusion
Dysrhythmia	Dizziness, syncope, poor feeding	Pulse deficit, irregular pulse, rapid or slow heart rate	ECG, electrolytes
Headache	May be fever, signs of sinusitis, vomiting	Neurologic findings	Depending on suspected etiology: CT, LP, tox screen
Hypoxia	Difficulty feeding	Cyanosis (late), hypoxia, murmur	CXR, ECG, Hgb/Hct
Night terrors	Appears frightened and agitated, amnesia for events	Signs of autonomic discharge	Consider referral for EEG
Overstimulation	Onset when around others, noise, movement	None specific	Diagnosis of exclusion
Persistent night awakening	Altered sleep habits	None specific	Diagnosis of exclusion
Teething	Biting, low-grade fever	Drooling, signs of tooth eruption	H&P
Temperament	Other behavior issues	None specific	Diagnosis of exclusion

CT: computed tomography; CXR: chest X-ray; ECG: electrocardiogram; EEG: electroencephalogram; GI: gastrointestinal; Hct: hematocrit; Hgb: hemoglobin; H&P: history and physical examination; IOP: intraocular pressure; IV: intravenous; LP: lumbar puncture; MRI: magnetic resonance imaging; NAT: nonaccidental trauma; ORT: oral rehydration therapy; SGA: small for gestational age; TM: tympanic membrane; UA: urinalysis; UGI: upper gastrointestinal; US: ultrasound.

Glucose

A rapid bedside fingerstick glucose can rapidly determine hypoglycemia or hyperglycemia. Hypoglycemia in an infant is associated with sepsis, errors of metabolism and certain toxic ingestions. Infants do not have glycogen stores and are therefore prone to develop hypoglycemia. If an inborn error of metabolism is suspected, a serum ammonia and urine ketones should be sent to elucidate the diagnosis. Hyperglycemia may be the first indication of diabetes.

Electrolytes

Serum electrolytes may be indicated in the evaluation of a crying infant. These may help identify hyper- or hyponatremia, hyper- or hypokalemia, metabolic acidosis and hypocalcemia.

Urinalysis/culture

A urinalysis and culture may be indicated in crying or irritable infants. Urinary tract infections are common and may present with nonspecific symptoms, such as inconsolable crying. A catheterized urine specimen is should be collected and assessed.

Lumbar puncture

A lumbar puncture with laboratory evaluation of the cerebrospinal fluid is part of the work-up of an infant with suspected meningitis or encephalitis, and should be strongly considered in any infant who is difficult to console and refuses feeds.

Table 19.3 Diagnoses in children with excessive crying

Diagnosis	Frequency
Idiopathic	18%
Otitis media	18%
Colic	11%
Corneal abrasion	5%
Constipation	5%
Viral illness with anorexia, dehydration	4%
Supraventricular tachycardia	4%
Urinary tract infection	2%
Mild prodrome of gastroenteritis	2%
Herpangina	2%
Herpes stomatitis	2%
Foreign body in the eye	2%
Foreign body in the oropharynx	2%
Tibial fracture	2%
Clavicle fracture	2%
Brown recluse spider bite	2%
Hair tourniquet syndrome	2%
Intussusception	2%
Gastroesophageal reflux with esophagitis	2%
Subdural hematoma	2%
Encephalitis	2%
Pseudotumor cerebri	2%
Vaccine reaction	2%
Inadvertent pseudoephedrine overdose	2%
Night terrors	2%
Overstimulation	2%
Glutaric aciduria, type 1	2%

Note: 61% of the 56 infants in this study had a condition that was considered serious.

Data from Poole SR. The infant with acute, unexplained, excessive crying. *Pediatrics* 1991;88:450–55.

Toxicologic screen

Toxicologic screening may be indicated in infants in whom acute or chronic exposures are suspected. Be aware, however, that many toxins are not included on routine drug screens. Toxicologic screening may also be negative in situations of drug withdrawal.

Amino and organic acid studies

Amino and organic acid studies may be ordered in cases where an inborn error of metabolism is suspected. These tests are rarely indicated in the ED, however.

Liver enzymes

Liver enzyme tests may be used to screen for liver injuries associated with blunt abdominal trauma. If the clinical suspicion of abdominal trauma is high or liver enzymes are elevated, a clinician should proceed to further imaging studies, such as computed tomography (CT).

Electrocardiogram

An electrocardiogram (ECG) is indicated to evaluate an infant with an abnormally fast or slow heart rate or an irregular pulse. A history of sweating or gasping with feeds may be the only clue to an underlying cardiac defect. Clinicians may have an increased suspicion based on a failure to thrive as well as hepatomegaly, abnormal cardiac findings on auscultation, or pulse differences between upper and lower extremities (seen in coarctation of the aorta). Infants do not develop pitting edema often found in adults with heart failure.

Radiologic studies

Skeletal X-rays

Focal areas of tenderness or deformity should prompt the ordering of skeletal X-rays. In addition, a "skeletal survey" is useful to look for signs of previous trauma. Several patterns of skeletal injuries are highly correlated with nonaccidental trauma (NAT). These include but are not limited to posterior rib fractures, scapular fractures, non-linear skull fractures and long-bone fractures (particularly in non-ambulatory infants) (Table 19.4).

Table 19.4 Skeletal injuries associated with nonaccidental trauma

Spiral fracture of a long bone (Figure 19.3)
Metaphyseal chip fracture
Multiple fractures at different stages of healing
Fractures at unusual sites, such as ribs, lateral clavicle, sternum, or scapula

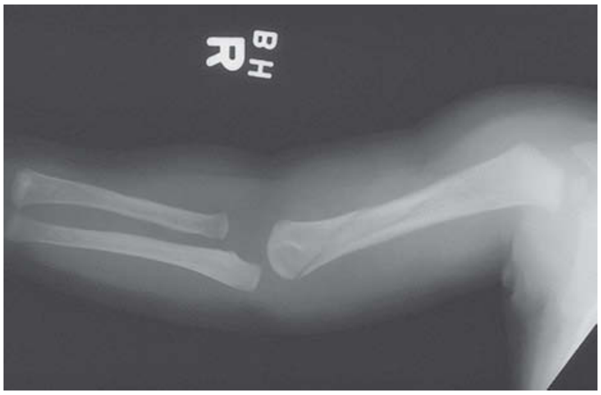

Figure 19.3
Spiral fracture. A spiral fracture courses from the distal portion to the upper third of the diaphysis. There is moderate soft-tissue swelling. Reprinted from Atlas of Pediatric Physical Diagnosis, 4th ed., Eds Zitelli BJ, Davis HW. Copyright 2002, with permission from Elsevier.

Chest X-ray

A chest X-ray may be useful for the diagnosis of pneumonia or aspirated foreign bodies. Radiolucent foreign bodies should be suspected on the basis of air-trapping, best seen on an expiratory view of the chest. In the crying infant, it may be extremely difficult to coordinate timing of the film exposure with expiration; in such cases, lateral decubitus films may be obtained. Using this approach, the dependent lung should be compressed by the child's own weight, simulating expiration.

Head computed tomography

In an infant with an abnormal neurologic examination (especially lateralizing or focal findings), retinal hemorrhages or other signs of head trauma, a head CT is indicated. This study will provide information about brain injuries (contusions and hematomas) as well as skull fractures. It may also be helpful in diagnosing hydrocephalus or other congenital abnormalities. An inconsolably crying infant may have to be sedated and closely monitored in order to obtain the study.

Air enema

An air contrast enema examination is indicated when there is concern for intussusception. It is not only diagnostic, but also often therapeutic, leading to reduction of the intussusception. Barium enema is used at some institutions, as success rates are historically better with this modality. However, due to increased morbidity associated with perforation and barium extravasation, many centers are moving to air enema studies only. Consultation with a pediatric surgeon and radiologist is warranted prior to the study in case of bowel perforation, which would require emergent surgical intervention. Abdominal ultrasonography may have diagnostic utility, but is only reliable in the hands of an experienced sonographer.

Esophagram

An esophagram may be necessary to diagnose esophageal abnormalities (tracheoesophageal fistula, webs, reflux), but is rarely necessary from the ED.

General treatment principles

The treatment of an infant with crying and irritability will be dictated by the ultimate diagnosis. There is no single medication that can or should be recommended to console a crying infant, because a wide variety of conditions and broad spectrum of diseases may present in this way, each requiring different treatment.

Special patients

Infant colic

Infant colic is a syndrome characterized by:

- Sudden attacks, usually in the evening
- Loud, almost continuous cry
- Episodes lasting more than 3 hours per day at least 3 days a week for 3 or more weeks
- Face is flushed occasionally circumoral pallor
- Abdomen is distended and tense
- Legs drawn up, feet are often cold, legs may extend periodically during forceful cries
- Fingers are clenched
- Relief with the passage of flatus or feces
- Not quelled for long by feeding
- Terminates from apparent exhaustion

The etiology of infant colic is not well understood. This condition affects up to 20% of newborns. Infant colic is most common at 1 month of life, typically peaks at 6 weeks and most often subsides by 3 months of age.

Several treatments have been advocated for infant colic, including:

- Use of a pacifier
- Rocking the infant
- Softly massaging the infant's back or abdomen
- Playing relaxing music
- Simethicone drops
- Changing the infant's diet or feeding schedule/ techniques

None of these, of course, is guaranteed to work.

Nonaccidental trauma

Risk factors for nonaccidental trauma include:

- Unwanted children: accidental pregnancies, illegitimate births, the opposite sex from what the parents desired, being born during periods of crisis or a former relationship
- Difficult to rear: poor feeders, fussy behavior, abnormal sleep patterns, excessive crying, developmental delay, hyperactivity, behavior disorders, handicaps, or chronic disease
- Poor maternal-child bonding: premature infants, infants separated from their mothers because of illness or incarceration, stepchildren, foster children, or very young parental age
- A parent who abuses alcohol or other drugs (these may become an excuse for abuse)
- Intergenerational patterns of abuse
- A parent with poor impulse control, or very rigid and unrealistic expectations of children

Findings that increase suspicion for nonaccidental trauma:

- Inconsistent histories
- Alleged self-inflicted injuries
- Unexplained injuries
- Accusatory histories
- Discrepant histories
- Histories inconsistent with developmental age of the child (3-week-old infant who "rolled out of bed")
- Delays in seeking medical care
- Past histories of abuse (or abused siblings)
- Presentations for apparently unrelated complaints
- Unusual bruising
- Unusual burns

- Skeletal injuries in different stages of healing, multiple fractures, metaphyseal injuries, features suggestive of NAT, or an exaggerated periosteal reaction
- Intracranial injuries
- Retinal hemorrhages
- Intra-abdominal injuries
- Renal injuries
- Bruising and laceration of the upper lip, frenulum, or floor of the mouth
- Psychiatric complaints
- Developmental delays

Disposition

The disposition of an infant with crying and irritability is dictated by the ultimate diagnosis. Many of the causes are serious and should prompt consultation and hospitalization. Some etiologies are benign and can be followed as an outpatient. If no etiology can be established after a thorough work-up, the family is reliable and can return with the infant should the condition change, and adequate follow-up arrangements can be made, an infant can be discharged without a definitive diagnosis. If the decision to discharge the patient is contemplated, it should be done only after a period of observation in the ED (2–4 hours). It is reasonable to allow an exhausted parent to go home and sleep while an infant with colic is cared for by ED staff. This has been advocated in the setting where parental fatigue and anxiety is particularly high and staff resources can absorb this function, thus allowing an exhausted and stressed parent to recuperate temporarily.

Pearls, pitfalls and myths

- Always ask about medication use, including over-the-counter medications and medications used by the mother during breastfeeding.
- Listen to the parents' descriptions of the cry and document their words. Specifically, how is the cry different or more concerning than the infant's typical cry? This may be helpful in making the diagnosis.
- Undress the infant completely and perform a comprehensive physical examination.
- The possibility of nonaccidental trauma *must* always be considered in an infant with excessive or inconsolable crying.
- The diagnosis of colic should be made only after a process of elimination, first ruling out more dangerous causes of excessive crying.
- Paradoxical inconsolability is almost always due to a serious pathologic condition (e.g., meningitis, peritonitis, fractures, or arthritis).
- Every infant who is not admitted needs reliable follow-up arranged prior to leaving the ED. The discharge instructions should include a list of things that should prompt immediate return to the ED. When returning for further or repeat evaluation, parents should never be made to feel uncomfortable. In fact, parents should be encouraged to return if their child's crying is causing them emotional distress.
- Sedatives (chloral hydrate, phenobarbital, alcohol, antihistamines) should never be used to treat inconsolable crying.
- All 50 states have laws requiring mandatory reporting of *suspected* nonaccidental trauma in children. These cases should be referred to designated child protective services for investigation.

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20 Dental pain

Kip Benko, MD

Scope of the problem

Between 1997 and 2000, there were approximately 3 million emergency department (ED) visits for dental-related complaints. Emergency physicians know all too well that complaints pertaining to teeth are common. They range in severity from simple odontalgia (toothache) to life-threatening deep space infections. Although patients generally understand that dental specialists provide definitive care, they commonly present to the ED because of severe pain, acute trauma, a lack of financial resources, or an inability to contact or gain access to their (or any) dentist. Treating dental emergencies in the ED is challenging, but also tremendously satisfying if the clinician understands dental anatomy and has familiarity with techniques required to relieve dental pain and preserve teeth.

Anatomic essentials

A thorough understanding of dental anatomy is essential for treatment of dental emergencies and effective communication with dental consultants (Figure 20.1). Adult permanent dentition consists of 32 teeth – eight incisors, four canines (cuspids), eight premolars (bicuspids) and 12 molars. Starting from the midline to the back of the mouth are a central incisor, a lateral incisor, a canine, two premolars and three molars; the last molar is the troublesome wisdom tooth. Though the teeth are numbered (Figure 20.2), it is more important to be able to describe the involved tooth (i.e., upper right lateral incisor) than determine its number.

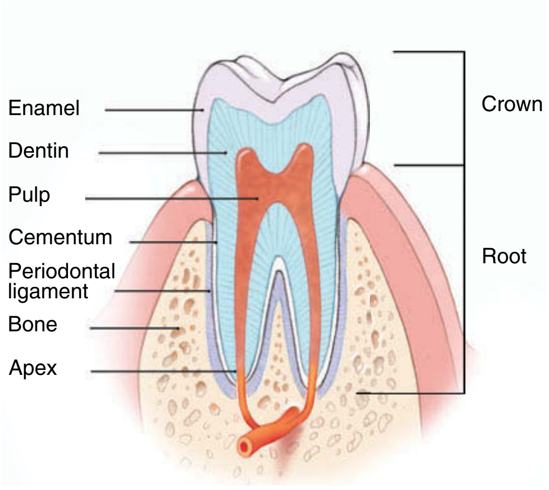

Figure 20.1
Tooth anatomy. © Chris Gralapp.

The primary ("baby") teeth are also best described by the involved tooth, not their official classification. At 4–8 months, the central incisors erupt. Children usually have a full set of primary teeth at about 3 years of age. The permanent teeth start replacing the primary teeth beginning with the incisors at approximately 5 years of age (Table 20.1).

A tooth consists of a central pulp surrounded by dentin and enamel. The pulp contains the tooth's neurovascular supply; the dentin consists of porous microtubules that carry nutrients from the pulp to the enamel. The dentin makes up the majority of the tooth and cushions it during mastication. The enamel is the white part of the tooth visible to the eye, and is the hardest part of the body. The tooth is often described in terms of the crown or the root. The crown is covered in enamel; the root serves to anchor the tooth to the alveolar bone.

Every portion of each tooth has a name. Understanding basic nomenclature improves communication with consultants (Table 20.2).

The periodontium, also known as the attachment apparatus, consists of two major subunits and maintains the integrity of the dentoalveolar unit. The gingival subunit consists of gingival tissue and junctional epithelium. The periodontal subunit consists of periodontal ligament, alveolar bone, and cementum of the root of the tooth.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 20.3).

History

The focused dental history should provide enough data to suggest a reasonable diagnosis while simultaneously ruling out potential life- or tooth-threatening emergencies.

In the patient presenting with dental pain, the following questions should be asked if trauma was involved:

When did the incident occur?

Time is critically important when evaluating avulsed permanent teeth, as the decision to reimplant an avulsed tooth is based largely on the amount of time the tooth is outside the oral cavity.

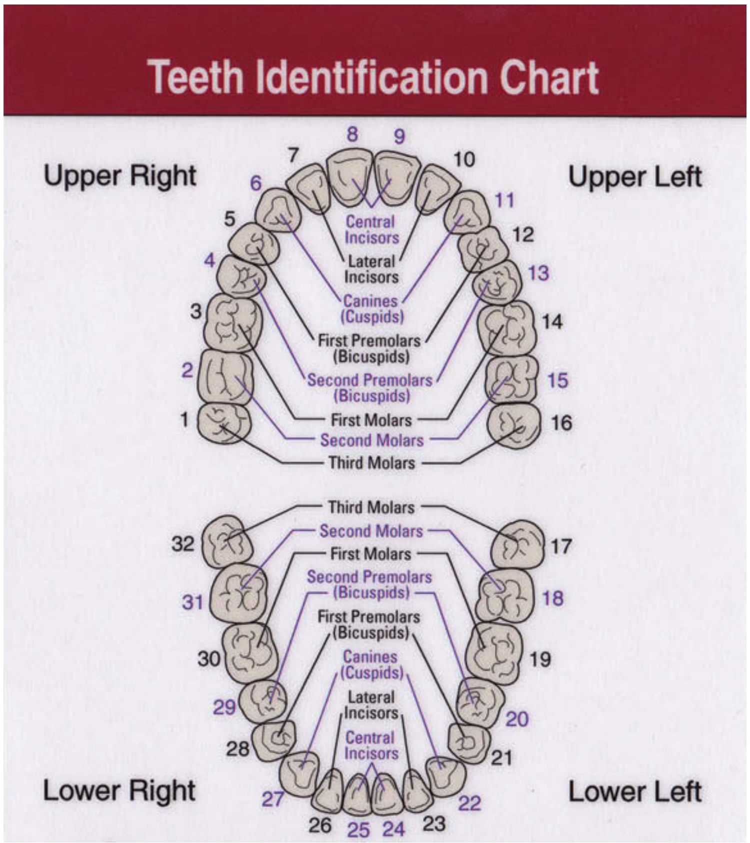
Teeth Identification Chart
Upper Right
Upper Left
8
9
7
10
6
11
5
12
4
13
3
14
2
15
1
16
Central Incisors
Lateral Incisors
Canines (Cuspids)
First Premolars (Bicuspids)
Second Premolars (Bicuspids)
First Molars
Second Molars
Third Molars
Third Molars
Second Molars
First Molars
Second Premolars (Bicuspids)
First Premolars (Bicuspids)
Canines (Cuspids)
Lateral Incisors
Central Incisors
32
17
31
18
30
19
29
20
28
21
27
22
26
25
24
23
Lower Right
Lower Left

Figure 20.2
Adult dentition. With permission from The Dental Box Company, Inc.

Table 20.1 Classification of primary and permanent teeth

Baby (primary) teeth		
Tooth designation	**Name of tooth**	**Appearance in the mouth**
A	Central incisor	4–14 months
B	Lateral incisor	8–18 months
C	Canine tooth	14–24 months
D	First molar	10–20 months
E	Second molar	20–36 months
Adult (permanent) teeth		
Tooth designation	**Name of tooth**	**Appearance in the mouth**
1	Central incisor	5–9 years
2	Lateral incisor	6–10 years
3	Canine tooth	8 1/2–14 years
4	First premolar (bicuspid)	9–14 years
5	Second premolar (bicuspid)	10–15 years
6	First molar (6-year molar)	5–9 years
7	Second molar (12-year molar)	10–15 years
8	Third molar (wisdom tooth)	17–25 years

Table 20.2 Tooth nomenclature

The anatomic surfaces of the tooth include:

Facial: portion of the tooth you see when an individual smiles. This is a general term applicable to all the teeth.
The following terms are more precise:
- *Labial*: refers to the facial surface of the canines and incisors
- *Buccal*: refers to the facial surface of the premolars and molars

Oral: portion of the tooth that faces the tongue or palate. This is a general term applicable to all teeth.

The following terms are more precise:
- *Lingual*: toward the tongue, the oral surface of the mandibular teeth
- *Palatal*: toward the palate, the oral surface of the maxillary teeth
- *Approximal/interproximal*: the contacting surfaces between adjacent teeth
- *Mesial*: the interproximal surface of the tooth facing anteriorly or closest to the midline
- *Distal*: the interproximal surface facing posteriorly or away from the midline
- *Occlusal*: the biting or chewing surface of the premolars and molars
- *Incisal*: the biting or chewing surface of the incisors and canines
- *Apical*: toward the root of the tooth
- *Coronal*: toward the crown or the biting surface of the tooth

Table 20.3 Dental pain red flags

History	Concerning diagnosis
Trauma	Avulsed, subluxed, or displaced tooth; tooth fracture; malocclusion; mandibular (or maxilla) fracture
Recent dental extraction	Alveolar osteitis ("dry socket"), hemorrhage, postextraction pain secondary to insufficient analgesia, infection
Diabetes, malnutrition, known dental problems (such as caries or poor dentition), alcoholism	Odontogenic infection
History of heart murmurs or known cardiac valve problems, intravenous drug use	Endocarditis
Change in voice, difficulty swallowing or speaking	Deep space infection, including Ludwig's angina, peritonsillar abscess, retropharyngeal abscess
Tooth sensitivity to cold or heat	Dental abscess, tooth fracture
Examination finding	**Concerning diagnosis**
Fever, neck mass, overlying erythema, malnutrition, poor dental hygiene, track or needle marks	Deep-space neck abscess, Lemierre's syndrome
Nuchal rigidity	Deep-space abscess, sepsis, meningitis
Trismus	Deep-space abscess
Tongue elevation, brawny discoloration of submandibular space	Ludwig's angina, possible airway compromise
Irregularity of teeth spacing, appearance, alignment, malocclusion	Dental fracture, avulsion, luxation, subluxation, intrusion, mandible fracture/dislocation
Abnormal pulmonary examination	Aspiration, Lemierre's syndrome
Tenderness to palpation	Dental abscess, mass, cavity, exposed nerve root

Were any teeth found at the scene?

If so, were they transported with the patient, and how were they transported?

Did symptoms suggesting tooth aspiration?

Coughing and choking suggest possible tooth aspiration. A decreased level of consciousness from drugs, alcohol, or trauma makes aspiration more likely.

Does the patient complain of pain, and is the pain associated with occlusion? Do the teeth feel as if they are touching normally during occlusion?

Mandibular fractures are often worse with jaw movement and are commonly associated with malocclusion. Pain from temporomandibular joint (TMJ) injuries is often referred to the ear. Roughness, pain, or a newly irregular surface when the patient rubs his or her tongue over the teeth suggests a dental fracture.

What did the patient do for the pain?

Over-the-counter anesthetics and analgesics are often used to decrease pain, but can cause sterile abscesses if applied to the pulp or dentin.

Is the tooth a primary or permanent tooth?

Traumatized primary teeth are managed more conservatively than permanent teeth.

Does the patient have a bleeding disorder?

This question is important in patients who experience dental trauma. Bleeding may not stop on its own if a platelet disorder exists, or severe hemorrhage may accompany an underlying coagulopathy.

The following questions should be asked for patients with atraumatic dental complaints:

Has there been any recent dental work or instrumentation performed?

Alveolar osteitis (dry socket) may present with pain 2–4 days following tooth extraction.

Does the patient have a history of poor dentition or multiple caries?

This increases the likelihood of dental abscesses, dental fractures, or other odontogenic infections.

Is there any difficulty opening the mouth or swallowing, voice changes, or shortness of breath? Is the patient at risk for being immunocompromised?

Dental infections may spread and become deep space infections, which often result in trismus, difficulty swallowing, or difficulty speaking. Tongue elevation may be seen with Ludwig's angina, and suppurative thrombophlebitis from dental infections may result in spread to the neck or lungs. Deep space infections can rapidly progress to the mediastinum or cavernous sinus in immunocompromised patients.

Does the patient have a history of rheumatic fever or valvular disease (such as mitral valve prolapse)? Does the patient have implanted devices such as artificial joints, cardiac valves or shunts?

Dental infections may predispose such patients to endocarditis or implant infections.

Is there a history of drug allergies?

Allergies to antibiotics (especially penicillin or clindamycin), anesthetic agents (lidocaine), or pain medications (ibuprofen, Vicodin) are important to identify, as these medications may be used in the treatment of dental complaints.

Physical examination

The examination of the oral cavity in patients presenting with tooth or facial pain must be thorough and meticulous. A bright light, tongue depressor and cooperative patient are integral to a successful physical examination. A dental mirror may also be helpful. Injuries to the dentition may be missed because of more impressive traumatic findings elsewhere, or because of an incomplete examination. The nooks and crannies of the mouth can hide fairly significant abscesses, foreign bodies, or injuries.

General appearance

Simple observation and discussion often provide clues to a patient's diagnosis. Voice change, muffling, drooling or other signs of airway involvement must be addressed. External inspection may disclose injuries such as mandibular dislocations and fractures, as these often result in asymmetry, swelling, or deformity of the face. Abscesses or deep space infections often result in swelling over the involved space, although this may be subtle. Therefore, the face should be inspected from multiple angles. Mouth movements should be smooth and complete without hesitation or limitations. Erythema or drainage suggests cellulitis, abscess or hematoma formation.

Vital signs

Fever and tachycardia are nonspecific signs, but may suggest an abscess or significant pain. Hypotension suggests an overwhelming infection, such as sepsis, or volume depletion.

Oropharynx

The oral cavity should be examined for bleeding, swelling, dental step-offs, tenderness, abrasions or lacerations. Each tooth should be accounted for. The buccal mucosa and mucobuccal folds should be visualized with a tongue blade and a strong light; blood should be wiped or suctioned away for better visualization. The cheek and the floor of the mouth should be palpated with a gloved hand, with any swelling or tenderness noted. If there has been dental or facial trauma, each tooth should be percussed with a tongue blade for sensitivity and palpated with fingers or tongue blades for mobility. Blood in the gingival crevice (where gingiva contacts enamel) suggests a traumatized tooth or an underlying mandibular fracture. The mandible and maxilla should be gently rocked to assess stability.

Teeth

The teeth should meet evenly and symmetrically when biting, and the patient should be able to exert firm pressure on a tongue blade with his or her molars. The inability to crack a tongue blade when twisted between the molars (tongue blade test) on either side suggests a mandibular fracture.

Neck and face

The neck and face should be inspected and palpated for swelling, erythema or tenderness suggesting a fracture, cellulitis, abscess or hematoma. Careful palpation of the mandible and maxilla are essential.

Differential diagnosis

Sources of non-odontogenic tooth pain are listed in Table 20.4.

Dental trauma

Fractures

Injury to maxillary central incisors accounts for 70–80% of all fractured teeth. Although not life-threatening, the morbidity associated with dental fractures can be significant and includes incomplete eruption, abscess, loss of space in the dental arch, tooth discoloration, ankylosis, abnormal exfoliation and root resorption.

There are many ways to classify dentoalveolar injuries and, in particular, tooth fractures. Crown fractures may be divided into complicated and uncomplicated categories. Uncomplicated crown fractures involve the enamel alone or the dentin and enamel; complicated crown fractures involve the pulp.

Uncomplicated crown fractures through the enamel only (Figure 20.3) are not usually sensitive to forced air, temperature, or percussion and typically pose no threat to the dental pulp.

Uncomplicated crown fractures through the enamel and dentin (Figures 20.4a and 20.4b) are at higher risk of pulp necrosis and need more aggressive treatment by emergency physicians. The risk of pulp necrosis (1–7%)

Table 20.4 Sources of non-odontogenic tooth pain

Diagnosis	Symptoms	Signs	Diagnostic evaluation
Cardiac sources	Typically presents with chest, arm or neck pain, but can present as isolated odontalgia. Pain is often aching or pressure-like and pulsatile, although may be intermittent.	Cardiac pain cannot be elicited or reproduced with provocation of teeth.	May be reduced with nitroglycerin, not with dental blockade.
Myofascial sources	Diffuse, constant, dull, aching sensation. May worsen with chewing.	Pain is triggered by contraction of masticatory muscles. Palpation of these muscles reproduces pain, percussion of the teeth does not.	Injection of trigger points often relieves pain.
Neurovascular sources	Migraine/cluster tend to have headache as the main symptom. Cluster headaches may present as isolated dental pain. Often there are intervals of pain-free syndromes between attacks.	Isolated tooth percussion pain is unusual. Photophobia or tearing more consistent with neurovascular sources.	Resolution with oxygen or traditional migraine treatment suggests a neurovascular cause. Intermittent nature of clusters may not make dental blockade a useful diagnostic test.
Neuralgia	Presentations vary (sharp, shooting pains with trigger zones suggest trigeminal neuralgia). Trigger zones may be intraoral and occur with chewing. Trigeminal neuralgia usually occurs in patients >50 years of age.	Symptoms occur with reproduction of the trigger. Lack of percussion tenderness.	Most patients with trigeminal neuralgia improve dramatically with Tegretol (carbamazepine). Dental blockade does not relieve symptoms.
Neuroma	Presentation similar to trigeminal neuralgia, except history of nerve injury must be present. Also, area peripheral to neuroma is dysesthetic or anesthetic. Can occur several months after tooth extraction.	Tinel's sign is often positive.	Dental blockade may relieve symptoms depending on which nerve has the neuroma.
Neuritis	Inflammation of the nerve from viral, bacterial or traumatic sources. Neuritis pain is typically persistent, nonpulsatile burning. Sinus infection may also cause neuritis. Certain dental procedures and chemicals also can cause neuritis.	May be associated with skin findings if viral etiology.	Difficult to diagnose; treatment is based on etiology. Rule out immediately obvious causes (zoster, sinusitis). Dental blockade may be beneficial depending upon the nerve involved.
Neuropathy	Constant pain in varying degrees of intensity in the focal area.	Area may show hyperalgesia or allodynia.	Dental blockade will be of benefit if the neuropathy is peripheral, but ineffective for central neuropathy.
Psychogenic sources/factitious disorders/ malingering	Differs from other painful conditions. Often moves around and affects multiple teeth. Worsens with stress. Psychogenic pain differs from factitious pain or malingering.	No evidence of physical cause. No local tissue damage. Findings on examination are not completely reproducible.	Response to therapy differs. Psychogenic pain differs from malingering in that there is no obvious benefit derived. Early psychiatry referral important to prevent unnecessary dental treatment.
Sinus/nasal mucosa sources	Aching, dull pain over in the mid-face area or upper teeth, often associated with congestion or nasal drainage. Pain worse when head is placed lower than the heart.	Tenderness to percussion or palpation over the sinuses or over several maxillary teeth, not just one isolated tooth.	Symptoms do not get better with dental anesthetic blockade.

increases as the treatment time extends beyond 24 hours. Patients often complain of sensitivity to extremes of temperature or forced air. Physical examination reveals the yellow tint of the dentin (compared with the white hue of enamel). Fractures closer to the pulp cavity will reveal a pink tinge to the dentin. These patients are usually sensitive to percussion with a tongue blade. The porous nature of the dentin allows bacteria to pass from the oral cavity to the pulp, which may result in inflammation and infection of the pulp chamber. This occurs most commonly after 24 hours, but may occur sooner if the fracture site is closer to the pulp. Similarly, patients less than 12 years of age have a higher pulp/dentin ratio than adults and are at increased risk for pulp contamination. Younger patients should be treated aggressively and be seen by their dentist within 24 hours.

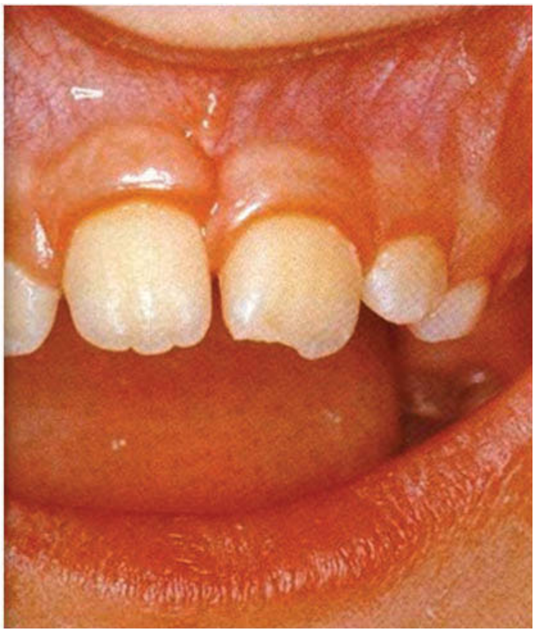

Figure 20.3
Uncomplicated crown fracture through the enamel.

Complicated crown fractures involving the pulp are true dental emergencies (Figures 20.5a and 20.5b). These fractures result in pulp necrosis in 10–30% of cases, even with appropriate treatment. *They are distinguished from fractures of the dentin by the pink color of the pulp.* The fracture surface of the tooth should be wiped off with gauze and observed for frank bleeding or a pink blush, indicating pulp exposure. Fractures through the pulp are often excruciatingly painful, but occasionally lack sensitivity secondary to disruption of the tooth's neurovascular supply.

Subluxation and luxation

Subluxation refers to teeth that are mobile but not displaced (Figure 20.6). *Luxation* refers to teeth that are displaced, either partially or completely, from their sockets. Luxation injuries are divided into four types:

Extrusion luxation: the tooth is forced partially out of the socket in an axial direction (Figure 20.7).

Intrusive luxation: the tooth is forced apically into the alveolar bone and may be accompanied by crushing or fracture of the apex of the tooth (Figure 20.8). These often result in disruption of the attachment apparatus or fracture of the supporting alveolar bone.

Lateral luxation: the tooth is displaced either facially, mesially, lingually or distally. This injury is often associated with injuries to the alveolar wall.

Complete luxation: the tooth is entirely lost (avulsed) from the socket.

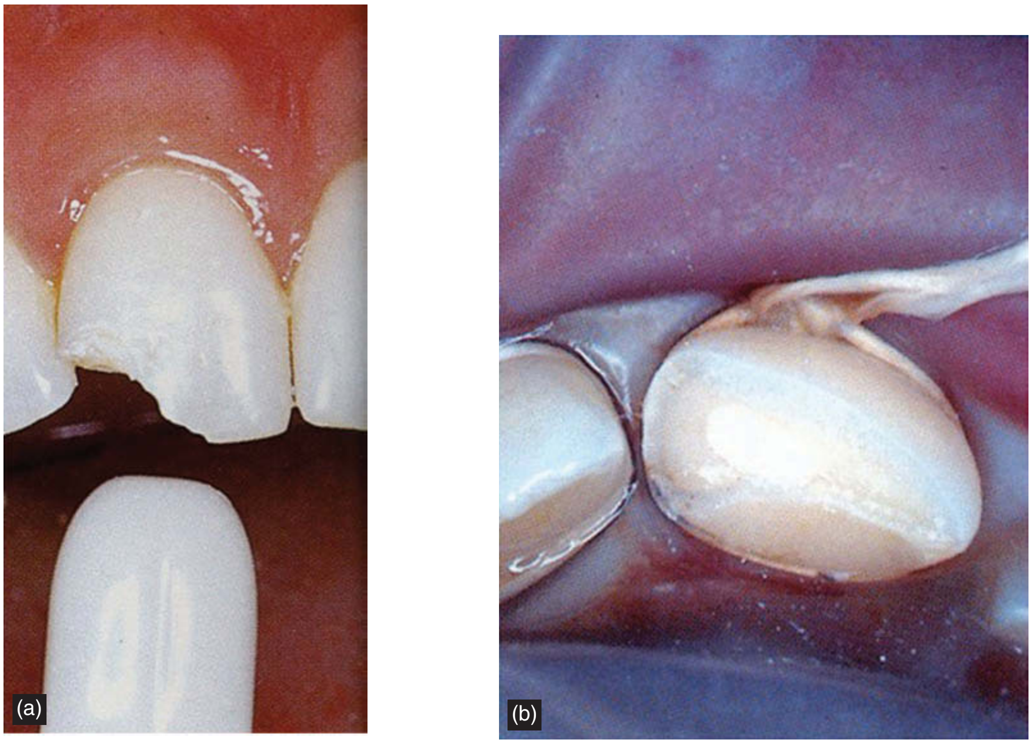

Figure 20.4
(a) and (b). Uncomplicated crown fractures through the dentin. Note the yellowish tint of the dentin.

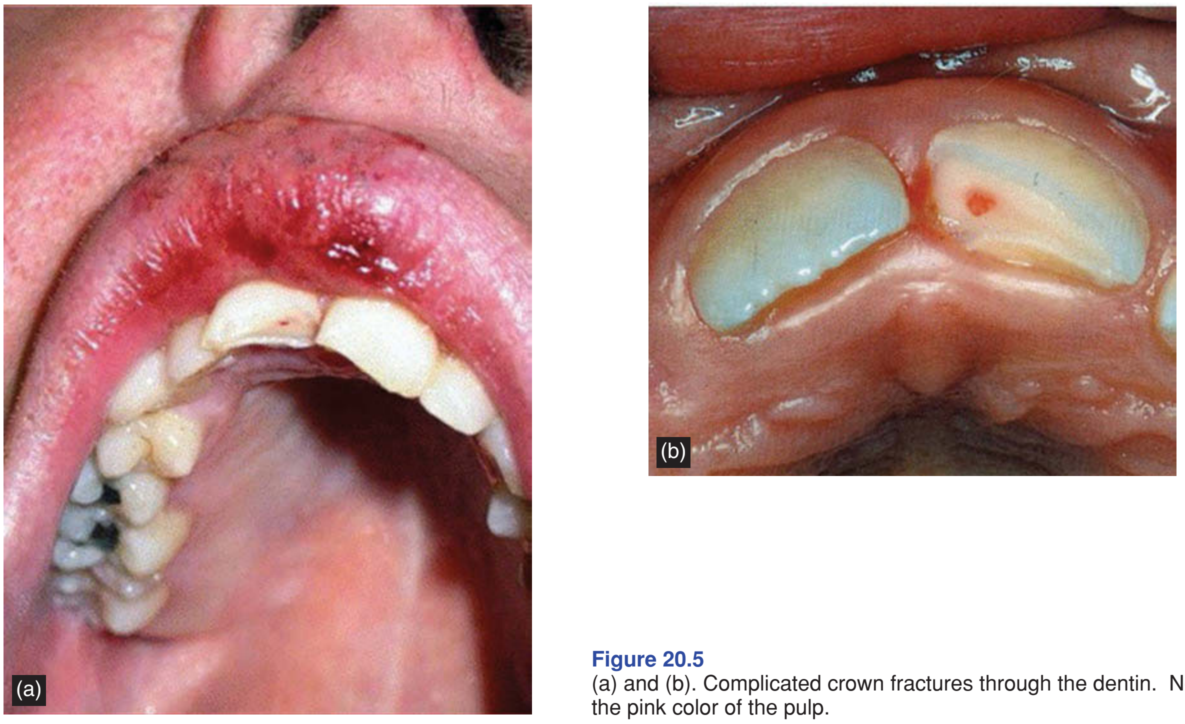

Figure 20.5
(a) and (b). Complicated crown fractures through the dentin. Note the pink color of the pulp.

Alveolar bone fractures

Trauma to the anterior teeth may result in fractures of the alveolus, the tooth-bearing portion of the maxilla or mandible. Alveolar ridge fractures often occur in multi-tooth segments. These may vary in the number of teeth involved, the amount of displacement, and the amount of mobility of the affected segment. This diagnosis is often obvious, as the examination reveals a misaligned and mobile section of teeth. Dental bite-wing X-rays confirm the diagnosis, and Panorex or facial films may show the fracture line apical to the roots of the involved teeth (although these films are often normal or inconclusive).

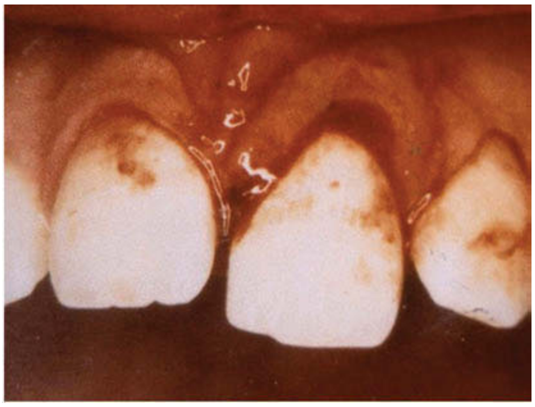

Figure 20.7
Extrusive luxation.

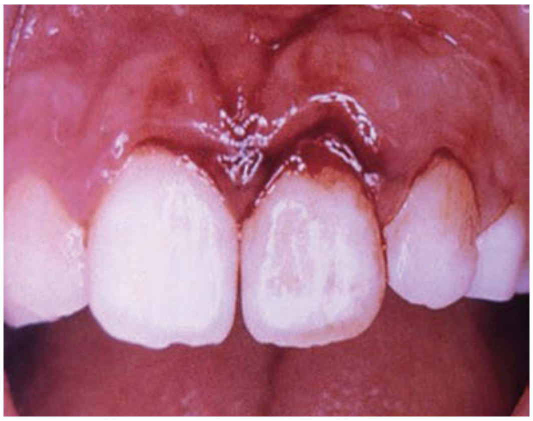

Figure 20.6
Tooth subluxation.

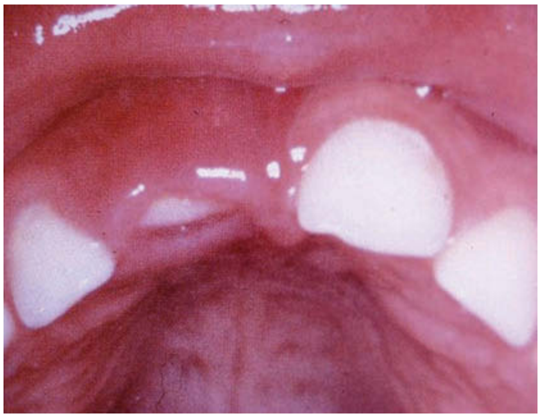

Figure 20.8
Intrusive luxation.

Hemorrhage

Bleeding from the oral cavity is commonly associated with dental procedures and may present in a delayed fashion. Spontaneous bleeding of the oral cavity or gingiva, not associated with dental manipulation or trauma, suggests advanced periodontal disease or a systemic process. Gingival bleeding after scaling or other minor routine dental procedures is usually controlled with direct pressure and saline/hydrogen peroxide rinses. Persistent bleeding from the gingival area despite pressure and rinses should raise suspicion for a bleeding abnormality (most commonly due to medications). Much more concerning is hemorrhage following a molar extraction. These patients typically present after several futile attempts to stop the bleeding at home.

Alveolar osteitis (dry socket)

Alveolar osteitis, a localized osteomyelitis, occurs when the alveolar bone becomes inflamed. This occurs when the clot, normally present after dental extraction, becomes dislodged or dissolves, 2–4 days later. The examination is usually unremarkable with the exception of the missing clot (which may not be obvious). Only 2–5% of patients develop a dry socket; however, this number increases with traumatic extractions or impacted third molars. Dry socket pain can be severe and requires definitive treatment.

Dental infections

Infections originating in the mouth run the spectrum from minor to life-threatening deep space infections that require airway management and operative intervention. Dental infections seen in the ED are most commonly secondary to pulp infection/inflammation or periodontal disease. Diseases of the periodontium are usually chronic. Over time, they can progress to form periodontal abscesses in which emergency treatment is required.

Pulpitis/periapical abscess

Diseases of the pulp may be secondary to trauma or instrumentation, but the most common cause is bacterial invasion after carious destruction of the enamel. As enamel is destroyed, caries may progress rapidly through the dentin and into the pulp chamber, causing an inflammatory reaction known as *pulpitis.* If the bacterial erosion is large enough to allow adequate drainage, the patient may remain asymptomatic for long periods of time. When drainage is obstructed, the process progresses toward the pulp and the periapical space, causing exquisite tenderness. A periapical abscess will follow the path of least resistance, which may be through the alveolar bone and gingiva into the mouth, or into the deep structures of the neck.

If the infection progresses apically through the alveolar bone and into the soft tissues, it then becomes a periodontal abscess. Incision and drainage is necessary once this occurs. These abscesses usually are seen at the gingival margin or on the attached gingiva.

Acute necrotizing ulcerative gingivitis

Periodontal disease is an infection of the gingiva, the periodontal ligament, or the alveolar bone, key components of the tooth's attachment apparatus. Periodontal disease, unlike pulpal disease, is often asymptomatic unless accompanied by abscess or ulceration.

Gingivitis is inflammation of the gingiva caused by bacteria. In advanced disease, the gingiva becomes inflamed, red and painful, and bleeds easily. Acute infectious gingivitis associated with ulcers and grayish pseudomembranes is termed *acute necrotizing ulcerative gingivitis* (ANUG). In ANUG, the inflamed and erythematous gingiva is friable and necrotic, and represents a destructive process of the periodontium. The patient often complains of pain, fever, generalized malaise and a bitter, metallic taste. Foul breath, regional lymphadenopathy and poor dental hygiene are present.

Periodontal abscess

When organisms become trapped in the periodontal pocket, an abscess may form. This purulent collection usually drains through the gingival sulcus. However, it can spread to the supporting tissues, alveolar bone and the periodontal ligament. This is known as *periodontitis*.

Pericoronitis usually occurs when the wisdom teeth erupt and the overlying gingiva becomes traumatized and inflamed. The gingiva can occasionally become infected. In rare cases, a localized infection can spread to deeper tissues (e.g., the pterygomandibular or submasseteric spaces). Patients with disseminated pericoronal infection present with trismus secondary to irritation of the masseter and pterygoid muscles.

Deep space infections of the head and neck

Odontogenic infections can spread to various potential spaces of the head and neck (Figure 20.9). Presenting signs and symptoms vary, but typically consist of pain, swelling, difficulty with speech or swallowing, trismus, fever and chills. Redness or erythema may be noted on the overlying skin. Certain teeth allow spread of infection to particular deep spaces of the head and neck more easily than others. Rapid spread of these infections make localizing the exact space responsible difficult. Potential spaces involved in deep space infection include the buccal, temporal, submasseteric, sublingual, submandibular, or parapharyngeal.

Maxillary extension of a periapical abscess can further spread to the infraorbital space, and possibly the cavernous sinus through the ophthalmic veins (resulting in cavernous sinus thrombosis). Cavernous sinus involvement from an odontogenic infection is usually associated with periorbital cellulitis, meningeal signs, and (possibly) a change in mental status.

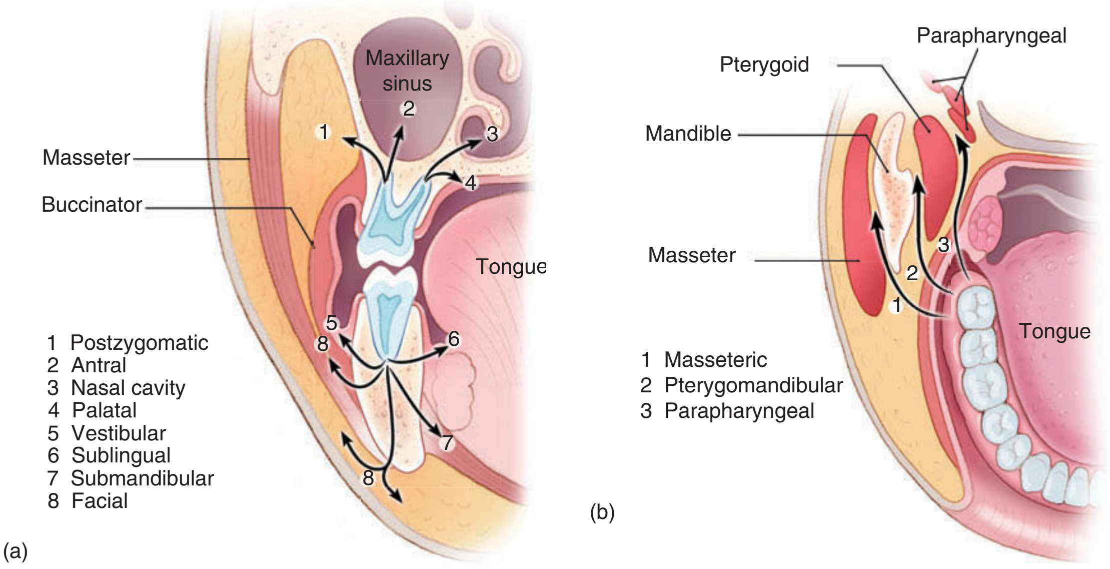

Figure 20.9
(a) and (b). Routes of odontogenic infections into the potential spaces of the face and neck © Chris Gralapp.

Infections of the anterior mandibular teeth often spread to the buccinator or sublingual space; those of the mandibular molars spread to the submandibular space, which connects to the sublingual space.

When bilateral involvement of the sublingual space occurs, a life-threatening condition known as *Ludwig's angina* results. Most cases of Ludwig's angina are secondary to dental infection. Specific attention must be paid to the floor of the mouth, the tongue and the neck during physical examination. Continuous airway assessment is paramount. As the infection progresses, the submandibular, submental and sublingual spaces become edematous, and elevation of the tongue and soft tissues of the mouth may occur. The soft tissues of the posterior pharynx can also become involved. The hyoid and suprahyoid regions of the neck become tense and indurated, and landmarks may become obscured. Such circumstances may result in airway compromise and complicated airway management.

Lemierre's syndrome is thrombophlebitis of the internal jugular vein with secondary spread of infection to the lungs, resulting from an initial infection of the oropharynx or teeth. This anaerobic infection is due to *Fusobacterium* species. It can occur in any patient, but is most common in those with poor dentition, poor nutrition and weakened immune systems.

Diagnostic testing

Laboratory studies

Routine blood work is not useful for most patients presenting with dental complaints, and should be considered on an individual basis. Bleeding times and coagulation profiles are unnecessary in routine cases of postextraction or traumatic intraoral bleeding, but should be considered if the patient is anticoagulated or has a history compatible with a bleeding disorder.

Radiologic studies

The routine evaluation and treatment of most dental emergencies does not require radiographic studies. Unlike definitive treatment in the dentist or oral surgeon's office, treatment of tooth or alveolar ridge fractures in the ED is usually not changed by information gained from X-rays. Radiographs can be helpful, however, if a tooth fragment is missing and thought to be lodged in the lip or buccal mucosa, or aspirated. Likewise, intruded teeth are not always apparent, and X-rays can help distinguish between an intruded and an avulsed tooth.

A Panorex (panoramic radiograph) view combined with a Townes view are probably the most useful and cost-effective X-rays to obtain when evaluating mandibular trauma in the ED. The Panorex of the mandible shows the mandible in its entirety and demonstrates fractures in all regions, including the symphysis. However, it can miss overriding anterior fractures; for such scenarios, an occlusal view or computed tomography (CT) is indicated. The Townes view allows slightly better visualization of the condyles and should be obtained if the condylar regions are not adequately visualized.

Coronal CT is more definitive and is often used in preoperative evaluation, but is usually unnecessary for diagnostic purposes in the ED. CT should be obtained if multiple facial fractures are suspected or if clinical suspicion for mandibular trauma is high despite an equivocal exam. If the patient is immobilized, in a cervical collar, or unable to sit still in a Panorex machine, plain mandibular films or CT should be performed. Mandibular plain films do not visualize the symphysis well; occlusal films may be required to visualize this region of the mandible.

Imaging for periapical abscesses is not clinically useful in the ED. Bite-wing radiographs performed in the dental office are much more likely to reveal periapical pathology than radiographs taken in the ED.

General treatment principles

Dental trauma

Fractures

Several general principles apply to the evaluation and management of dental trauma:

1. Identify all fracture fragments and mobile teeth, and note if a mandible fracture is open or closed. Radiographs should be taken if there is intrusion of fragments into the mucosa or alveolar bone. Obtain a chest radiograph if a patient with a missing tooth has pulmonary complaints (i.e., cough or shortness of breath) after the injury. Patients who present with a tooth aspiration may not recall coughing because of intoxication or other trauma.
2. The dentition is more easily manipulated following local anesthesia. Tooth infiltration and dental block anesthesia should be part of the emergency physician's skill set. Narcotic and non-narcotic alternatives usually do not provide enough pain relief to perform most dental manipulations. If the procedure is simple (such as applying glue to a lost cap or filling), a dental block is unnecessary.
3. Administer tetanus vaccination if indicated.

Management of fractured teeth in the ED depends on (1) the extent of fracture with regard to the pulp, (2) the degree of development of the apex of the tooth, and (3) the patient's age.

Uncomplicated crown fractures through the enamel only
Immediate treatment is not necessary but may consist of smoothing the sharp edge of the tooth with an emery board or rotary disk sander (Figure 20.10). It is important to reassure the patient that a dentist can restore the tooth to its normal appearance using composite resins and bonding materials. Follow-up is important as pulp necrosis and color change can occur (0–3%).

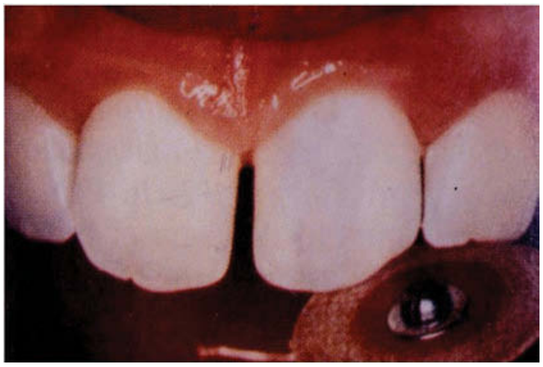

Figure 20.10
Smoothing enamel fracture with rotary disc sander.

Uncomplicated fractures through the enamel and dentin
The goal of treating dentin fractures is two-fold: cover the exposed dentin to prevent secondary contamination or infection and provide pain relief. Once a tooth is covered in the ED, a dentist can later rebuild it with modern composites. A dental block performed prior to any tooth manipulation allows easier application of the dressing. Dressings that may be applied to the surface of the tooth include calcium hydroxide and light-cured composites. The ease of applying calcium hydroxide paste and the fact that it can be used by itself make it attractive for use in the ED (Figure 20.11). Light-cured composites are more durable, but are more time-consuming and expensive than calcium hydroxide. Skin adhesives and bone wax are used in some EDs. They are not recommended, however, as bone wax is relatively porous, and skin adhesives are not approved for intraoral use. Many patients sustaining a fracture through the dentin will eventually require a root canal or other definitive endodontic treatment. The timely application of an appropriate dressing in the ED may prevent contamination of the pulp and make root canal unnecessary. As with any trauma to the anterior teeth, the patient should be told that disruption of the neurovascular supply is possible, and long-term complications such as pulp necrosis, discoloration and root resorption may occur.

Complicated fractures of the crown involving the pulp
Immediate management includes referral to a dentist, oral surgeon, or endodontist. These patients often require a *pulpectomy* (complete removal of the pulp) or, in the case of primary teeth, a *pulpotomy* (partial removal of the pulp) as definitive treatment. The longer the pulp is exposed, the greater the chance of contamination and ensuing abscess formation or pulp necrosis. If a dentist cannot see the patient immediately, the emergency physician should attempt to relieve the pain and cover the exposed pulp. Supraperiosteal infiltration should be performed if significant pain is present. Subsequently, the tooth should be covered with one of the dressings described above. After the covering is applied, the patient is instructed to consume only liquids and see the dentist as soon as possible. Not more than 24 hours should lapse before definitive treatment is initiated.

There are currently no definitive studies addressing whether antibiotics should be prescribed for fractured teeth seen in the ED. Patients who present to the dentist and then undergo definitive treatment do not routinely receive antibiotic prophylaxis. However, the treatment

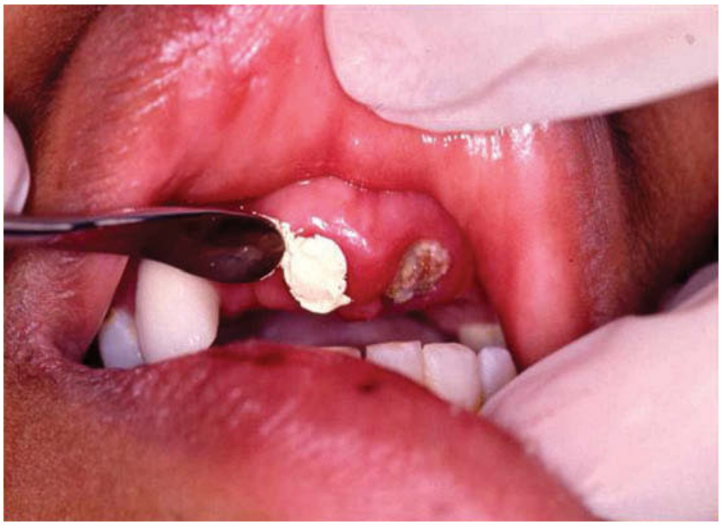

Figure 20.11
Application of calcium hydroxide paste to fracture site.

of dental fractures in the ED is often challenging because the patient's underlying dentoalveolar health is usually unknown and rapid dental follow-up cannot be guaranteed. Delayed fracture care and poor gingival health increase the risk of pulp necrosis and, potentially, periapical abscess. Therefore, antibiotic prophylaxis should be considered in the ED if prompt dental evaluation is unlikely.

Subluxation, luxation, intrusion and avulsion

Teeth that are minimally mobile and are not displaced generally do well with conservative treatment only. The tooth will tighten up in the socket if not re-traumatized. Patients should be instructed to eat a soft diet for 1–2 weeks and follow up with their dentist as soon as possible. Grossly mobile teeth require some form of stabilization as soon as possible. In certain patients with poor gingival health, luxated teeth may not be salvageable due to disease of the attachment apparatus. Fixation is best performed by a dental specialist using enamel bonding materials or wire ligation.

Temporary splinting techniques suitable for use by emergency physicians include periodontal paste and self-cure composite. A commercially available form of periodontal paste known as Coe-Pak consists of a base and a catalyst that when mixed together form a moderately sticky clay-like dressing that becomes firm after application (Figure 20.12). Self-cure composite is another splinting option (Figure 20.13). Both periodontal paste

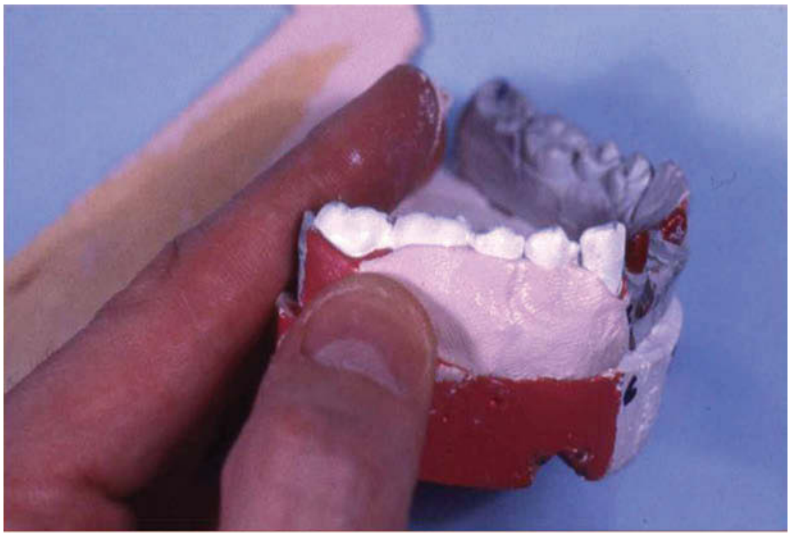

Figure 20.12
Periodontal paste for splinting loose teeth.

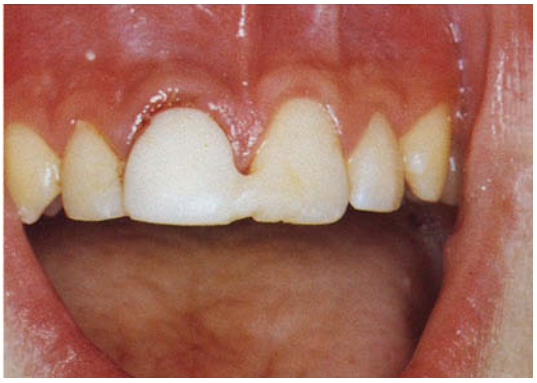

Figure 20.13
Self-cure composite for splinting loose teeth.

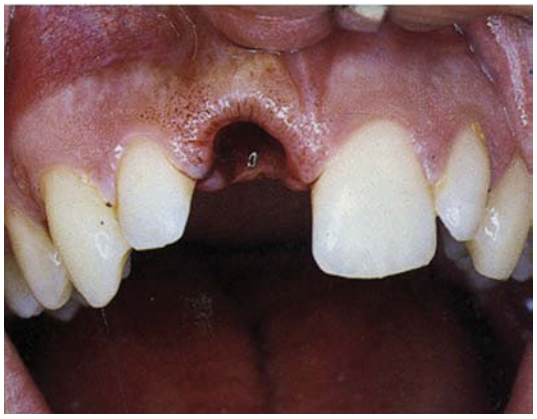

Figure 20.14
Avulsed tooth.

and self-cure composites are easy to remove during formal restoration by the dentist.

Teeth that are luxated in either the horizontal or axial planes or are slightly extruded can also be splinted using the above techniques. The teeth do not need to be in perfect alignment prior to discharge from the ED; final adjustments can be made in the dentist's office.

Intrusion

Intruded teeth are often immobile and therefore do not require stabilization in the ED; however, they often require delayed endodontic treatment because of pulp necrosis. Always consider the possibility of an intruded tooth when there is an abnormal space in the dentition. Undiagnosed intruded teeth can cause infection and craniofacial abnormalities. Radiographs should be obtained if there is uncertainty regarding whether a tooth is intruded or avulsed. A dental specialist should manage intruded teeth, with referral within 24 hours. Permanent teeth often require repositioning and immobilization, but primary teeth are usually given a trial period to erupt on their own before any intervention.

Avulsion

Avulsed teeth are true dental emergencies. The first question to ask is "Where is the tooth?" Missing teeth may be intruded, aspirated, fractured, swallowed, or embedded in the oral mucosa (Figure 20.14). Radiography including Panorex, or facial or chest films may be necessary to find fractured teeth fragments or an avulsed tooth. ED management is based the patient's age, time elapsed since avulsion, presence of other maxillofacial trauma (such as alveolar ridge fractures), and the overall health of the periodontium. Primary teeth are not replaced because they can fuse to alveolar bone, potentially cause craniofacial abnormalities or infection, and may prevent normal eruption of permanent teeth. Parents should be reassured that prosthetic replacements can be worn until the permanent teeth erupt, if desired.

Time is the essential consideration when deciding whether to replace an avulsed tooth. In general, the longer the tooth is out of the socket, the greater the incidence of periodontal ligament necrosis and subsequent reimplantation failure. Periodontal ligament cells lining the root of the avulsed tooth generally begin to die within 20 minutes if they are not placed in appropriate transport media. Milk and commercial preservatives (Save-A-Tooth, EMT Toothsaver) preserve the periodontal ligament for at least 4 and 12 hours, respectively; however, reimplantation should take place at the earliest opportunity (Figure 20.15). The critical factor is to get the tooth into some sort of transport media because even 5–10 minutes outside the oral cavity can lead to desiccation and death of the periodontal ligament cells. Saline should be used at the scene if nothing else is available. Saliva and water are less desirable alternatives, although saliva is preferable to water. The tooth should not be scrubbed or polished, which removes cells important for reattachment.

The best scenario is having the patient or prehospital providers attempt to reimplant the tooth at the scene, provided this doesn't pose a risk of aspiration. If the patient or medics are reluctant to do this, or there are conditions preventing reimplantation in the field, it will need to be done in the ED using the following guidelines:

Save·A·Tooth
THE EMERGENCY TOOTH PRESERVING SYSTEM
PATIENT NAME HERE
INSTRUCTIONS

Figure 20.15
Save-A-Tooth toothsaver.

1. Store the tooth in an appropriate media if reimplantation is delayed for any reason.
2. A supraperiosteal infiltration should be performed prior to the manipulation or replacement of any tooth. This makes the procedure more comfortable for the patient. Regional blocks also are acceptable and are especially useful if more than one tooth is involved.
3. Check the oral cavity for trauma; if an alveolar ridge fracture is present or the socket is severely damaged, the tooth should not be reimplanted.
4. If available, use a Frazier tip suction catheter to gently remove any accumulated clot from the socket; overly aggressive suctioning can damage the periodontal ligament fibers lining the socket. Next, gently irrigate the socket to remove any remaining clot. Reimplantation and realignment is difficult if the clot is not entirely removed. Handle the tooth by the crown. Debris on the tooth should be gently rinsed, *not scrubbed*, with saline. It is better to reimplant the tooth with a small amount of debris than to wipe off the periodontal ligament. Implant the tooth using firm but gentle pressure.
5. The tooth will require splinting (see above) after reimplantation if it is still loose. Teeth that remain mobile after reimplantation are less likely to develop firm attachment of the periodontal ligament.
6. Tetanus should be updated as indicated, and the patient should be instructed to take a soft diet.
7. Antibiotics are controversial in the management of fractured and avulsed teeth. Although the American Association of Endodontics (AAE) does not recommend the routine use of antibiotics for fractures or avulsions, other authors recommend prophylactic antibiotics that cover oral flora (penicillin or clindamycin) to decrease the inflammatory resorption of the root. It is probably prudent to use antibiotics if the root is heavily soiled; otherwise, treatment should be tailored to the individual patient and discussed with the consultant.

Prognosis depends on many factors, the most critical being time to reimplantation. In addition, the patient's age, the root's stage of development (younger is better), and the overall health of the gingiva are very important.

The emergency physician's goal in any tooth avulsion or fracture is to "save" the native tooth if possible. A reimplanted tooth usually loses the majority of its neurovascular supply and undergoes pulp necrosis. If the periodontal ligament remains intact, however, there is a much greater likelihood that the tooth will remain functional. It is important to remind the patient that some root resorption will occur after reimplantation, and there is always the chance of tooth loss despite the best efforts.

Alveolar bone fractures

Treatment of alveolar bone fractures involves rigid splinting of the affected segment, which should be done urgently (within 24 hours) by an oral surgeon or dentist. The urgency of immobilization depends on the mobility of the teeth, as well as the extent and displacement of the involved segment. A large and mobile fragment presents an aspiration risk and should therefore be fixed immediately. An open fracture requires immediate attention, including antibiotics. A stable, small segment of alveolar bone fracture could be repaired in 48–72 hours.

Hemorrhage

There are a number of options available in the ED to control bleeding:

- *Direct pressure*. Patients have probably tried this at home, but several simple techniques make this more effective. After providing local anesthesia, any excessive clot that has built up around the oozing site should be removed with suction. Then, the socket should be gently irrigated; adherent clot can be left intact. Next, insert dental roll gauze (dental tampon) over the bleeding site and cover with 2 × 2 gauze. Dental roll gauze has the advantage of fitting more precisely between the teeth, thereby affording more pressure. It helps to first moisten the gauze with a topical vasoconstrictor such as epinephrine. Instruct the patient to bite down and hold for 15 minutes.
- If bleeding persists after 15 minutes, infiltrate the bleeding area and gingiva surrounding the socket with lidocaine and epinephrine (1:100,000) until blanching occurs. Reapply the gauze over the site and instruct the patient to bite down for another 15 minutes. The injection provides vasoconstriction and anesthesia so that adequate pressure can be generated during biting.
- If bleeding persists, insert coagulating agents (e.g., Gelfoam, Surgicel, Avitene, Instat) into the socket and then loosely close the gingiva surrounding the socket with silk suture. Instruct the patient to bite down on gauze placed over the sutures.
- The hemostatic bandages also work well for dental bleeding; some are made to fit directly into the postextraction socket (Figure 20.16). Hemostatic bandages (e.g., Hemcon, QuikClot and QwickAid) utilize the electrical charge on the RBC to bind to the oppositely charged bandage.
- Electrocautery or thermal cautery units utilize battery power and do not require the patient to be grounded. The site should be anesthetized prior to cauterization.
- If the above measures are not successful in controlling bleeding, consult a specialist and consider using fresh-frozen plasma or platelets if coagulopathy is present.

Patients whose bleeding has been controlled may be discharged, and are instructed not to take anything by mouth for 4 hours, then only cold liquids and soft foods. Silk sutures require removal in 7 days. Prompt dental referral should be made.

Alveolar osteitis (dry socket)

A patient presenting with pain several days after an extraction and a relatively normal examination is likely to

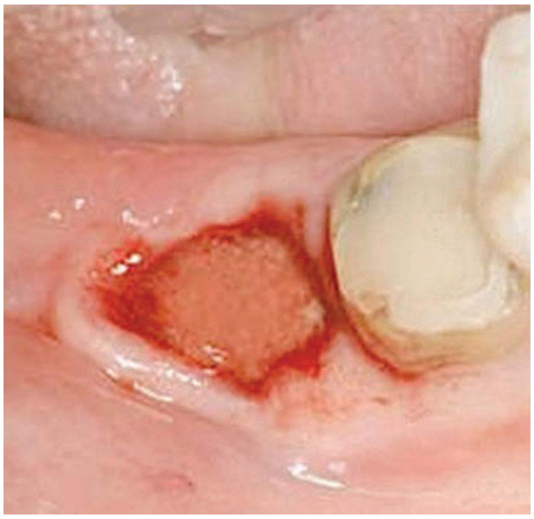

Figure 20.16
HemCon hemostatic gauze in a socket.

have alveolar osteitis. Although the pain is rarely relieved with traditional pain medications, a dental block provides immediate relief. The alveolar osteitis can be adequately treated after the pain is relieved. Irrigate the socket and gently suction any accumulated debris with a Frazier tip suction catheter. Next, the socket should be packed with a slurry of Gelfoam and eugenol, which prevents the recurrence of pain and allows healing. Gelfoam acts as a matrix to hold the eugenol in place. The product Dry Socket Paste is a thick paste containing eugenol, which can be applied by itself or mixed with Gelfoam to form a thicker slurry. It often stays in place longer than gauze and doesn't dry out. Other commercial products from dental supply companies are also available for treatment of dry sockets. Antibiotics are usually not necessary.

Dental infections

Pulpitis/periapical abscess

In the ED, differentiation of periapical abscess from pulpitis is very difficult because dental bite-wing X-rays are usually not available. Therefore, in the absence of recent trauma or instrumentation, some physicians initiate antibiotics therapy if the patient complains of odontalgia and has percussion tenderness on exam. Routine antibiotics for dental pain caused by pulpitis, instrumentation, or localized abscess are not recommended by dental societies. A recent study confirms that antibiotics are unnecessary for undifferentiated dental pain; however, these recommendations assume dental follow-up is available in 5–10 days. If follow-up is not assured, many emergency physician experts recommend consideration of antibiotics for undifferentiated dental pain. Antibiotics should be given for odontogenic infections that have spread beyond the immediate periapical area or have associated systemic signs, such as fever, swelling, or trismus. Supraperiosteal infiltration with bupivacaine (dental block) should be performed in most cases, as this not only provides immediate and long-acting pain relief, but also decreases the requirement for narcotic analgesia, even after the anesthetic has worn off. Abscesses of dental origin that do not extend into deep spaces, have well-defined boundaries and are easily accessible should be drained in the ED.

Acute necrotizing ulcerative gingivitis

Treatment is based on improving the mouth's environment with chlorhexidine and saline rinses, antibiotics directed at mixed anaerobes and spirochetes, and dental follow-up in 24–48 hours. Follow-up is mandatory for cases of ANUG, as the infection can quickly destroy the soft tissue and periosteum of the mandible. Systemic penicillin, clindamycin, tetracycline or doxycycline is the preferred antibiotic.

Periodontal abscesses

Periodontal abscesses that do not adequately drain through the gingival sulcus should be drained in the ED and treated with antibiotics. Penicillin is the initial drug of choice for odontogenic infections. Clindamycin may be used if a patient is allergic to penicillin. Saline rinses promote drainage; chlorhexidine rinses may be substituted for saline for more severe cases.

If pericoronal infection is localized, saline rinses and oral antibiotics are recommended, with dental follow-up in 24–48 hours.

Deep space infections of the head and neck

The management of complicated head and neck infections focuses primarily on airway management, surgical debridement and antibiotics. CT is the imaging modality of choice for deep space infections of the head and neck, and should be utilized to localize an abscess and delineate the boundaries of cellulitis that cannot be determined precisely by physical examination. The emergency physician should administer IV antibiotics and obtain otolaryngology or oral-maxillofacial (OMF) surgical consultation early in the evaluation and treatment of these patients. Airway intervention should be performed early if there is any suggestion of compromise.

The bacteria typically isolated from deep space infections of the head and neck consist of mixed flora, with anaerobes outnumbering aerobes. Although the mouth contains hundreds of bacterial isolates, most deep infections of dental origin are polymicrobial (five or six primary isolates, mostly anaerobic). *Peptostreptococcus*, *Bacteroides*, *Prevotella* and *Fusobacterium* species predominate, but aerobic alpha-hemolytic streptococci are present as well. Recommended antibiotic choices are penicillin G plus metronidazole or an extended-spectrum penicillin as a single agent (e.g., ampicillin/sulbactam, ticarcillin/clavulanate, or piperacillin/tazobactam). Clindamycin is a good alternative for patients allergic to penicillin, but it should be combined with a cephalosporin (e.g., cefotetan or cefoxitin) to cover resistant organisms. Remember that antibiotics are adjunctive therapy and not a substitute for definitive surgical therapy.

Special patients

Pediatric

It is important to remember that primary dentition differs from permanent dentition, especially with respect to intrusion and avulsion. Intruded baby teeth are usually allowed to erupt, whereas intruded permanent teeth usually require repositioning and fixation. Avulsed primary teeth should not be replaced. If there is a question of whether a tooth is permanent or not, replace the tooth or place the tooth in an appropriate storage media and obtain a Panorex. The presence of a tooth bud in the alveolar bone indicates a permanent tooth that has yet to erupt.

Elderly

The older population is much more likely to have prosthetic teeth, implants, or other dental appliances. This must be taken into consideration when determining what steps are necessary to best preserve the function of a traumatized or infected tooth.

Immune compromised

Patients with immune-compromising conditions are not only more susceptible to chronic periodontal disease, but are also at greater risk for ANUG and deep space infections. Any patient who presents to the ED with severe dentoalveolar disease should be questioned about HIV, diabetes, tuberculosis and malignancy, and their associated treatments. Appropriate testing and close follow-up should be arranged.

Disposition

Table 20.5 provides guidelines regarding disposition of patients presenting with dental complaints.

Pearls, pitfalls and myths

- Every ED should have basic dental materials to take care of simple fractures, avulsions, subluxations and bleeding. Having the right equipment is invaluable (Table 20.6).
- It is important to be familiar with dental anesthetic techniques, including dental blocks, as successful ED

Table 20.5 Disposition of patients presenting with dental complaints

Condition	Emergent dental consult	Next-day dental consultation	Admission
Fracture through enamel	No, follow up in 1–2 weeks is reasonable	No	No
Fracture through dentin	No	Yes, or in several days if covered appropriately	No
Fracture through pulp	Yes, can be seen in several days if covered appropriately		
Alveolar ridge fracture	Yes		Possibly, if alveolar ridge fracture unstable
Subluxation, luxation, intrusion	No	Yes, may be seen in several days if splinted adequately	No
Avulsion with replacement	No, unless adequate stabilization cannot be achieved	Yes	No
Avulsion without replacement	No	No, may be seen in several days	No
Alveolar osteitis (dry socket)	No	No, but follow-up should take place within several days	No
Hemorrhage	No, unless bleeding cannot be controlled	No, but follow-up should take place within several days	No
Pulpitis/odontalgia	No	No, but antibiotics should be considered if follow-up >10 days	No
Acute necrotizing ulcerative gingivitis (ANUG)	No	Yes, next-day follow-up essential	In select cases
Deep space infection	If airway compromise or immunocompromised	Yes	In select cases or Ludwig's angina

Table 20.6 Dental equipment needed in the ED

Packing gauze
Dental roll gauze
Calcium hydroxide paste *or* Self-Cure composite *or* Light-cure composite
Dry Socket Paste *or* eugenol
Topical anesthetic gel (20% benzocaine *or* 5% lidocaine)
Topical bactericidal intraoral solution (Ora-5)
Periodontal paste (Coe-Pak) *or* bupivacaine cartridges with epinephrine
EMT Toothsaver Preservation System *or* Save a Tooth *or* fresh milk
Zinc oxide/eugenol temporary cement (Temrex)
Ringed injection syringe
Stainless steel spatula and mixing pads
Tongue blades and cotton tipped applicators
Hemostatic gauze *or* disposable electrocautery *or* Gelfoam *or* ActCel *or* topical thrombin

treatment often relies on adequate pain relief. Dental anesthesia not only speeds care, but also increases patient satisfaction.

- Proper dental terminology is important for communicating with consultants. The Ellis classification of dental fractures, often cited in emergency medicine texts, is not typically part of dental specialist's terminology.
- Do not allow avulsed teeth to dry out because the appropriate storage media is not available. Milk is sufficient for temporary storage of avulsed teeth as is saliva.
- Primary teeth should not be replaced.
- Do not assume the tooth is lost; consider aspiration or intrusion as possible explanations.
- Alveolar ridge fractures that are grossly mobile should not be sent home without adequate stabilization, as these represent a serious aspiration risk.
- Failure to cover a fractured tooth may result in abscess or necrosis of the pulp. A simple calcium hydroxide cover will provide pain control as well preservation of the pulp, and may prevent the need for a root canal.

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21 Diabetes-related emergencies

Christopher RH Newton, MD and Stefanie A. Simmons, MD

Scope of the problem

Diabetes mellitus affects approximately 16.8 million people in the United States and over 180 million worldwide. Approximately 90% of cases are type 2 (non–insulin-dependent) diabetes mellitus. The remaining 10% of individuals have type 1 (insulin-dependent) diabetes. The increasing frequency of obesity has increased the prevalence of diabetes and lowered the age of onset of type 2 diabetes.

Diabetes is characterized by chronic hyperglycemia that often requires lifelong treatment. Untreated, chronic hyperglycemia leads to both micro- and macrovascular complications affecting virtually every organ system. As a result, diabetics frequently present to the emergency department (ED) with severe infections, myocardial infarction (MI), stroke, renal disease, lower extremity ischemia and skin ulcerations.

This chapter focuses on the diagnosis and management of acute metabolic derangements frequently encountered in diabetic patients. These consist of diabetic ketoacidosis (DKA), hyperosmolar hyperglycemic state (HHS) and hypoglycemia.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 21.1).

Diabetic ketoacidosis

DKA is a potentially life-threatening medical emergency. It occurs predominantly in type 1 diabetics and accounts for the initial presentation of glucose-related problems in about 25% of diabetics. The mortality rate for this condition is 2–4%. There has been a decline in the death rate from DKA, perhaps due to advances in diabetes care and the treatment of diabetic emergencies.

DKA is a syndrome characterized by hyperglycemia, ketonemia and metabolic acidosis caused by either relative or absolute insulin deficiency. Treatment consists of fluid and electrolyte replacement with continuous low-dose insulin infusion. Infection, MI, trauma,

Table 21.1 Diabetes-related emergencies red flags

History	**Concerning diagnosis**
Chest pain, shoulder pain, dyspnea	Myocardial infarction or ischemia
Fever	Infection or sepsis
No history of medication mismanagement or noncompliance	Organic precipitant to hyper- or hypoglycemic state
Patient's medications administered by someone else	Caregiver abuse or neglect
Oral hypoglycemic use	Prolonged, severe hypoglycemia
Dark vomit or stool	GI bleed
Melena or hematemesis	GI bleed
History of renal disease	Possible fluid overload (if fluid resuscitation required, possible need for dialysis or intubation)
History of depression/suicidality	Intentional insulin OD
Examination finding	**Concerning diagnosis**
Focal neurologic findings	CVA, hypoglycemia (can cause focal neurologic findings)
Hypoxia, diffuse rales	Fluid overload, ARDS (result of resuscitative measures)
Disheveled, poor hygiene, nonambulatory, poor vision	Inability to access or administer medications
Skin ulcer	Potential infectious etiology of DKA/HHS
Hypo- or hyperthermia	Infection, meningitis, sepsis
Kussmaul respirations, fruity breath	DKA
Focal abdominal pain, peritonitis	Surgical cause of DKA/HHS
Perineal erythema, inflammation, swelling	Fournier's gangrene/necrotizing fasciitis

ARDS: acute respiratory distress syndrome; CVA: cerebrovascular accident; DKA: diabetic ketoacidosis; GI: gastrointestinal; HHS: hyperosmolar hyperglycemic state; OD: overdose.

pregnancy, or stress may precipitate DKA. In many cases, a coexisting or precipitating disease process is not identified. Noncompliance with insulin therapy is recognized as a significant precipitant of DKA. Errors of insulin dosage may occasionally contribute.

Pathophysiology

The primary abnormality in DKA is an absolute or relative insulin deficiency. This leads to a rise in the counterregulatory hormones (i.e., catecholamines, glucagon, growth hormone and cortisol). Changes in these hormone levels produce three major effects:

1. Hyperglycemia resulting from decreased glucose utilization and increased hepatic gluconeogenesis
2. Increased lipolysis leading to ketone body formation
3. Increased metabolism of protein and reduction in protein synthesis

Hyperglycemia causes a profound osmotic diuresis resulting in progressive dehydration. Ketonemia and acidosis may lead to nausea and vomiting, exacerbating fluid and electrolyte losses.

History

Have you had increased thirst or urinary frequency?

Typically, patients describe the gradual onset of polyuria (increased urinary frequency) and polydipsia (increased thirst), with fatigue and progressive weight loss.

Have you had nausea, vomiting, or abdominal pain?

A combination of increased ketones and prostaglandin release is thought to contribute to nausea and vomiting. This can lead to a misdiagnosis of gastroenteritis in early DKA. Abdominal pain is frequently reported in DKA and has many causes, including gastric distension and ileus.

Have you been following your usual insulin schedule recently? Have you missed insulin doses or changed your diet? Have you changed your activity level recently?

This has been increasingly recognized as a precipitant of DKA, particularly in adolescents who often find it more difficult to comply with insulin regimens and eat appropriate meals at regularly scheduled times. Furthermore, changes in levels of activity and growth (which change caloric needs) must be taken into consideration, especially in adolescents.

Have you had a fever, painful urination, cough or shortness of breath? Have you had any chest pain or dark stool?

Infection, acute MI and gastrointestinal (GI) bleeding are all common precipitants of DKA. Systemic inquiry should be directed at uncovering these precipitants.

Physical examination

The vital signs are often abnormal in DKA. Tachycardia is most frequently observed. As fluid deficits increase, orthostatic hypotension is common. An elevated temperature is rarely caused by DKA itself and suggests the presence of infection. Hypothermia can also be associated with infection, and has an increased mortality rate in the setting of DKA. As the metabolic abnormalities progress, the patient becomes acidemic, leading to direct stimulation of the respiratory center in an attempt to compensate. This leads to an increased rate and depth of respiration, referred to as *Kussmaul respirations*. Systemic ketosis is often associated with an unusual fruity odor that may be detected by some clinicians on the breath of patients. Progressive dehydration may also lead to changes in mental status or coma. A careful abdominal examination is particularly important in patients presenting with abdominal pain , as peritoneal irritation from surgical conditions can precipitate DKA.

Evidence of infection should always be sought on examination of individuals with diabetes. Particular attention should be paid to the feet, genitourinary (GU) and rectal areas, especially in elderly, immunocompromised and obese diabetics.

Diagnostic testing

Once intravenous (IV) access has been established, a point-of-care (bedside) rapid blood glucose test should be performed. This is generally accurate if the serum blood glucose is <500 mg/dL. If the bedside glucose is >300mg/dL, fluid resuscitation should be initiated prior to obtaining formal laboratory results.

Required laboratory investigations include serum electrolytes, blood urea nitrogen, creatinine, glucose, calcium, magnesium, phosphate, and a complete blood count. Serum ketones should also be ordered, as the urine dipstick for ketones can be falsely negative. A blood gas should be obtained to document the pH. Recent literature has shown a significant correlation between venous and arterial blood gases; therefore, a venous sample is most often used for diagnostic purposes.

The serum potassium level is extremely important. Patients are usually severely potassium depleted, yet may have high serum levels on first sampling. This is caused by acidosis that enhances potassium release from cells in exchange for hydrogen ions in the serum, in an attempt to normalize the pH. Pseudohyponatremia is common and results from hyperglycemia. The sodium level can be corrected by adding 1.6 mEq of sodium for every 100 mg of glucose >100 mg/dL.

Patients with abdominal pain should have amylase or lipase and liver function tests ordered to consider pancreatitis or liver/gallbladder disease as possible etiologies for DKA. A septic work-up including blood and urine cultures and a chest X-ray should be considered, especially in febrile patients, although this may be modified depending on the patient's presentation. An electrocardiogram (ECG) is essential to look for evidence of hyperkalemia and to search for a possible precipitant of DKA, such as MI.

Guidelines published by the American Diabetic Association (ADA) outline three biochemical requirements for the diagnosis of DKA:

1. Glucose >250 mg/dL
2. Arterial pH <7.35, venous pH <7.30, or bicarbonate <15 mEq/L
3. Ketonemia or ketonuria

General treatment principles

Many EDs now have clinical guidelines and pathways available for the management of DKA. Treatment is usually initiated after obtaining a history suggestive of DKA and confirming an elevated bedside glucose.

1. Fluid replacement

Vigorous fluid resuscitation is mandatory and should be initiated prior to the return of laboratory results. Fluid restores intravascular volume and improves perfusion to vital organs. It also begins to lower the serum glucose. The initial fluid of choice is normal saline. Generally, in adults, the first 2 L should be given over the first 2 hours. An additional 2 L should then be given over the next 4 hours. After that, fluid can be titrated to the patient's clinical improvement and perceived hydration state. A number of studies suggest that hypotonic solution should be used after the initial resuscitation. A 5% dextrose-containing fluid should be started when the glucose falls <300 mg/dL. Excessive fluid replacement has been previously cited as a cause of important complications, such as cerebral edema and adult respiratory distress syndrome. However, patients in DKA are more commonly under-resuscitated than fluid overloaded. It is now recognized that fluid resuscitation probably has little or no role in the development of these complications.

2. Insulin therapy

Insulin therapy can be initiated once the serum electrolytes become available. It is critical to ensure that the patient is not hypokalemic, because insulin therapy will further drive potassium intracellularly, resulting in life-threatening dysrhythmias or respiratory paralysis in severely hypokalemic patients. Insulin therapy is administered with an initial 0.1 units/kg IV bolus, followed by a continuous infusion of short-acting insulin at 0.1 units/kg/hr (maximum initial dose 10 units/hr). The IV tubing should be flushed prior to initiating the infusion because insulin adheres to the tubing's walls, which can make the initial dosing erratic. A bedside glucose should be monitored hourly when an insulin infusion is used. It is important that the order for insulin spells out "Units" rather than "U," as this might prevent a dosing error and is required by Joint Commission.

The insulin infusion should be continued until serum ketones are cleared and the patient's anion gap (sodium minus chloride plus bicarbonate) has normalized (<12–14). Resolution of hyperglycemia usually occurs prior to this; therefore, dextrose must be provided to ensure normoglycemia.

3. Potassium replacement

Patients with DKA usually have profound depletion of total body potassium caused by insulin deficiency, acidosis, osmotic diuresis and vomiting. However, the initial potassium is usually normal or even high secondary to acidosis that drives hydrogen intracellularly and potassium extracellularly. Once fluid and insulin replacement are initiated, potassium is forced intracellularly and the serum level can drop dramatically. The decision to replace potassium is made only after the serum potassium result becomes available because of the potentially life-threatening consequences of giving potassium to a patient with hyperkalemia. Peaked T-waves, prolonged PR intervals and widened QRS complexes on the ECG provide early evidence of hyperkalemia prior to the serum electrolyte results becoming available. For this reason, an immediate ECG should be obtained on every patient presenting with DKA as part of the initial evaluation. A life-threatening or stat serum potassium may be run off arterial blood from an arterial blood gas.

Potassium replacement should be initiated in all patients unless the serum level is >5.5 mEq/L or the patient is anuric. Potassium is usually given as potassium chloride at an IV rate not faster than 5–15 mEq/hr. Patients whose initial potassium is <3.5 mEq/L need more aggressive replacement *prior* to initiation of the insulin infusion. The goal is to maintain potassium in the normal range of 4–5 mEq/L while avoiding life-threatening hypo- or hyperkalemia.

4. Additional therapies

Bicarbonate replacement is controversial and is not routinely recommended for the emergency management of patients with DKA. Despite a number of clinical trials assessing the efficacy of bicarbonate, none has shown improvement in clinical outcomes. Bicarbonate can be considered for patients with an initial pH of less than 7.0.

The replacement of phosphate remains controversial and is not routinely done in the ED. Despite theoretical benefits, there appears to be no clinical benefit from the routine administration of phosphate to patients with DKA.

5. Monitoring

Serum glucose and electrolytes should be checked at 0, 2 and 4 hours from presentation, and then every 4 hours during insulin infusion and potassium replacement. Bedside serum glucose should be checked at 0, 1 and 2 hours after presentation, and then every 1–2 hours while on the insulin drip. Many hospitals now use DKA flowsheets to keep track of vital signs and laboratory results, which makes patient management and documentation more efficient and reliable.

Special patients

Pediatric patients with DKA have a higher rate of developing cerebral edema than adults. The exact reasons for this are unknown. A recent study demonstrated that children with DKA who have low partial pressures of arterial carbon dioxide and high serum urea nitrogen concentrations at presentation, and are treated with bicarbonate are at increased risk for cerebral edema. Children should have fluid replaced judiciously and be admitted to a hospitalist or specialist in the management of pediatric DKA.

Pregnancy predisposes individuals to both diabetes (gestational) and DKA. Pregnant patients in DKA have higher rates of maternal and fetal complications. Maternal acidosis decreases fetal blood flow, and may cause fetal demise. As with children, pregnant patients in DKA should be cared for by specialists comfortable with the care of diabetes in pregnancy.

Patients with congestive heart failure can be particularly difficult to manage. They require fluid resuscitation yet can easily become fluid overloaded because of poor cardiac function. Although usually unnecessary, monitoring of central venous pressure or pulmonary artery wedge pressure should be considered for patients with a prior history of congestive heart failure.

Patients with renal failure and DKA require careful fluid and potassium replacement. Management often requires input from a nephrologist or critical care specialist.

Disposition

The vast majority of patients with DKA require admission to a setting where frequent monitoring of vital signs and serial blood draws can occur. Patients with altered mental status, hypotension, or severe acidosis should be admitted to the intensive care unit. Many hospitals have policies that dictate admission criteria for DKA patients on insulin drips.

Complications

Cerebral edema and adult respiratory distress syndrome (ARDS) are rare but life-threatening complications of DKA. Cerebral edema occurs primarily in pediatric patients. It manifests as progressive deterioration in mental status 6–10 hours after the initiation of therapy. There are no warning signs or clinical predictors of cerebral edema, which has a high mortality. Patients who develop cerebral edema should be treated aggressively with mannitol and dexamethasone in collaboration with a pediatric intensivist.

Dyspnea, hypoxemia and diffuse pulmonary edema on chest X-ray are classic findings of ARDS. Patients often require ventilatory support, either invasive or noninvasive. As with cerebral edema, mortality is high.

Iatrogenic complications include pulmonary edema from over-aggressive fluid resuscitation, hypoglycemia from inadequate glucose monitoring and failure to add glucose to the fluids when the serum glucose falls <300 mg/dL, and hypokalemia. Poor outcome may also result from under-resuscitation or electrolyte abnormalities. Strict nursing adherence to DKA management guidelines minimizes the risk of these complications.

Hyperosmolar hyperglycemic state

HHS or hyperosmolar hyperglycemic nonketotic syndrome (HHNS) is characterized by hyperglycemia, hyperosmolarity and dehydration. Unlike DKA, ketosis and acidosis are usually minimal or absent. HHS is most frequently observed in poorly controlled or undiagnosed type 2 diabetics. Alteration of consciousness is a common finding and may progress to coma, leading to the former name hyperosmolar nonketotic coma (HNKC). However, this term is confusing as the majority of these patients are not actually comatose. Mortality in HHS is much greater than in DKA, usually between 15% and 30%. This higher rate is likely related to both the underlying disease precipitants and the elderly population that it affects.

Pathophysiology

Insulin resistance leads to inadequate tissue utilization of glucose, resulting in hyperglycemia. Hepatic gluconeogenesis and glycogenolysis further elevate the serum glucose level. As the serum glucose increases, it creates an osmotic gradient that draws water out of the intracellular space and into the intravascular compartment. When the serum glucose level exceeds the kidneys' capacity to reabsorb it, glucose spills into the urine, creating glucosuria and an osmotic diuresis.

Patients may be able to keep up with the volume losses, especially since there is less acidosis causing less nausea and vomiting. However, many elderly patients in skilled nursing facilities do not have access to fluids or are unable to keep up with the excessive fluid losses. Therefore, they become progressively dehydrated. These fluid losses often exceed 20% of total body weight.

The absence of ketoacidosis in patients with HHS has a number of potential causes, including lower levels of counter-regulatory hormones, higher levels of insulin, and inhibition of lipolysis by the hyperosmolar state.

History

HHS is usually seen in elderly patients with a variety of nonspecific complaints, including weakness and fatigue. If able to answer questions, patients may complain of polyuria and polydipsia for days or weeks prior to seeking medical attention. Inquiries about symptoms consistent with precipitants of HHS, such as infection, MI, stroke, or GI bleeding, should be made.

Physical examination

Altered mental status and abnormal vital signs are the most frequently encountered findings in HHS. It is important to remember that elderly patients may have an underlying degree of baseline cognitive impairment, making it essential to obtain a detailed history from the family or caregiver about any change from that baseline. The degree of lethargy and coma exhibited correlates well with their serum osmolality. Patients in HHS usually exhibit evidence of volume depletion, such as poor skin turgor, dry mucus membranes and orthostatic hypotension. Evidence of cellulitis or melena should be sought during the physical examination.

Diagnostic testing

The initial diagnostic work-up for HHS is similar to that for DKA, with the addition of sending a serum osmolality to the laboratory. An ECG should be performed as early as possible. Precipitants of HHS should be considered when ordering other studies. Blood cultures, cardiac enzymes, chest X-ray, head computed tomography (CT) and lumbar puncture (LP) should be guided by the clinical presentation. Arterial blood gases are usually unnecessary unless there is a pulmonary component to the acid–base abnormality.

HHS is defined by:

1. Serum glucose >400 mg/dL
2. Calculated plasma osmolality >315 mOsm/L in the absence of ketosis

In practice, the serum glucose level is usually >600 mg/dL and the osmolality is >350 mOsm/L, with marked electrolyte abnormalities. Acidosis and ketones can be seen occasionally and are usually explained by the precipitant of the HHS.

General treatment principles

1. Fluid replacement

Similar to DKA, the initial resuscitation in HHS is aimed at restoring adequate tissue perfusion and decreasing serum glucose. The average fluid deficit in HHS is 8–12 L, often double the deficit encountered in DKA. Half of this deficit should be replaced IV over the first 12 hours, and the remainder over the next 24 hours. The actual rate of fluid administration is highly variable and depends on the estimated fluid deficit, the patient's weight, and the degree of renal and cardiac impairment. Isotonic saline (0.9% NS) is the most appropriate crystalloid for initial volume restoration. This can then be switched to half-normal saline (0.45% NS) once vital signs have normalized and there is adequate urine output.

2. Potassium replacement

All patients with HHS have deficits in total body potassium. An IV infusion of potassium at 10 mEq/hr should be initiated in all patients who are making urine and are not hyperkalemic. Higher rates of potassium replacement may be necessary if the patient is initially hypokalemic. Potassium levels should be monitored every hour until consistently in the normal range.

3. Insulin infusion

After adequate fluid replacement and determination of the serum potassium, regular insulin may be given as a continuous IV infusion at 0.1 units/kg/hr. The insulin infusion should be discontinued once the blood glucose is <250 mg/dL. At this time, 5% dextrose should be added to maintenance fluids to prevent hypoglycemia.

Special patients

HHS most commonly occurs in elderly diabetics who may have underlying cardiac or renal disease. As discussed in the DKA section, this makes therapy much more complicated and results in higher morbidity and mortality.

Disposition

Most patients with HHS require admission to an intensive care unit for frequent evaluation, monitoring of vital signs and serial blood tests.

Hypoglycemia

Although there is no universal definition, hypoglycemia is best defined as a low serum glucose (usually <50 mg/dL) with symptoms that resolve upon administration of glucose or carbohydrate. The glucose level at which patients become symptomatic is highly variable, as many patients report symptoms with normal serum glucose levels, whereas others remain asymptomatic at serum levels less than 50 mg/dL. Hypoglycemia is most commonly encountered in type 1 diabetics who have missed meals, increased their exercise or activities, or increased their dose of insulin. It occurs more frequently in young diabetics as a result of the increased emphasis on tight glycemic control. It is also encountered in diabetics taking oral hypoglycemic agents both during the course of normal therapy and as a result of an intentional overdose. Sepsis, alcohol intoxication, starvation and liver disease also may result in hypoglycemia. Infants, adolescents, and the elderly are at increased risk for hypoglycemia.

Hypoglycemia should be considered in any patient presenting to the ED with altered mental status or focal neurologic deficits. Hypoglycemia may mimic an acute stroke, and must be kept in the differential diagnosis of a stroke patient. Rapid diagnosis is essential, as a delay in the restoration of carbohydrate substrate can lead to seizure, permanent neurologic deficits, or death.

Pathophysiology

Glucose homeostasis involves the intake of food as well as the complex interactions between insulin, glucagon, and other counter-regulatory hormones. Following a meal, insulin is the major regulatory hormone enhancing glucose utilization for fuel and storage, while also inhibiting glucose production. In the fasting state, low insulin levels promote mobilization of stored fuel. Hepatic glycogen is broken down first, and is depleted in 24–48 hours in an individual with normal underlying nutritional status. With prolonged fasting, gluconeogenesis becomes the primary source of glucose, with potential breakdown of adipose tissue and protein.

Alcohol inhibits hepatic gluconeogenesis and causes problems when malnourished alcoholics use up already depleted glycogen stores. Sepsis also inhibits gluconeogenesis, which in turn can lead to hypoglycemia. The brain requires a continuous supply of glucose for normal function. When glucose levels fall, patients develop neurologic symptoms directly from a lack of glucose within the brain. They are likely to also develop adrenergic symptoms from increased levels of counter-regulatory hormones.

History and physical examination

Hypoglycemia is a great mimic and has a variety of clinical presentations that can fool even the most experienced provider. Adrenergic symptoms are most prominent when there is an abrupt drop in the blood glucose. These include anxiety, nervousness, irritability, nausea, vomiting, palpitations, tremors, diaphoresis and sweating. They are often referred to as the "classic" warning symptoms of hypoglycemia. Diabetics are usually able to recognize these symptoms and respond by ingesting glucose. For this reason, they are not commonly encountered in the ED. Adrenergic symptoms are less prominent or may be absent in some patients, especially those on beta-blockers. For these patients, symptoms related to decreased cerebral glucose predominate. They range from lethargy and confusion to combativeness and agitation. Hypoglycemia can also cause seizures, focal neurologic deficits and coma.

Diagnostic testing

The diagnosis of hypoglycemia can be made at the patient's bedside with capillary glucose testing, and confirmed in the laboratory with serum glucose testing. Rapid diagnosis of hypoglycemia is imperative so that treatment can be instituted in a timely fashion. Early diagnosis can also minimize costly work-ups for patients with altered mental status or focal neurologic deficits.

Further evaluation depends on the patient's clinical improvement and possible precipitants. The response to IV dextrose is usually rapid. However, hypoglycemia and altered mental status can be an initial presentation of sepsis. Therefore, patients should undergo additional work-up including blood cultures, LP, antibiotics, and admission if an appropriate clinical picture exists.

General treatment principles

Once the diagnosis of hypoglycemia is made or suspected, treatment should be initiated immediately. In adults, this consists of 1 g/kg of 50% dextrose IV (initially 1–2 ampules of D50). In children less than 8 years old, 2–4 mL/kg of 25% dextrose is administered; 1–2 mL/kg of 10% dextrose is used in neonates. These doses can be repeated if the patient or glucose level does not respond. An infusion of either D5W or D10W can then be started to maintain the glucose above 100 mg/dL if the patient is unable to consume glucose or complex carbohydrates.

In patients without IV access, 1 mg glucagon can be given intramuscularly or subcutaneously. It usually takes 5–20 minutes before clinical effects are seen. If the patient is awake and alert, he or she can drink something containing sugar, such as orange juice, and then have a meal of complex carbohydrates.

IV thiamine should be administered to alcoholic patients prior to dextrose due to the theoretical risk of precipitating Wernicke's encephalopathy. IV corticosteroids should be considered for hypoglycemia resistant to dextrose therapy.

Special patients

All patients on oral hypoglycemic agents (i.e., sulfonylureas) should be observed for an extended period (either in the hospital or an ED observation unit, depending on hospital custom), because these drugs have long biological half-lives and a propensity for causing prolonged and severe hypoglycemia. Octreotide in combination with dextrose has been recommended as first-line therapy in the treatment for sulfonylurea-induced hypoglycemia, especially when it is refractory to dextrose therapy alone.

Homeless and alcoholic patients may require a more prolonged period of observation. They may also benefit from being seen by a social services professional who may offer referrals to appropriate outpatient care settings, or provide psychologic and possibly financial support.

Geriatric patients appropriate for discharge may benefit from being given prefilled insulin syringes or scheduled with a home health aid to assist them with their medications. Social services are integral to the care of elderly diabetics, especially those with hypoglycemia.

Disposition

Patients with diabetes who are not on oral hypoglycemic medications can be discharged home following a short period of observation if they have an appropriate response to treatment and are able to eat without difficulty. They should be instructed to follow-up with their primary care physician, including a phone call that day or the next. Advice should be given regarding consumption of regular meals, including small snacks, and the warning symptoms of hypoglycemia. If they are on insulin, the dose can be reduced until they follow up with their physician who manages their diabetes. These individuals should be instructed to be extremely careful driving, operating machinery, and climbing or working at height at least until they are rechecked and their condition has stabilized.

Pearls, pitfalls and myths

- A thorough search for precipitants of DKA, HHS, or hypoglycemia (MI, stroke, infection, sepsis, or GI bleeding) should always be performed. It is particularly important to examine the urine, feet and perineal area of elderly diabetics for a source of infection. Drug and alcohol ingestion can alter metabolic rate, food intake and insulin use, so a careful social history is essential.

- A rapid bedside capillary glucose should be assessed in any patient who is confused or presents with a neurologic deficit (including coma, seizure, or focal neurologic deficit); this is especially true in ill patients or the extremes of age. If the point-of-care bedside glucose is low, glucose should be given immediately. For alcoholic patients, thiamine should be given prior to glucose administration.
- Insulin should not be administered in patients with DKA or HHS until the serum potassium level is known. The initial therapeutic intervention is fluid resuscitation with normal saline, not insulin.
- Always remember to replace potassium in patient with DKA and HHS *unless* the initial potassium level is >5.5 mEq/L or the patient is anuric.
- In patients with DKA, an insulin drip must be continued until the anion gap is normal. Adding dextrose to the IV fluids when the glucose level falls below 300 mg/dL will prevent iatrogenic hypoglycemia. Closely monitor the glucose and potassium levels of patient on insulin drips.
- Two main ketoacids produced in DKA are acetoacetate and beta-hydroxybutyrate. The nitroprusside test for urine ketones detects acetoacetate but not beta-hydroxybutyrate. Beta-hydroxybutyrate, identified by serum ketone testing, often predominates in early DKA; hence, false-negative results can occur if only the urine is tested for ketones.
- Excessive fluid replacement has been previously cited as a cause of complications in the management of DKA, although recent evidence refutes this. In fact, most patients with DKA are under-resuscitated.
- The capillary glucose should be rechecked regularly on patients presenting with hypoglycemia.
- All patients taking long-acting oral hypoglycemic agents and presenting with hypoglycemia should be observed for an extended periods. Intentional sulfonylurea overdose is particularly challenging to treat; octreotide and dextrose serve as initial treatments in these cases.

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22 Diarrhea

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Scope of the problem

There are roughly 375 million cases of acute diarrhea in the United Stated each year (1.4 episodes per person per year), accounting for an estimated 5% of emergency department (ED) visits during the fall and winter months. The direct and indirect cost of diarrheal illnesses in the United States approaches $6 billion annually (for medical care and lost productivity). Worldwide, diarrheal illnesses affect as many as 5 billion people a year, accounting for nearly 10 million deaths in developing countries. A 2009 report released by the United Nations Children's Fund and the World Health Organization indicated that diarrhea claims the lives of approximately 1.5 million children less than 5 years old annually. Etiologies range from benign conditions, such as viral gastroenteritis, to life-threatening invasive diarrheal illnesses. The most common causes of acute diarrhea are infectious agents, such as viruses, bacteria and parasites.

Anatomic essentials

Diarrhea can be defined as the rapid passage of excessively fluid stool, or stool that takes the form of the container rather than remaining in its natural form, or stool frequency greater than three times a day. The gastrointestinal (GI) tract resorbs over 9 L of fluid a day (majority by the small intestine), leaving approximately 100 mL/day excreted in stool. Alteration in this process may lead to diarrhea. This can occur from an increase in volume load presented to the GI tract (secretory diarrhea), diminished ability to resorb fluids by the bowel (osmotic diarrhea), inflammatory processes, or an increase in gut motility.

Toxigenic agents (e.g., *Vibrio cholerae* or *Escherichia coli*) result in a *secretory diarrhea* by increasing the amount of fluid secreted into bowel beyond the amount absorbed. *Osmotic diarrhea* occurs when unabsorbable or poorly absorbed molecules such as lactulose and/or laxatives challenge the small intestine. *Inflammatory diarrhea* occurs with inflammation of bowel mucosa, limiting its ability to resorb fluid. This can occur with numerous agents, including *Shigella* and *Giardia*. Diarrhea caused by increased gut motility can be seen in patients with irritable bowel syndrome or gut-altering surgery. The key differences between inflammatory and non-inflammatory diarrhea are summarized in Table 22.1.

Lastly, clinicians should be able to distinguish between gastroenteritis and dysentery. Gastroenteritis refers to patients who have both diarrhea and vomiting; dysentery refers to diarrhea containing blood and pus.

Table 22.1 Typical findings in inflammatory and non-inflammatory diarrheal illnesses

Inflammatory	Non-inflammatory
Often bloody	Infrequently bloody
Fever	Nausea and vomiting
Moderate to severe abdominal pain and tenderness	Mild abdominal pain
Frequent small volume stools	Watery, large volume stools

Adapted from Schertzer K, Garmel GM. Acute infectious diarrhea. In Chin RL (ed). *Emergency Management of Infectious Diseases*. Cambridge University Press, New York, 2008.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 22.2).

History

The exact etiology of diarrheal illnesses is rarely determined in the emergency department. However, a thorough history is essential to identifying important pathogens and non-infectious etiologies. Ensure privacy and empathy, as many patients are uncomfortable discussing diarrhea, and likely feel ill. Historical findings for common diarrheal pathogens can be found in Table 22.3.

How would you describe the diarrhea?

Loose, watery, or bloody stools suggest an invasive process or gastrointestinal bleeding. Abnormal rectal discharge, or greasy or foul-smelling stools suggest malabsorption or giardiasis.

How many episodes of diarrhea have you had?

The greater the number of daily stools, the greater the risk of dehydration or electrolyte abnormality.

What is the duration of your symptoms?

Distinguishing acute versus chronic diarrheal diseases gives insight into the etiology as well as comorbid diseases. Acute diarrheal illnesses are usually less than 2 weeks in duration; chronic diarrhea results in symptoms beyond this time frame. Patients with advanced

Table 22.2 Diarrhea red flags

History	Concerning diagnosis
Duration of symptoms >2 weeks	Immune compromise, pancreatitis, inflammatory bowel disease
Recent ingestion of fried rice, seafood, or egg-based products	Infection with *B. cereus*, Scombroid, or *Salmonella*, respectively
Recent travel to Mexico	Montezuma's revenge *(E. coli)*
Camping near fresh-water streams	Giardiasis
New medications and/or antibiotic use	Drug-induced diarrhea or *C. difficile* colitis
Fever and/or bloody diarrhea	Infection with an invasive organism – *E. coli, Shigella, Salmonella, Yersinia*, or *Campylobacter*
Children or the elderly	Severe dehydration
Examination finding	**Concerning diagnosis**
Fever, tachycardia, or hypotension	Invasive diarrhea causing severe dehydration, bacteremia, and/or sepsis
Altered mental status	*Salmonella typhi*, *Shigella* and *Campylobacter*
Dry mucous membranes, poor skin turgor	Acute dehydration
Bloody stools	Invasive diarrhea
Fecal impaction	Tumor or Hirschsprung's disease
Perianal fissures or fistulas	Inflammatory bowel disease

Table 22.3 Historical information relevant to diarrhea

Character of stools	Temporal characteristics	Exogenous factors	Associated symptoms	Medical history
Amount Consistency Color Odor Mucus Blood Pus	Acute Chronic Recurrent Frequency Duration	Diet Medications Travel Exposures to others with same symptoms Sexual habits	Fever Nausea Vomiting Abdominal pain Oral intake	GI disease HIV/AIDS Endocrine Diabetes Adrenal insufficiency Uremia

Adapted from Collier RE, Gough JE, Clement PA. In Marx J et al. (ed). *Emergency Medicine: Concepts and Clinical Practice*, 7th ed. Mosby, St. Louis, MO, 2010.

HIV/AIDS, pancreatitis, inflammatory bowel disease, or complex gastric or bowel surgery may suffer from chronic diarrhea requiring long-term therapy. Acute diarrheal illnesses usually require only a short course of symptomatic treatment, as they are commonly the result of self-limiting viral or foodborne etiologies.

What do you think caused your symptoms?

Most patients attribute their acute illness to something they ate or being around someone with similar symptoms. This is helpful if a history of ingesting fried rice (*Bacillus cereus*), seafood (Scombroid), or egg-based products (*Salmonella*) is obtained. Food poisoning should be considered when the patient's symptoms begin 1–6 hours after eating a high-risk meal.

Have you traveled recently?

Travel to foreign countries where water purification and food handling is not closely regulated may suggest bacterial or parasitic causes of diarrhea. The most notable of these disorders is "Montezuma's revenge" (traveler's diarrhea) caused by enterotoxigenic *E. coli*. Patients who give a history of camping and drinking water from lakes or streams may be suffering from giardiasis.

Are you taking any new medications?

Many classes of drugs can cause acute diarrheal illnesses. These include laxatives, cholinergic drugs and antacids. Alcohol may also cause diarrhea. Antibiotics are by far the most common cause of drug-induced diarrheal illness. Disruption of the native colonic bacterial flora can lead to overgrowth of other species, as occurs in *Clostridium difficile* colitis.

Associated symptoms

Most patients with diarrhea complain of nonspecific abdominal cramps, nausea and vomiting. Significant potassium loss may occur, which can cause weakness or muscle cramps. Patients with fever or bloody diarrhea may have a more serious invasive disease caused by *E. coli, Shigella, Salmonella, Yersinia*, or *Campylobacter*. In children and the elderly, severe dehydration, sepsis and death can occur without adequate therapy. Weight loss suggests prolonged disease, malabsorption, or carcinoma. Excessive flatulence may suggest malabsorptive diseases or parasitic infection.

Past medical

Patients with comorbid illnesses or taking certain medications may have chronic diarrhea requiring long term care. These include HIV/AIDS or other immune compromised states; chronic corticosteroids, chemotherapy or other immune-modulating agents; advanced diabetes; and, chronic alcoholism.

Social

Asking about sexual habits or HIV risk factors may provide a clue to the diagnosis of a diarrheal illness in an immune-compromised host. Work history may also be helpful, as this may determine infectivity and affect return to work status (e.g., food handlers).

Physical examination

The physical examination assists with the diagnosis as well as helps gauge the patient's clinical status. The most important initial clinical assessment in a patient complaining of diarrhea is *volume status*. As with any chief complaint, consideration of all organ systems is important in avoiding missed diagnoses.

General appearance

The general impression of whether a patient is sick or not is a critical piece of clinical information. In patients who appear toxic with fever, tachycardia, and/or hypotension, suspect invasive diseases causing bacteremia and sepsis. Patients with more benign diseases usually appear mildly uncomfortable or even relatively well.

Vital signs

The presence of tachycardia, hypotension, or both suggests dehydration. Although the importance of orthostatic hypotension is debated, it may be present in patients who are acutely dehydrated. Dehydration accompanied by fever should merit consideration of an invasive etiology.

Head, ears, eyes, nose, throat and neck

Dry mucous membranes, sunken fontanel in children, and sunken eyes all suggest severe volume depletion. The thyroid should be evaluated for a mass or venous hum, which may suggest hyperthyroidism.

Abdomen

Most patients with acute diarrhea have mild generalized tenderness and increased bowel sounds. Patients with inflammatory bowel disease will often have mild focal findings. In general, peritoneal signs should be absent; if present, the clinician should be alerted to a possible surgical condition, such as appendicitis.

Neurologic

Alteration in mental status may be related to volume loss or electrolyte abnormalities, and can be associated with specific infectious agents such as *Salmonella typhi*, *Shigella* and *Campylobacter*.

Skin/extremities

Poor skin turgor and poor capillary refill suggest dehydration.

Rectal

Digital rectal examination is not routinely necessary in patients with diarrheal illnesses. However, examination of the perirectal area as well as hemoccult testing of the stool may be helpful in certain circumstances. Extraintestinal manifestations of inflammatory bowel disease include perianal fissures or fistulae. Grossly bloody stool, pus, or mucus supports either an inflammatory, invasive, or ischemic process. Lastly, fecal impaction can result in liquid stool passing around the impaction ("overflow diarrhea") in elderly patients or those with Hirschsprung's disease.

Differential diagnosis

Tables 22.4 and 22.5 list important infectious and noninfectious causes of diarrhea. Table 22.6 describes the presentations of common pathogens that cause diarrheal illnesses and therapeutic options.

Diagnostic testing

Diagnostic testing is rarely necessary for previously healthy patients presenting to the ED with diarrhea. Testing should be driven by clues obtained in the history and physical examination. Patients with chronic diarrhea can typically be managed in an outpatient setting. Patients who appear ill, present with bloody diarrhea, or are immune compromised are more likely to have unusual pathogens, and therefore warrant diagnostic investigation.

Table 22.4 Infectious causes of diarrhea

Toxin producers	**Invasive organisms**
Aeromonas species	*Aeromonas* species
Bacillus cereus	*Campylobacter*
Ciguatera fish toxin	*Entamoeba histolytica*
Clostridium difficile	Enteroinvasive *E. coli*
Clostridium perfringens	Norwalk agent
Cryptosporidium	Rotavirus
Enteropathogenic/adherent *E. coli*	*Salmonella*
Enterotoxigenic *E. coli*	*Shigella* species
Giardia organisms	*Vibrio parahaemolyticus*
Helminths	*Yersinia*
Hemorrhagic *E. coli*	**Parasites**
Klebsiella pneumoniae	*Cryptosporidium*
Staphylococcus aureus	*Entamoeba histolytica*
Vibrio cholera	*Giardia lamblia*
	Isospora belli

Table 22.5 Non-infectious causes of diarrhea

Bowel-altering surgery
Bowel obstruction
Cancer
Constipation (“overflow” diarrhea)
Hyperthyroidism
Inflammatory bowel disease
Irritable bowel disease
Lactose intolerance
Laxative abuse
Mesenteric ischemia
Reaction to medications

Laboratory studies

Complete blood count

A complete blood count should be reserved for patients with significant blood loss or systemic toxicity. Nonspecific findings may include leukocytosis with a leftward shift. Eosinophilia is rarely seen; when present, it is strongly associated with *Strongyloides stercoralis* infections. Anemia may be seen with any agent causing bloody diarrhea, such as *Shigella* or *Salmonella*.

Electrolytes

A complete set of electrolytes should be considered in patients with signs or symptoms of severe dehydration or comorbid illnesses that may lead to electrolyte alteration. Diabetics, the elderly and patients on diuretics are more susceptible to developing electrolyte disturbances. Hypokalemia, hyponatremia and metabolic acidosis may be identified secondary to bicarbonate loss (this may be preceded by a contraction alkalosis).

Fecal leukocytes (Wright stain)

Normally, stool should not contain leukocytes. The presence of fecal leukocytes and occult blood suggests a bacterial etiology for the diarrheal illness.

Stool cultures

Stool cultures are rarely necessary in the ED. They may be helpful, however, in patients with invasive diarrhea, stool (Wright stain) positive for fecal leukocytes, or public health concerns (food handlers, daycare workers, health care workers, local outbreak). Stool cultures may be most beneficial in children, toxic patients, immune-compromised patients, and patients with bloody diarrhea, purulent diarrhea, or diarrhea that lasts longer than 7 days.

Stool ova and parasites

This test should not be routinely obtained and should be reserved for patients with chronic diarrhea (*Entamoeba histolytica, Cryptosporidium*), history of travel to developing countries (*Giardia*, *Cyclospora*, *Cryptosporidium*), daycare exposure (*Cryptosporidium*, *Giardia*), or HIV/AIDS (*E. histolytica*, *Giardia*).

Giardia*-specific antigen (GSA)*

This test is often used in disease outbreaks (e.g., schools and daycare settings). It should also be considered if the patient has been exposed to poor sanitation, has traveled to developing countries, or has been exposed to fresh-water lakes.

Clostridium difficile *toxins A and B*

This test should be ordered if *C. difficile* colitis is suspected in patients with a diarrheal illness preceded by antibiotic use. Other risk factors for this condition include age over 65 years, recent hospitalization, or residence in or exposure to extended-care facilities.

Escherichia coli *O157:H7 toxin*

This test should be ordered in patients with diarrhea (bloody or not) at risk for exposure to this toxin (contaminated beef or water, or exposure to someone with known disease). *E. coli* O157:H7 may be responsible for up to 36% of all cases of bloody diarrhea. The lab should be informed that you suspect this disease, since a special MacConkey sorbitol agar media is needed.

Pregnancy test

A pregnancy test is recommended for women of child-bearing age with any abdominal complaint (cramping, burning or pain). A positive pregnancy test may alter the diagnostic or therapeutic approach in a patient with diarrhea.

Table 22.6 Summary of common pathogens that cause diarrheal illnesses, their presentations and therapeutic options

Infectious agent	Setting	Fever	Abdominal Pain	Emesis	Bloody Stool	Fecal Leukocytes	Therapy (adults)	Clinical pearls
Norovirus (e.g., Norwalk virus)	Epidemic winter outbreaks in nursing homes, cruise ships; undercooked shellfish contaminant	+/–	+	+	–	–	Symptomatic therapy	Vomiting more common in children; diarrhea more common in adults
Rotavirus	Infants and young children	–	–	+	–	–	Oral rehydration solution	Early caloric refeeding; vaccines now available
Vibrio parahaemolyticus, *Vibrio vulnificus*	Seafood contaminant	+/–	+/–	+/–	+	+	Levofloxacin 500 mg PO QD × 3 days	Also cause wound infections from contaminated water
Campylobacter jejuni	Undercooked poultry	+	+	+	+	+	Erythromycin 500 mg PO BID × 5 days	High prevalence of resistance to fluoroquinolones
Yersinia enterocolitica	Pork, raw milk	+	+	+	+	+	Ciprofloxacin 500 mg PO BID × 3 days	Blood transfusion contaminant
Shigella	Community acquired, person-to-person	+	+	+	+	+	Ciprofloxacin 500 mg PO BID × 3 days	Can also cause HUS
Salmonella	Foodborne transmission	+	+	+	+	+	Ciprofloxacin 500 mg PO BID × 3 days	Symptomatic therapy for mild illness; reserve antibiotics for severe illness
Shiga toxin producing *Escherichia coli* (including O157:H7)	Ground beef, sprouts	–	+	+/–	+	+/–	Avoid antibiotics and antimotility agents	Antibiotic and antimotility therapy increase the risk of HUS
E. coli	Contaminated uncooked vegetables, fruits	+	+	+	+	+	Ciprofloxacin 500 mg PO BID × 3 days	Most common cause of traveler's diarrhea
Staphylococcus aureus	Undercooked meat	+	+	+	–	–	Symptomatic therapy	Reheated, undercooked foods
Clostridium botulinum	Preserved meat, fish, vegetables, honey					–	None	Very difficult to culture organism
Clostridium difficile	Nosocomial; postantibiotic complication	+	+	–	+	+	Metronidazole 500 mg PO TID × 10–14 days Vancomycin 125 mg PO QID × 10–14 days	Reports of more virulent strains and of community-acquired infection
Clostridium perfringens	Incompletely cooked meat	+	+	+	–	–	Metronidazole 500 mg PO TID × 10 days	Enterotoxin-mediated illness
Vibrio cholerae	Waterborne transmission	–	+/–	+/–	–	–	Doxycycline 300 mg PO × 1 dose	Primary therapy is rehydration
Giardia sp	Contaminated water; day-care centers	–	+	+/–	–	–	Metronidazole 500 mg PO TID × 5 days	Most common GI parasite in United States
Entamoeba histolytica	Travel to tropics	+	+	+/–	+	–	Metronidazole 750 mg PO TID × 10 days *then* Paromomycin 25 mg/kg/day PO TID × 7 days	Associated with liver abscesses
Cryptosporidium sp	Contaminated water; immunocompromised hosts	+/–	+/–	+/–	–	–	Metronidazole 750 mg PO TID × 10 days *then* Paromomycin 25 mg/kg/day PO TID × 7 days	Highly active antiretroviral therapy sufficient to achieve immunologic restitution in AIDS patients
Cyclospora sp	Foodborne transmission	+/–	+/–	+	–	–	TMP/SMX DS 1 tab PO BID × 7–10 days	Fatigue, which may be profound, is present in ≥90% of patients; immunocompromised patients may require a longer duration of therapy
Isospora sp	Fecal–oral transmission, contaminated water	+/–	+/–	+/–	–	–	TMP/SMX DS 1 tab PO BID × 10 days	Prevalent in AIDS patients; immunocompromised patients may require a longer duration of therapy

+: common; –: not common; +/–: occasional; AIDS: acquired immunodeficiency syndrome; BID: twice a day; DS: double strength; GI: gastrointestinal; HUS: hemolytic uremic syndrome; PO: by mouth; QID: four times a day; TID: three times a day; TMP/SMX: trimethoprim-sulfamethoxazole.

Adapted from Salen PN. Diarrhea. In Wolfson AB (ed), *Harwood-Nuss' Clinical Practice of Emergency Medicine*, 5th ed. Lippincott Williams & Wilkins, Philadelphia, PA, 2010.

Radiologic studies

Radiologic studies are only indicated when another disease process, such as small or large bowel obstruction, constipation, or toxic megacolon, is suspected.

General treatment principles

Figure 22.1 describes an algorithmic approach to patients with diarrheal illnesses.

Rehydration and electrolyte replacement

Initial treatment consists of volume repletion. Mild dehydration can be treated with oral fluids. Oral solutions should contain some glucose to stimulate resorption of water by the small intestine. This is especially important in children, who have lower glycogen stores. Milk products should be avoided since many patients develop a temporary lactase deficiency. Moderate to severe dehydration should be treated with intravenous (as well as oral) fluids. One to two liters of normal saline or D5 normal saline is generally sufficient in adults. In patients with electrolyte abnormalities (most commonly hypokalemia and hypochloremia), intravenous fluids containing these depleted electrolytes can be administered in the ED to correct imbalances (it is usually not necessary to supplement potassium in the initial fluid bolus). In rare cases, these electrolyte abnormalities can be corrected using enteral replacement therapy.

Antimotility agents

Antimotility agents may be used for symptomatic relief in patients with acute diarrhea but without fever or bloody stools (Table 22.7). These medications should be used with caution in patients with infectious diarrhea, as they may precipitate toxic megacolon and/or delay the excretion of pathogens (leading to prolonged symptoms). These agents should be avoided in patients with suspected *E. coli* O157:H7 infection, as they may facilitate the development of the hemolytic-uremic syndrome (HUS). Additionally, diphenoxylate should be administered with caution in children, particularly less than 2 years of age, as it has been linked to respiratory failure, coma and death.

Antibiotics

The use of antibiotics for acute diarrheal illnesses should be scrutinized since the majority of cases are viral and self-limited. Of the empiric therapies available, fluoroquinolones offer the greatest proven advantage. Resistance to this class of antibiotics is generally low (refer to local

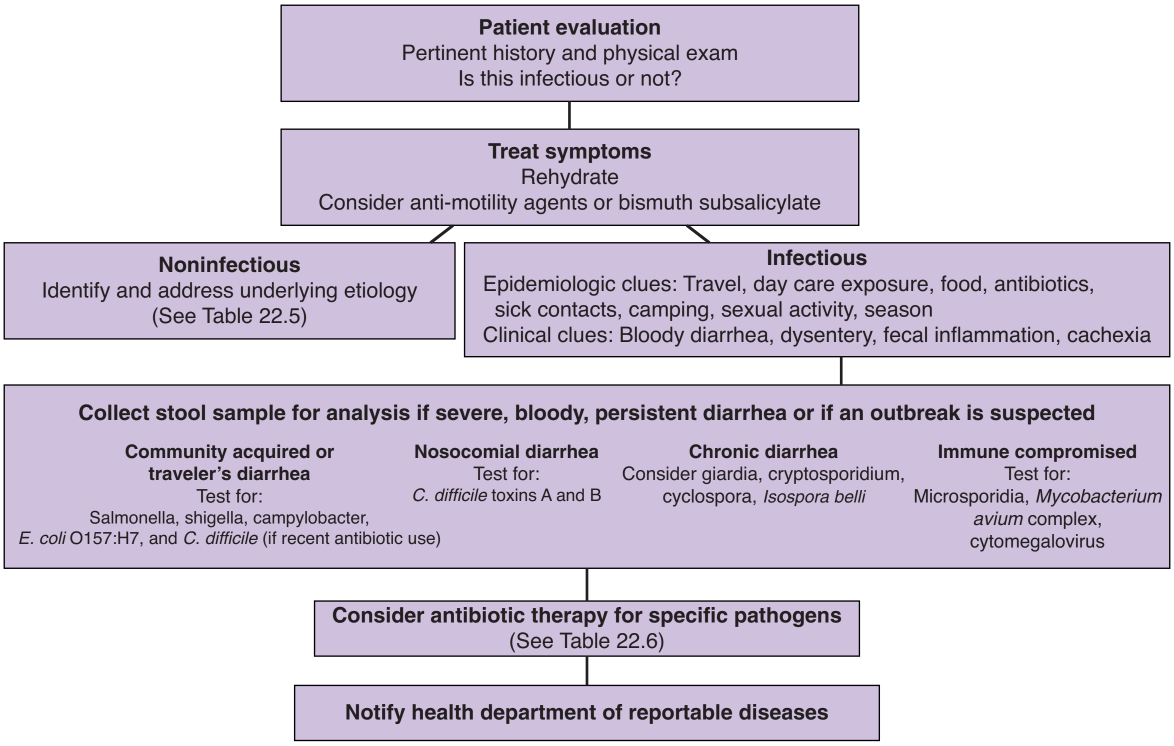
Patient evaluation
Pertinent history and physical exam
Is this infectious or not?
Treat symptoms
Rehydrate
Consider anti-motility agents or bismuth subsalicylate
Noninfectious
Identify and address underlying etiology
(See Table 22.5)
Infectious
Epidemiologic clues: Travel, day care exposure, food, antibiotics, sick contacts, camping, sexual activity, season
Clinical clues: Bloody diarrhea, dysentery, fecal inflammation, cachexia
Collect stool sample for analysis if severe, bloody, persistent diarrhea or if an outbreak is suspected
Community acquired or traveler's diarrhea
Test for:
Salmonella, shigella, campylobacter, *E. coli* O157:H7, and *C. difficile* (if recent antibiotic use)
Nosocomial diarrhea
Test for:
C. difficile toxins A and B
Chronic diarrhea
Consider giardia, cryptosporidium, cyclospora, *Isospora belli*
Immune compromised
Test for:
Microsporidia, *Mycobacterium avium* complex, cytomegalovirus
Consider antibiotic therapy for specific pathogens
(See Table 22.6)
Notify health department of reportable diseases

Figure 22.1
Approach to the patient with diarrhea. Adapted from Thielman NM, Guerrant RL. Acute infectious diarrhea. *N Engl J Med* 2004;350(1):38–47.

resistance patterns), and fluoroquinolones minimally interfere with endogenous colonic flora (minimizing the risk of overgrowth by *C. difficile*). In general, antibiotics decrease the length of disease by about 1 day and may be indicated in individuals with fever, fecal leukocytes, bloody diarrhea, symptoms greater than 3 days, and/or history of foreign travel (see Tables 22.6 and 22.7).

Some experts caution against the use of antibiotics in patients with suspected *E. coli* O157:H7 infection, as they may increase the risk of hemolytic uremic syndrome. This observation has been supported by small retrospective trials and *in vitro* tests that demonstrate an increase in toxin formation after antibiotic administration.

Probiotics

Probiotics are a novel therapeutic option for acute diarrheal illnesses. They are non-pathogenic microorganisms (most commonly lactic acid bacilli or the yeast *Saccharomyces boulardii*) that are used to compete against infectious agents in the treatment of acute diarrheal illnesses. A Cochrane systematic review on probiotic use for diarrhea found that in addition to standard supportive care, probiotic therapy resulted in a modest reduction in the duration of diarrheal illnesses in both adults and children.

Antiemetics

Antiemetics are generally not indicated in patients presenting with diarrhea if they are not vomiting. Some oral antiemetics, such as ondansetron ODT, may cause diarrhea in children.

Other agents

Bismuth subsalicylate may be helpful in the treatment of diarrhea.

Dietary restriction

Foods that should be avoided until the patient's diarrhea subsides include raw fruits (may induce an osmotic diarrhea), caffeine (increases motility), milk/lactose

Table 22.7 Various agents used in the management of diarrheal illness

Agent	Adult dosage	Pediatric dosage	Indication
Cefixime	400 mg PO QD	8 mg/kg/day PO QD	Gram-negative bacteria
Ceftriaxone	1–2 g IV/IM Q24h	50 mg/kg/day IV/IM divided QD/BID; not to exceed 2 g/day	Gram-negative and Gram-positive bacteria
Ciprofloxacin	500 mg PO BID	Not recommended	Empiric first-line therapy for presumed bacterial etiologies
Metronidazole	250 PO QID or 500 mg PO TID or 500 mg IV QID	30–50 mg/kg/day PO divided TID; not to exceed adult dose	Giardia, protozoa, anaerobic organisms, *C. difficile* colitis
Trimethoprim-sulfamethoxazole	1 DS tab PO BID	8–10 mg/kg/day PO in divided doses or 40 mg of trimethoprim per tsp (5 mL) PO	Empiric second-line therapy for bacterial etiologies
Vancomycin	500 mg PO QID	40–50 mg/kg/day PO divided QID; not to exceed 2 g/day	Second-line therapy for *C. difficile* colitis
Furazolidone	100 mg PO QID	5 mg/kg/day PO divided QID	Giardia and *Vibrio cholerae*
Iodoquinol	650 mg PO TID	30–40 mg/kg/day PO divided TID; not to exceed adult dose	Acute/chronic intestinal amebiasis
Paramomycin	25–35 mg/kg/day PO divided TID	25–35 mg/kg/day PO divided TID	Acute/chronic intestinal amebiasis
Bismuth subsalicylate	525 mg PO QID; not to exceed 4.2 g/day	<12 years: Not established >12 years: Administer as in adults	Cytoprotective for GI mucosa
Diphenoxylate HCl–atropine sulfate	2 tabs PO QID until the diarrhea is controlled	≤2 years: Not recommended 2–5 years: 2 mg of diphenoxylate PO TID 5–8 years: 2 mg of diphenoxylate PO QID 8–12 years: 2 mg of diphenoxylate 5 times/day	Antimotility
Loperamide	4 mg once, followed by 2 mg after each loose stool; do not exceed 16 mg/day	*Initial doses*: *2–6 years*: 1 mg PO TID *6–8 years*: 2 mg PO BID *8–12 years*: 2 mg PO TID *Maintenance*: 0.1 mg/kg PO after each loose stool, not to exceed initial dose	Antimotility

BID: twice a day; DS: double strength; GI: gastrointestinal; IM: intramuscular; IV: intravenous; PO: per os; QD: once a day; QID: four times a day; TID: three times a day.

(lactase deficiency) and sorbitol (increases the osmotic load).

Handwashing

Strict handwashing initiatives (at home, in the workplace, and especially in the hospital) play an important role in minimizing the spread of diarrheal illnesses and preventing relapse. A Cochrane systematic review projected that handwashing reduces the spread of diarrheal illnesses in children by as much as 39%.

Special patients

Although most diarrheal illnesses are self-limited and otherwise benign, special care must be taken with certain patient populations. Individuals at greatest risk for poor clinical outcomes include those who are immunecompromised, elderly, have multiple chronic comorbidities (e.g., end-stage kidney disease), and children. These patients tend to have less reserve, and therefore have much less ability to withstand even minor fluid, electrolyte, hematologic, or hemodynamic disturbances that can occur with acute or chronic diarrheal diseases. Treatment in these individuals should be dictated by their underlying condition and hydration status, as well as the likelihood of systemic toxicity.

Pediatric

Aside from the development of sepsis, the major concern in children is hydration status. This can be assessed historically by determining changes in urine output, oral intake, and/or number of wet diapers. The majority of cases in children are viral in origin, with rotavirus accounting for up to 50% of cases. Fortunately, the duration of illness is usually short and self-limited.

Elderly

The elderly may develop numerous complications from diarrheal disease. Like children, however, they are more likely to become ill faster, and may not tolerate the manifestations of their illness because they have less physiologic reserve. They may require more aggressive therapy, diagnostic studies and hospital admission.

Immune compromised

Individuals with HIV/AIDS may present with unusual infections, such as *Isospora belli*, *Cryptosporidium parvum*, *Mycobacterium avium-intracellulare*, or cytomegalovirus. They may also require more diagnostic studies, including stool ova and parasites, and stool cultures. Individuals with CD4 counts <200 tend to suffer from severe volume loss, weight loss and intractable illness despite appropriate therapy.

Travelers

Travelers with diarrheal illness are usually infected with *E. coli*, *Rotavirus*, *Salmonella*, or *Campylobacter*. Fortunately, these disease processes are self-limited, and usually resolve in a few days after a short course of antibiotics and symptomatic therapy.

Disposition

Most patients can be safely discharged from the ED with symptomatic therapy alone. A diet consisting of clear fluids containing some sugar and a few simple, solid foods started gradually and carefully (e.g., bananas, rice, applesauce and toast [BRAT]) should be recommended. Patients should also be instructed to perform strict handwashing, and limit unnecessary contacts to prevent spread (especially food handlers, daycare workers, and school attendees). Patients should follow-up with their primary care physician as needed.

All patients should be instructed to return to the ED if their symptoms worsen. Worrisome symptoms include worsening dehydration, change in behavior or mental status, severe abdominal pain, blood and/or mucus in the stool that did not previously exist, worsening fever, or simply feeling worse. Public health officials should be notified when agents such as *E. coli* O157:H7 toxin, *Salmonella*, *Shigella*, cholera, amebiasis (*E. Histolytica*), *Giardia*, or typhoid fever are diagnosed or strongly suspected (reportable diseases vary by locale).

Patients who have persistently abnormal vital signs, continued nausea, vomiting, or copious stool output may require further observation, hospital admission, and/or additional therapies such as antibiotics. Almost all patients who return to the ED for reevaluation or worsening of their symptoms will require intravenous fluid administration and careful observation, either in an observation unit, the ED, or the hospital.

Pearls, pitfalls and myths

Pearls

- Diarrhea is a common presenting ED complaint and requires a thorough understanding of its pathophysiology, epidemiology and treatment.
- History should focus on recent travel, medications, comorbid disease(s) and associated symptoms.
- Disease severity should be assessed based on vital signs and the physical examination in the context of the overall clinical picture.
- Grossly bloody diarrhea is almost always from invasive bacteria, not viral pathogens.
- The treatment of any diarrheal illness begins with rehydration.
- Laboratory and radiographic studies are rarely warranted for patients with diarrhea unless dictated

by physical findings of hypotension, tachycardia, severe dehydration, or mental status changes.
- Not all diarrheal illnesses are due to infectious etiologies.

Pitfalls

- Failure to perform a thorough history and physical examination to elicit important clues to disease etiology.
- Failure to address abnormal vital signs, such as tachycardia, fever, hypotension and tachypnea.

Myths

- *The use of antimotility agents should be avoided in all cases because it may precipitate toxic megacolon.* In fact, these agents can ameliorate symptoms of most diarrheal illnesses. There is (weak) evidence that recommends avoiding these agents if infection with *E. coli* O157:H7 is suspected.
- *All antibiotics prolong the Salmonella carrier state*. In fact, fluoroquinolones do not prolong the *Salmonella* carrier state.

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23 Dizziness and vertigo

Andrew K. Chang, MD

Scope of the problem

Dizziness, a common complaint in patients presenting to the emergency department (ED), is a disorder of spatial orientation. It is the most common complaint in patients over the age of 75 years. Approximately 7% of ED patients present with dizziness. Dizzy patients account for 1.5% of hospital admissions.

Evaluating the dizzy patient can be challenging, since it is a nonspecific symptom and is difficult to objectively measure. Although most cases are usually benign, emergency physicians need to be wary about life-threatening causes of dizziness, such as cardiac dysrhythmias and cerebrovascular events. In some cases, however, the patient can be cured at the bedside.

Anatomic essentials

Two studies performed approximately 30 years apart found that there are four general subtypes of dizziness: vertigo, near-syncope, disequilibrium and psychophysiologic dizziness. It is important to realize, however, that a person may describe more than one subtype, and on occasion will describe elements of all four.

Pertinent anatomy contributing to dizziness includes the vestibular, visual, proprioceptive, cardiac and central nervous systems (CNS).

Vertigo

Vertigo is defined as an illusion of motion. The CNS coordinates and integrates sensory input from the visual, vestibular and proprioceptive systems. Vertigo occurs when there is a mismatch of information from two or more of these systems. Vertigo is divided into central and peripheral causes (Table 23.1). Central vertigo is generally more serious, whereas peripheral vertigo is usually benign. Often, the most effective way to rule out a central cause of vertigo is to rule in a peripheral cause.

Central vertigo indicates involvement of the cerebellum or the vestibular nuclei within the pons and medulla. Central vertigo associated with CNS ischemia is often associated with diplopia, dysarthria, dysphagia, or focal motor and/or sensory deficits, since the vertebrobasilar arterial system provides blood supply to the inner ear, brainstem and cerebellum. Ataxic gait and dysdiadochokinesis most likely signifies cerebellar disease. Some causes of central vertigo may require emergency intervention, such as cerebellar infarction or hemorrhage, basilar artery occlusion, vertebral artery dissection, and tumors of the posterior cranial fossa. Other central causes of vertigo include migraines, multiple sclerosis and hereditary ataxia.

Table 23.1 Comparison of peripheral and central vertigo

	Peripheral	Central
Onset	Sudden	Gradual
Intensity	Severe	Mild
Duration	Seconds	Continuous
Nystagmus	Fatigable	Non-fatigable
Direction of nystagmus	Unidirectional	Pure vertical, multidirectional (possibly unidirectional), may change with direction of gaze
Associated neurologic findings	None	Usually present
Hearing loss or tinnitus	May be present	None
Associated nausea or vomiting	Frequent, severe	Infrequent, mild

Peripheral vertigo indicates involvement of either the eighth cranial nerve (CN) or the vestibular apparatus of the inner ear. Benign positional vertigo (BPV), the most common cause of vertigo, results from the inappropriate presence of calcium particles (otoliths) in the semicircular canals of the inner ear. Movement of the head causes these otoliths to inappropriately trigger receptors in the semicircular canal, causing the sensation of vertigo. In Ménière's disease (another cause of peripheral vertigo), there is an increase in the volume of endolymph associated with distension of the endolymphatic system (endolymphatic hydrops), causing vertigo, fluctuating sensorineural hearing loss, and tinnitus. Ruptures of the membranous labyrinth are thought to cause the sudden episodic attacks of Ménière's disease.

Near-syncope

Near-syncope is the sensation of feeling faint. Like vertigo, it is a common experience. Unlike vertigo, there is no illusion of motion. Near-syncope is due to the global reduction of blood flow to the brain. As people rise from supine or sitting positions throughout the day, the CNS puts out a stimulus that causes vasoconstriction to combat gravitational pooling of blood in the lower extremities while preserving blood flow to the brain. There are many things that can interfere with this reflex, such as orthostatic hypotension, cardiac disease, vasovagal syndrome, hyperventilation and environmental factors. When this neural reflex fails, pallor, nausea, rubbery legs, diaphoresis and constriction of the visual fields occur. These warning signs are the brain's way of signaling the person to lie down, which makes it easier to perfuse the brain. If the person is unable to lie down, he may progress from near-syncope to syncope. If this still does not cause him to lie horizontally, the body will make antigravity postures that may be misinterpreted as a seizure. This unfortunate chain of events can lead to an unnecessary work-up.

Disequilibrium

The third category of dizziness is disequilibrium, which is a sense of unsteadiness and loss of balance involving the legs or trunk. Disequilibrium occurs because of disruption between the sensory inputs and motor outputs, which often results in an unsteady gait. This is usually a disease of the elderly, as there is an age-related decline in the ability of the CNS to process sensory inputs and control postural reflexes. Disequilibrium is often exacerbated by unfamiliar surroundings, uneven ground, or poor lighting. A common cause is cervical spondylosis, which leads to spinal cord myelopathy. Patients have poor proprioception in the legs, which leads to a stiff-legged gait. These patients usually demonstrate a positive Romberg test. Other causes of disequilibrium are listed in Table 23.2.

Table 23.2 Causes of disequilibrium

Cervical spondylosis
Parkinson's disease
Cerebellar disease
Hydrocephalus
Multi-infarct syndrome
Peripheral neuropathy
Bilateral vestibulopathy

Psychophysiologic dizziness

The fourth category of dizziness is psychophysiologic dizziness, also known as nonspecific dizziness. The mechanism is poorly understood but is felt to result from impaired central integration of sensory signals. Patients experience feelings of dissociation, as though one has left one's own body. These patients are often in a hypervigilant state and constantly monitor themselves for signs of impending dizziness. Their exaggeration of reactions to normal changes often induces great psychologic stress, including hyperventilation. In reality, their symptoms are actually quite mild, and anxiety is felt to be the *sine qua non* of psychophysiologic dizziness. Indeed, dizziness is the most common somatic symptom associated with panic disorder.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 23.3).

History

The medical history provided by the patient or witnesses is the most important source of information in the evaluation of the dizziness. Two office-based studies found that the etiology of dizziness could be made using history alone in approximately 70% of patients.

Table 23.3 Dizziness and vertigo red flags

History	Concerning diagnosis
Headache with vertigo	Lateral brainstem or cerebellar infarction
Chest pain, shortness of breath, or palpitations	Cardiac dysrhythmia
Recent viral illness	Vestibular neuritis and/or labyrinthitis
Duration of each episode of vertigo	*Seconds:* Benign positional vertigo *Minutes:* Transient ischemic attack or vertebrobasilar insufficiency *Hours:* Ménière's disease, migraine *Days:* Vestibular neuritis, labyrinthitis
Hearing loss	Cerebellopontine angle tumor Ménière's disease (fluctuating hearing levels) Medication toxicity (aspirin, aminoglycosides)
Examination finding	**Concerning diagnosis**
Vertical or downbeat nystagmus	Central cause of vertigo
Focal neurologic deficits	Central cause of vertigo or dizziness
Hypertension	Vertebrobasilar insufficiency, cerebellar infarction or hemorrhage
Hypotension, tachycardia, bradycardia	Cerebral hypoperfusion leading to near-syncope or syncope
Ataxia	Cerebellar disease

What do you mean by dizzy?

In emergency medicine, time frames are often constrained, and the history of present illness is usually obtained in the form of closed-ended questions. However, the dizzy patient is best approached using open-ended questions. It is usually counterproductive to suggest definitions for patients, such as asking them whether the room spins or whether they feel lightheaded. Patients who present with dizziness are often very suggestible and tend to answer affirmatively to suggestive questions. In addition, their symptoms can persist or recur. Their history thus becomes distorted and can cause confusion for future emergency physicians or consultants.

Patients with vertigo will frequently offer that the "room is spinning." However, other descriptions such as rocking, tilting, somersaulting, or descending in an elevator may also be used to describe vertigo.

Patients with near-syncope generally respond that they feel like they are "going to faint."

Patients with disequilibrium typically respond that they feel like they are "going to fall" or that they feel "unsteady on their feet."

Patients with psychophysiologic dizziness commonly share that they feel as if they have "left their own body," or that they are "floating or swimming." Some patients describe a spinning sensation inside their head. Unlike vertigo, this type of spinning is not associated with an illusion of motion of the environment, and the patient does not have nystagmus on examination. In some cases, patients may be unable to describe their dizziness using words other than "dizzy."

Have you had any (recent or past) history of head trauma?

Because BPV is generally a disease of the elderly, the emergency physician should ask younger patients about a history of head trauma (head trauma can dislodge otoliths from the utricle, allowing them to enter the semicircular canals).

Do you have any new neurologic symptoms?

Diplopia, dysarthria, dysphagia, gait abnormalities and other focal neurologic complaints are concerning for a central cause of vertigo and dizziness.

Have you recently had a viral illness?

Labyrinthitis and vestibular neuritis, which are common causes of peripheral vertigo, are often associated with current or recent viral illness.

How long do your symptoms of vertigo last?

In general, episodes of vertigo vary depending on the disease process. For BPV, episodes last for seconds at a time; for a transient ischemic attack (TIA) or vertebrobasilar insufficiency, episodes last for minutes; for Ménière's disease and migraines, episodes last for hours; and for vestibular neuritis and labyrinthitis, episodes last for days.

The duration of symptoms is helpful in differentiating BPV from labyrinthitis and vestibular neuritis. The patient with BPV has episodes of vertigo that last only seconds at a time and are caused by head movements. However, the patient may describe continuous vertigo when in fact he is experiencing repeated attacks every time he turns his head. For this reason, it is important for the emergency physician to elicit how long each *individual* episode of vertigo lasts. Labyrinthitis and vestibular neuritis, on the other hand, tend to be continuous and last for several days. Vertigo may or may not be worsened with head movement. Therefore, during history taking, if a patient states that the room is spinning and his head is still (and has not been manipulated or moved), the diagnosis is probably not BPV.

Is your hearing affected?

The key to the diagnosis of Ménière's disease is fluctuating hearing levels in patients with episodic vertigo. Hearing is also affected in labyrinthitis, which distinguishes it from vestibular neuritis. Cerumen impaction, otitis media and cerebellopontine angle tumors may also result in hearing loss. Tinnitus (the perception of sound in the absence of an acoustic stimulus) occurs with Ménière's disease, acoustic neuromas and medication toxicity.

Do you have a headache?

Vertigo associated with migraine is thought to be due to vasospasm or to an inherited metabolic defect. Vertebrobasilar insufficiency is usually caused by atherosclerosis of the subclavian, vertebral and basilar arteries. Vertigo and headache may represent infarction of the lateral brainstem or cerebellum.

Do you have chest pain, shortness of breath, or palpitations?

Cardiac dysrhythmias produce spontaneous episodes of dizziness that can occur in any position and can be associated with other cardiac symptoms, such as palpitations and chest pain. Intermittent dysrhythmias may not be identified on a single electrocardiogram, so patients with episodic near-syncope of unknown cause should undergo cardiac monitoring to search for sinus pauses, sinus bradycardia, atrial fibrillation, and sustained supraventricular and ventricular tachycardias.

What makes your dizziness worse? Standing from a sitting or reclining position? Exertion? Walking or standing compared with sitting or lying down?

Orthostatic hypotension is usually due to acute blood loss, dehydration, over-diuresis, or antihypertensive medications. Gravitational pooling of blood in the legs occurs when the patient stands.

Symptoms that are exacerbated with head turning, lying down, or rolling over in bed are more suggestive of vertigo.

In vasovagal or neurally mediated near-syncope, the blood pressure is not necessarily reduced immediately upon standing, as it is in orthostatic hypotension.

Disequilibrium is typically worse while walking or standing, but patients are relatively asymptomatic while sitting or lying down.

Do your attacks occur with certain foods?

In susceptible patients, panic attacks can be precipitated by numerous substances, including caffeine and lactose. One hypothesis is that panic attacks result from loss of central control of the locus ceruleus, leading to the episodic release of catecholamines. These patients often have symptoms of agoraphobia as well.

Additional questions

Additional important historical questions that should be asked include how the dizziness began, previous episodes of dizziness, how frequently the attacks occur, what the provoking and palliating factors are, and associated symptoms.

Past medical

A detailed medical history is important to obtain since there are many factors that cause or exacerbate dizziness. Drug and alcohol abuse, previous psychiatric history, medical diseases including diabetes and heart disease, and certain neurologic diseases, such as seizures and migraine, are important to elicit from the patient.

Do you have a history of anxiety or panic attacks?

Hyperventilation, which commonly occurs in anxious patients, lowers the carbon dioxide concentration in the blood. This leads to vasoconstriction of cerebral blood vessels, which may contribute to near-syncope. However, it is important to be aware that the panic attack may actually be intermittent episodes of supraventricular tachycardia, which can also cause anxiety and palpitations.

What medications do you take?

A medication history is important, as many medications are vestibulotoxic. Common examples include aspirin and aminoglycosides. However, vertigo itself is rarely caused by medications, since both sides of the vestibular apparatus are usually affected equally.

Physical examination

General appearance

The general appearance of the dizzy patient varies widely, from the healthy young adult to the frail elderly patient. Patients who are dizzy and vomiting generally appear extremely uncomfortable, and may even be ashen and diaphoretic.

Vital signs

Blood pressure

Hypertension in a dizzy patient should raise concern for vertebrobasilar insufficiency, or cerebellar infarction or hemorrhage as a possible cause. Hypotension can lead to decreased cerebral perfusion and may be associated with near-syncope. Orthostatic vital signs can be checked but are notoriously unreliable in elderly patients. Differences in blood pressures between arms can indicate subclavian steal syndrome (which may result in vertebrobasilar insufficiency) or aortic dissection.

Heart rate

Both tachycardia and bradycardia can impair cardiac output and lead to near-syncope via cerebral hypoperfusion.

Respiratory rate

Hyperventilation can contribute to hypoperfusion of the brain through vasoconstriction of cerebral blood vessels.

Temperature

Fever alone may produce a sensation of dizziness, and may also accompany CNS or other infections.

Head, eyes, ears, nose and throat

Eyes

The emergency physician should ask the patient to look to the right and left to check for the presence of nystagmus. Avoid having the patient fixate on an object, such as a pen or finger, since visual fixation can inhibit nystagmus. The physician should note the nature of the nystagmus (horizontal, rotary or torsional, horizontal-rotary, vertical, or vertical-rotary), its direction (based on the direction of the fast component), and its duration. With vestibular disease, the fast component usually beats toward the side of the lesion. In peripheral vertigo, spontaneous nystagmus usually continues in one direction even when the direction of the gaze changes. In contrast, central causes (such as cerebellar or brainstem infarction or hemorrhage) result in nystagmus that changes direction with change in the gaze direction. Purely vertical nystagmus almost always indicates a central cause. The nystagmus of peripheral vertigo is typically fatigable (extinguishes with repeated testing), whereas the nystagmus of central vertigo is not. There are exceptions to this rule, such as BPV caused by cupulolithiasis, which results in non-fatigable nystagmus. In addition, the presence of nystagmus at extreme end-gaze is seen in up to 60% of normal people. In patients with suspected BPV, the Dix-Hallpike test confirms the diagnosis, with reproduction of the vertigo as well as ipsilateral and upbeat (towards the patient's forehead) nystagmus.

Ears

The external auditory canal should be inspected for vesicles (Ramsay Hunt syndrome), cerumen and cholesteatoma. The tympanic membranes should be visualized for signs of otitis media. A perforated or scarred tympanic membrane may indicate perilymphatic fistula, which can be confirmed with pneumatic otoscopy. Hearing should also be tested. The emergency physician can use a ticking watch or rub fingers near the patient's ears. Unilateral hearing loss is suggestive of labyrinthitis, cerumen impaction, Ménière's disease or acoustic neuroma, although the latter usually presents with gradual hearing loss.

Cardiovascular

The heart should be auscultated for the presence of dysrhythmias and murmurs. The presence of murmurs may indicate aortic stenosis or hypertrophic obstructive cardiomyopathy, both which may decrease cardiac output. In addition, the carotid arteries should be auscultated for the presence of bruits, which may indicate carotid stenosis as a contributing cause of cerebral hypoperfusion.

Neurologic

All patients with dizziness need a comprehensive neurologic examination, with special attention to the CNs, cerebellar examination, and gait testing.

Cranial nerves

CN abnormalities strongly suggest a central process:

- CN I dysfunction is suggested by uni- or bilateral decrease in or loss of smell.
- CN II dysfunction is suggested by loss in visual acuity and abnormalities on funduscopic examination (papilledema, optic atrophy).
- CN III, IV, or VI dysfunction is suggested by dysconjugate gaze with formal extraocular muscle (EOM) testing.
- CN V dysfunction is suggested by weak or absent contraction of the temporal and masseter muscles, or decrease in or loss of facial sensation. Loss of the corneal reflex also suggests CN V dysfunction.
- CN VII dysfunction is suggested by facial droop or weakness of one side of the face.
- CN VIII dysfunction is suggested by decreased hearing.
- CN IX or X dysfunction is suggested by hoarseness or a nasal quality to the patient's voice, a history of swallowing difficulty, and asymmetric movements of the soft palate and pharynx when the patient is asked to say "aah."
- CN XI dysfunction is suggested by atrophy or weakness of the trapezius and platysma muscles.
- CN XII dysfunction is suggested by dysarthria and deviation of the protruded tongue towards the involved side.

Cerebellar

Cerebellar function can be evaluated using rapid alternating movements or point-to-point testing. Slow, irregular and clumsy movement that occurs with rapid alternating movements indicate cerebellar disease, and is called *dysdiadochokinesis*. Cerebellar disease also results in movements that are clumsy, unsteady and inappropriately varying in their speed, force and direction.

The Romberg test is a functional test of position sense (Figure 23.1). The patient stands with his feet together and is then told to close his eyes. In ataxia due to loss of position sense, vision compensates for the sensory loss. When the eyes are closed, the patient loses balance, resulting in a positive Romberg sign. With cerebellar ataxia, the patient has difficulty standing with his feet together regardless of whether the eyes are open or closed.

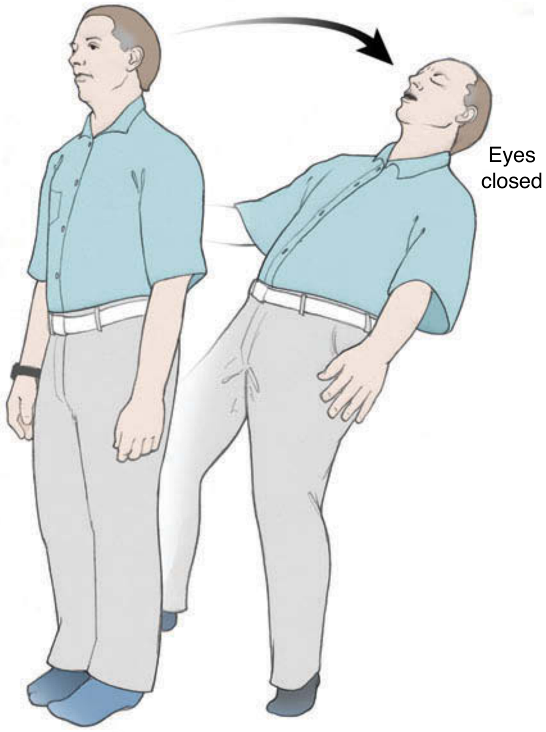

Figure 23.1
Positive Romberg test. © Chris Gralapp.

Gait

Whenever possible, gait should be tested. Ataxia (a gait that lacks coordination, with reeling and instability) may be due to cerebellar disease, loss of position sense, or intoxication. Tandem walking may bring out an ataxia not previously obvious. The broad-based ataxic gait of cerebellar disorders is readily distinguished from the milder gait disorders seen with vestibular or sensory loss.

Rectal

A rectal examination may identify gastrointestinal bleeding suggestive of anemia, and should be considered in the dizzy patient with a history consistent with near-syncope.

Clinical tests

Orthostatic vital signs

Orthostatic hypotension is generally defined as a drop in systolic blood pressure of at least 15–20 mmHg within 2 minutes of standing upright. Orthostatic vital signs may suggest hypovolemia but are nonspecific, especially in the elderly, and should not be considered pathognomonic.

Hallpike

For patients with a history consistent with vertigo, a Hallpike test (also known as the Dix–Hallpike, Nylan–Barany, or Barany test) should be performed at the bedside (Figure 23.2). This diagnostic test confirms BPV of the posterior semicircular canal, the most common cause of BPV. The test is performed as follows: the patient sits upright in the gurney with the head turned 45° to one side. The patient is then guided down to the supine position with the head overhanging the edge of the gurney. The eyes are viewed for evidence of torsional nystagmus, and the patient is questioned regarding reproduction of symptoms. By initially turning the head 45° to one side, the posterior semicircular canal becomes aligned in the direction of movement when the head is laid down. This serves as the most provocative way to move the otoliths (if they are present in the posterior semicircular canal) and reproduce symptoms. In the head-hanging position, the eyes beat upward (toward the forehead) and toward the affected ear in the fast phase. The nystagmus fatigues with repeated positioning, and there is usually a brief latency from the time the head-hanging position is achieved to the onset of nystagmus. The patient is then returned to the supine position and the test is then repeated with the head turned in the opposite direction. This test does not need to be done rapidly, as it is a "positional" as opposed to a "positioning" test. Although it is theoretically possible to have bilateral BPV, generally only one side tests positive in patients with BPV. This positive side serves as the starting point for the Epley maneuver, described in the treatment section.

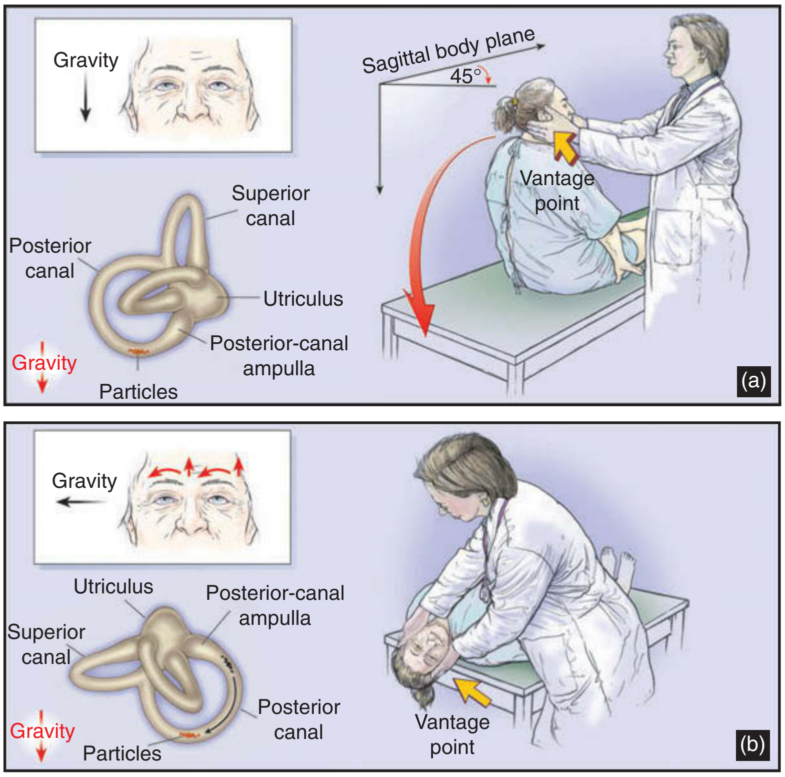

Figure 23.2
The Dix–Hallpike Test of a patient with benign paroxysmal positional vertigo affecting the right ear. In Panel A, the examiner stands at the patient's right side and rotates the patient's head 45 degrees to the right to align the right posterior semicircular canal with the sagittal plane of the body. In Panel B, the examiner moves the patient, whose eyes are open, from the seated to the supine right-ear-down position and then extends the patient's neck slightly so that the chin is pointed slightly upward. The latency, duration and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The red arrows in the inset depict the direction of nystagmus in patients with typical benign paroxysmal positional vertigo. The presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown. Reprinted from Furman JM, Cass SP, Benign paroxysmal positional vertigo, *N Engl J Med* 1999;341(21):1590–6. Copyright 1999 Massachusetts Medical Society. All rights reserved.

Head-thrust

This test should be performed if unilateral peripheral vestibular loss is suspected, as in vestibular neuritis or labyrinthitis. These acute peripheral vestibular dysfunction syndromes are characterized by a rapid onset of severe vertigo, nausea, vomiting and gait instability. To perform the head-thrust test, the patient's head is quickly rotated about 15° to the side while the patient fixates his vision on the examiner's nose. With unilateral peripheral vestibular loss, the eyes cannot maintain focus, and a saccade (quick rotation of the eyes from one fixation point to another) will occur, bringing the eyes back to the examiner's nose. If a saccade is present, the test is positive and helps confirm the diagnosis of vestibular neuritis or labyrinthitis.

Hennebert

This tests for the presence of a perilymphatic fistula. If the patient develops reproduction of symptoms (vertigo, nausea and nystagmus) on pneumatic otoscopy, the diagnosis may be perilymphatic fistula. Ménière's disease and otosyphilis can cause false-positive results.

Hyperventilation

A 2-minute hyperventilation challenge is occasionally used when psychophysiologic dizziness is thought to be the cause of dizziness. However, the utility of this test remains unclear, and symptom reproduction cannot be considered diagnostic.

Differential diagnosis

Features of conditions causing peripheral and central vertigo are listed in Tables 23.4 and 23.5, respectively.

Table 23.4 Conditions causing peripheral vertigo

	Symptoms	Signs
Benign positional vertigo	Vertigo for seconds at a time	Positive Hallpike test
Cerebellar pontine angle tumors (acoustic neuroma, meningioma, dermoid)	Vertigo, deafness	Ataxia, ipsilateral facial weakness, loss of corneal reflex, cerebellar signs
Cholesteatoma	Facial twitching, various degrees of hearing loss	May have positive insufflation test
Labyrinthitis	Continuous vertigo for hours to days, decreased hearing	Positive head-thrust test, decreased hearing
Ménière's disease	Episodic vertigo, fluctuating hearing loss, ear fullness, roaring tinnitus	Low-frequency hearing loss (unilateral in most cases)
Otitis media or tympanic membrane rupture	Vertigo	Bulging or ruptured tympanic membrane, drainage (possibly)
Ototoxic drugs	Vertigo uncommon since both inner ears affected, hearing loss	Ataxia, oscillopsia
Perilymphatic fistula	"Popping" sound, hearing loss, tinnitus	Positive insufflation test
Vestibular neuritis	Continuous vertigo for hours to days, normal hearing	Positive head-thrust test

Table 23.5 Conditions causing central vertigo

	Symptoms	Signs
Basilar artery migraine	Vertigo, tinnitus, headache, visual aura	Decreased hearing, diplopia, dysarthria, ataxia, bilateral paresis, bilateral paresthesias, decreased level of consciousness
Cerebellar infarction or hemorrhage	Mild vertigo	Truncal or limb ataxia, abnormal Romberg test
Multiple sclerosis	Discrete episode of vertigo lasting several hours to weeks, usually non-recurrent	Ataxia, optic neuritis
Temporal lobe seizures	Vertigo as part of an aura	Amnesia during seizure, other associated aura symptoms present
Vertebrobasilar insufficiency	Vertigo lasting for minutes, may be provoked by position	May include diplopia, dysphagia, dysarthria and bilateral loss of vision
Wallenberg syndrome (lateral medullary infarction)	Vertigo, nausea or vomiting, dysphagia and dysphonia	Ipsilateral Horner's syndrome, facial numbness, loss of corneal reflex, paralysis or paresis of the soft palate, pharynx and larynx

Diagnostic testing

Diagnostic testing should be based on the emergency physician's history and physical examination. Not every patient with dizziness requires diagnostic testing. For example, patients who have a classic history for BPV, a positive Hallpike test on one side, and a normal neurologic examination do not necessarily need laboratory tests or imaging studies. Any patient with a focal neurologic examination should receive computed tomography (CT) of the brain and, if possible, magnetic resonance imaging (MRI) of the brain.

Laboratory tests

Screening laboratory tests may be helpful in the evaluation of the dizzy patient. A hemoglobin and hematocrit may detect anemia, and a glucose level may exclude hypo- or hyperglycemia, especially in the diabetic patient. In addition, electrolytes, renal function tests, and a toxicology screen may be helpful in certain cases.

Electrocardiogram

An electrocardiogram is appropriate if the emergency physician suspects a cardiac cause for a patient's dizziness, especially if the history is suggestive of near-syncope. This may help establish evidence of dysrhythmia or ischemia.

Radiologic studies

Any patient with concern for central vertigo or focal neurologic deficits on examination should receive advanced cranial imaging.

Cranial computed tomography

Cranial CT is commonly available but has limited utility when evaluating the posterior fossa. A negative CT in a patient with focal neurologic deficits demands further testing or subspecialty consultation (Figure 23.3).

Cranial magnetic resonance imaging

Cranial MRI is more likely to detect subtle brainstem or inferior cerebellar infarction (Figure 23.4).

General treatment principles

Symptomatic care is usually all that is needed for the dizzy patient. If a patient is nauseated or actively vomiting, intravenous (IV) hydration and antiemetic medication should be given. IV fluids should also be given if the physician suspects hypovolemia or dehydration as a contributing cause. If a cardiac cause is being considered, oxygen should be applied and an electrocardiogram obtained.

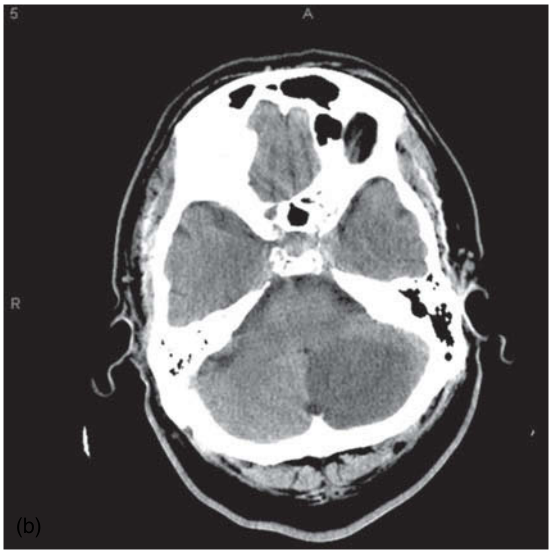

Figure 23.3
Left cerebellar infarct. Non-enhanced head CT revealing left cerebellar hemisphere infarct. Courtesy: Gus M. Garmel, MD.

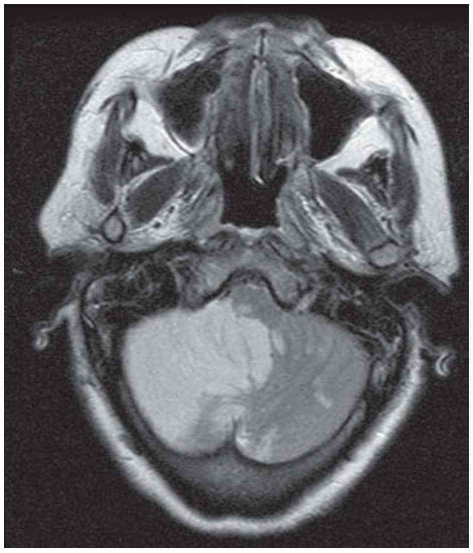

Figure 23.4
Right cerebellar infarct. T2-weighted MRI of the brain revealing high signal intensity consistent with a large right cerebellar infarct. Courtesy: Mahesh Jayaraman, MD.

Peripheral vertigo

After supportive care has been initiated, the emergency physician should determine whether or not the patient has BPV, the most common cause of vertigo. The diagnosis is based on characteristic historical features and a positive Hallpike test on one side.

The Epley (canalith repositioning) maneuver

The goal of the Epley maneuver is to move the otoliths from the posterior semicircular canal back to the utricle where they belong. Patients with BPV should have

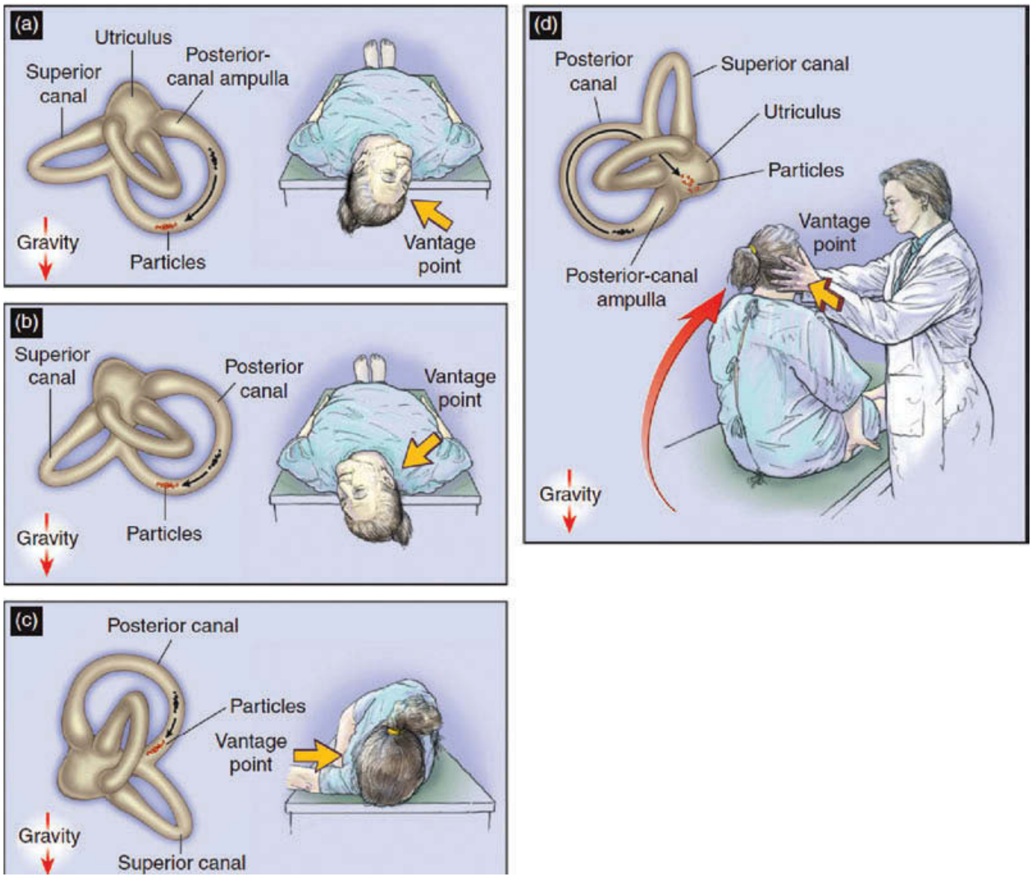

Figure 23.5
Bedside maneuver for the treatment of a patient with benign paroxysmal positional vertigo affecting the right ear. The presumed position of the debris within the labyrinth during the maneuver is shown in each panel. The maneuver is a three-step procedure. First, a Dix–Hallpike test is performed with the patient's head rotated 45 degrees toward the right ear and the neck slightly extended with the chin pointed slightly upward. This position results in the patient's head hanging to the right (Panel A). Once the vertigo and nystagmus provoked by the Dix–Hallpike test cease, the patient's head is rotated about the rostral-caudal body axis until the left ear is down (Panel B). Then the head and body are further rotated until the head is face down (Panel C). The vertex of the head is kept tilted downward throughout the rotation. The maneuver usually provokes brief vertigo. The patient should be kept in the final, face-down position for about 10 to 15 seconds. With the head kept turned toward the left shoulder, the patient is brought into the seated position (Panel D). Once the patient is upright, the head is tilted so that the chin is pointed slightly downward. Reprinted from Furman JM, Cass SP, Benign paroxysmal positional vertigo, *N Engl J Med* 1999;341(21):1590–6. Copyright 1999 Massachusetts Medical Society. All rights reserved.

the modified Epley maneuver (Figure 23.5) performed as follows: the patient's head is turned 45° to the side causing symptoms (as determined by the Hallpike test). The patient is guided to the supine position with the head hanging over the edge of the gurney. The head is then rotated 90° in the opposite direction with the face upwards, maintaining a dependent position. The patient is then asked to roll onto his side while holding the head in this position. The head is then rotated so that it is facing obliquely downward, with the nose 45° below horizontal. The patient is then raised to a sitting position while maintaining head rotation. Finally, the head is rotated to a central position and moved forward 45°. Each position is held until resolution of nystagmus occurs or for at least 30 seconds. It is not clear if the Epley maneuver should be repeated multiple times. Epley himself will perform the maneuver up to five times (personal communication) at one sitting. Other experts perform the maneuver only once since they feel that the particles will continually reintroduce themselves into the canals if the procedure is repeated. The Epley maneuver is performed at the bedside and takes approximately 2–3 minutes to complete. Patients are expected to have their symptoms reproduced during each stage of the maneuver, which indicates successful movement of the otoliths within the semicircular canal. Aside from the expected reproduction of symptoms and possible vomiting, no adverse events have been reported from performing the Epley maneuver. After the maneuver, patients are generally advised to stay in an upright position. Once the otoliths enter the utricle where they belong, they need time to reattach to the utricular macule. The time required for this process is not clear, but recent research suggests only 20 minutes is needed. The patient should remain upright for 20–30 minutes, and the Hallpike test can then be repeated. If significant nystagmus or symptoms persist, the Epley maneuver can be performed a second time.

Vestibular suppressants

The use of vestibular suppressants is based primarily on the *sensory conflict theory*. This states that when there is a mismatch of information from any of the three main sensory inputs (vestibular, visual and proprioceptive), nausea and emesis result in the acute phase, but habituation occurs over time. This mismatch of information is compared with learned prior stimuli. This process is thought to be mediated by three or four neurotransmitters: gamma amino butyric acid (GABA), acetylcholine, histamine and serotonin. Benzodiazepines work by preventing the mismatched information from being compared with prior learned stimuli. However, many experts avoid using benzodiazepines since they prevent the process of vestibular rehabilitation. Anticholinergics work by decreasing the signal-size conflict, and are thought to be the most useful agents. However, atropine is rarely used due to its serious side effects, and scopolamine has an onset of several hours, limiting its use in the ED. Antihistamines and antiserotonergic agents block the emesis response. These medications, which include promethazine and meclizine, also have anticholinergic side effects. Intravenous promethazine (Phenergan) is felt by many to be the best medication for the acutely symptomatic patient experiencing vertigo. However, the FDA recently placed a black box warning on promethazine secondary to severe tissue damage and gangrene. IV ondansetron is an alternative medication that may be used in place of promethazine.

Central vertigo

The treatment of central vertigo depends on the cause. Antiplatelet agents should be started in consultation with a neurologist.

Near-syncope

For near-syncope due to orthostatic hypotension, removal of offending medications or correction of volume depletion will often be therapeutic. In patients with autonomic insufficiency, increased salt intake can increase blood volume, and elastic stockings can prevent pooling of blood in the lower extremities. For vasovagal near-syncope, reassurance is usually all that is needed. Patients can also increase their fluid intake and avoid conditions that predispose them to hypotension and dehydration. Near-syncope associated with impaired cardiac output can be a serious warning sign. Cardiac dysrhythmia management depends on the actual rhythm, and many patients can be helped with the insertion of a pacemaker even if the diseased heart cannot be treated. Hyperventilation-induced near-syncope should be treated by educating and reassuring the patient that this is a benign disorder. If associated with panic disorder, pharmacologic treatment (i.e., tricyclic antidepressants or selective serotonin reuptake inhibitors) may be considered after discussion with the patient's primary care physician.

Disequilibrium

For disequilibrium, gait and balance training may be beneficial for those patients without cerebellar lesions. A cane or walker often helps most patients. Patients with alcoholic cerebellar degeneration may show improvement following discontinuation of alcohol consumption. Parkinson's disease may be improved with L-DOPA. Hydrocephalus-induced disequilibrium can be reversed with shunt placement.

Psychophysiologic dizziness

For psychophysiologic dizziness, supportive psychotherapy in addition to medications (i.e., benzodiazepines, tricyclic antidepressants and selective serotonin reuptake inhibitors) may be helpful. These medications should be started only after consultation with the patient's primary care physician or specialist.

Special patients

Pediatric

Children rarely complain of dizziness. When they do, they present with similar vestibular and non-vestibular problems as adults. Otitis media and its complications (suppurative labyrinthitis or mastoiditis) can lead to vestibular complaints. Acute cerebellar ataxia can follow a viral infection and usually occurs in children under the age of 6 years. Infection or volume depletion may be important clues to the diagnosis. Disequilibrium in a young person suggests neurologic disease. Also, near-syncope in a young athlete who was exercising may indicate serious cardiac disease, such as hypertrophic obstructive cardiomyopathy or aortic stenosis.

Elderly

Dizziness is more common in the elderly and can result in falls, causing hip fractures and intracerebral hemorrhage. Elderly patients are more likely to present with central causes of vertigo, such as ischemic cerebrovascular disease, and are more likely to be debilitated by symptoms of peripheral vertigo.

Disposition

Admission of patients with dizziness or vertigo should be based on the underlying etiology or associated symptoms. Patients with peripheral vertigo may be discharged home, unless they present with intractable vomiting or vertigo that cannot be controlled in the ED. Patients with an abnormal neurologic examination or increased suspicion for a serious neurologic cause should have a formal neurologic consultation or be admitted for observation. Similarly, patients in whom cardiac dysrhythmias are likely should be admitted for observation and cardiac monitoring.

Pearls, pitfalls and myths

- A detailed neurologic examination is important in differentiating central from peripheral vertigo. Since the cerebellovestibular nuclei are tightly packed with other tracts in the brainstem, any lesion that affects these nuclei will likely affect others as well. Therefore, patients with central vertigo typically have an abnormal neurologic examination.
- The Hallpike test should be performed in patients who present with vertigo, but this test does not need to be performed rapidly. Gently guide the patient into the head-hanging position.
- It is important to differentiate BPV from other causes of peripheral vertigo, such as vestibular neuritis, labyrinthitis and psychophysiologic dizziness, since the Epley maneuver only works for BPV.
- A chief complaint of dizziness should not result in the knee-jerk reflex to prescribe meclizine. Although meclizine is effective in many cases of vertigo, it may worsen symptoms due to the other subcategories of dizziness.

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24 Ear pain, nosebleed and throat pain (ENT)

24A EAR PAIN

Gregory H. Gilbert, MD and S.V. Mahadevan, MD

Scope of the problem

Ear pain (otalgia) is a common emergency department (ED) complaint, prompting over 30 million physician visits each year. By the third year of life, 80% of the population will have experienced otalgia at least once. Though many conditions may cause ear pain, otitis media (OM) is by far the most common diagnosis, especially in the pediatric population. The potential for serious causes of otalgia, such as necrotizing otitis externa (OE) and mastoiditis, underscore the need for early and accurate diagnosis and treatment.

Anatomic essentials

The anatomic ear may be divided into three distinct sections: external, middle and inner (Figure 24.1). The *external ear*, consisting of the auricle (pinna) and the external auditory canal (EAC), originates at the pinna and ends at the tympanic membrane (TM). The *middle ear* is an air-containing cavity in the petrous temporal bone that houses three auditory ossicles: the malleus, incus and stapes. The middle ear is separated from the outer ear by the TM, and connects anteriorly with the nasopharynx via the eustachian tube and posteriorly with the mastoid air cells. The *inner ear* includes the cochlea, which contains the auditory receptors, and vestibular labyrinth, which contains the balance receptors. Sensory innervation of the ear is derived from branches of cranial nerves (CNs) V, VII, IX and X, as well as cervical nerves C2 and C3. These nerves course through the head, neck and chest, explaining why remote conditions may cause ear pain.

Primary otalgia (Table 24.1) refers to ear pain that results from structures directly within or adjacent to the anatomic ear. In cases of primary otalgia, the ear examination is usually abnormal and the diagnosis is usually apparent. OM and OE are the most common causes of primary otalgia.

The development of OM is thought to be associated with dysfunction of the eustachian tube. The eustachian tube protects the middle ear from nasopharyngeal secretions, allows drainage of middle ear secretions, and helps equilibrate air pressure in the middle ear. Eustachian tube dysfunction traps fluid, secretions and bacteria within the middle ear, resulting in infections. Mechanical obstruction of the eustachian tube is caused by localized inflammation secondary to upper respiratory infections (URI), allergies, or hypertrophied adenoids. Functional obstruction or collapse of the eustachian tube typically occurs in young children as a result of less fibrocartilaginous support. As we age, the eustachian tube widens, lengthens and stiffens (which may explain the decreased incidence of OM in adults).

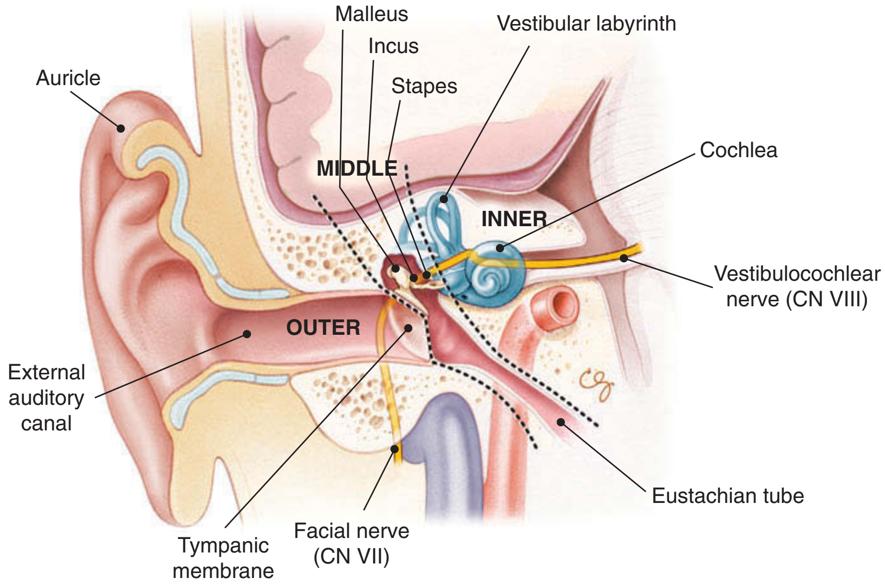

Figure 24.1
Ear anatomy. © Chris Gralapp.

Table 24.1 Causes of otalgia

Primary	Referred	Neuralgias
Trauma	Dental	Trigeminal (tic douloureux)
Infection	Temporomandibular joint disease	Herpetic geniculate (Ramsay Hunt syndrome)
Otitis externa	Abscessed teeth/dental caries	Foraminal narrowing
Otitis media	Malocclusion	
Mastoiditis	Bruxism	
Bullous myringitis	Trauma	
Pinna cellulitis	Retro- and oropharyngeal	
Foreign bodies	Tonsillitis	
Cerumen impaction	Abscess	
Cholesteatoma	Neoplasm	
Neoplasms	Nasal cavity	
	Sinusitis	
	Deviated septum	
	Throat and neck	
	Foreign body	
	Thyroid disease	
	Cervical strain	
	Neoplasm	

Adapted from Tintinalli JE (ed). *Emergency Medicine: A Comprehensive Study Guide*, 7th ed. McGraw Hill, New York, 2010.

OE results from inflammation or infection of the EAC. Prolonged exposure to moisture (e.g., swimming) or local trauma (e.g., cotton swabs or hearing aids) can disrupt the protective outer layer of the EAC, allowing bacterial penetration and ensuing infection. Necrotizing OE, also known as malignant otitis externa, is an invasive infection that affects elderly diabetics and immunocompromised patients. As the infection extends to the skull base (i.e., osteomyelitis), patients may develop facial nerve palsy and other serious neurologic sequelae (e.g., meningitis, brain abscess).

Not all ear pain originates from the anatomic ear. In patients with *referred otalgia*, the pain typically originates from outside sources (Table 24.1) as a result of shared sensory innervation by the anatomic ear and other head and neck structures. Referred otalgia may arise from pathology in the parotid glands, teeth, muscles of mastication, mandible, nasopharynx, paranasal sinuses, thyroid gland, cervical spine, upper gastrointestinal tract or upper respiratory tract (Figure 24.2). In referred otalgia, the ear examination is typically normal. Dental disorders are the most common cause of referred otalgia, and elderly patients are most likely to present with a referred cause of otalgia.

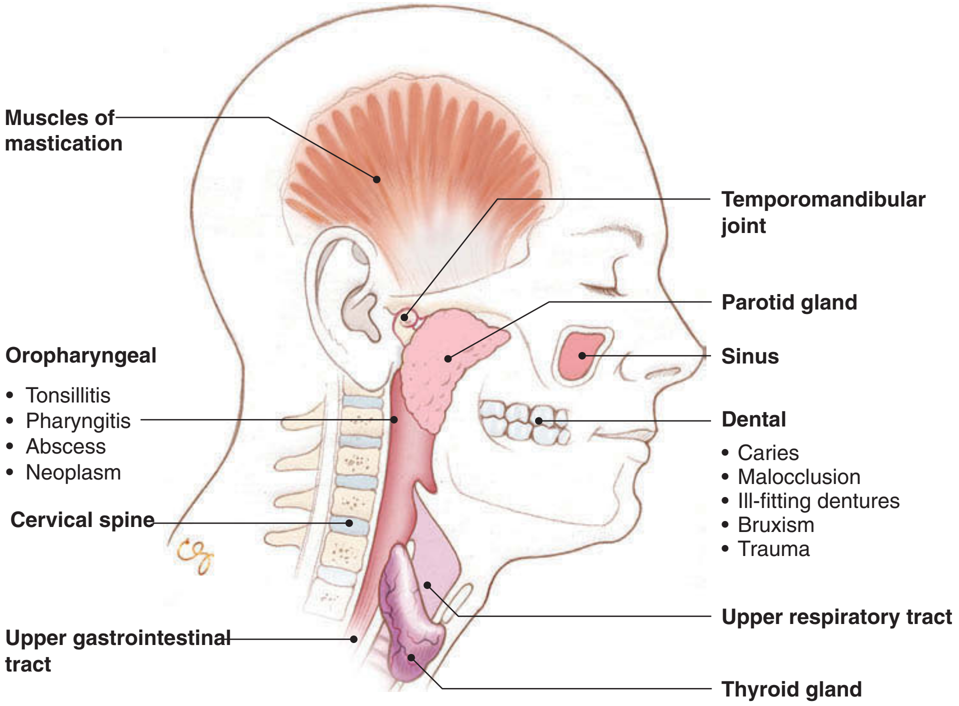
Muscles of mastication
Temporomandibular joint
Parotid gland
Sinus
Oropharyngeal
• Tonsillitis
• Pharyngitis
• Abscess
• Neoplasm
Dental
• Caries
• Malocclusion
• Ill-fitting dentures
• Bruxism
• Trauma
Cervical spine
Upper respiratory tract
Upper gastrointestinal tract
Thyroid gland
CG

Figure 24.2
Sources of referred otalgia. © Chris Gralapp

Table 24.2 Ear pain red flags

History	Concerning diagnosis
Longstanding and undiagnosed; tobacco and alcohol use; dysphagia, odynophagia, or hoarseness; weight loss; age >50 years	Head or neck malignancy
Sudden decrease in pain; direct blow to head; history of diving, air travel or noise trauma	TM perforation
Hearing loss	Otitis media, otitis externa, foreign body, TM perforation, head or neck malignancy, cholesteatoma
Chronic ear pain and drainage	Mastoiditis, cholesteatoma
High fever	Infectious etiology other than otitis media
Headache	Necrotizing otitis externa, mastoiditis, meningitis, brain abscess, cavernous sinus thrombosis
Dizziness, vertigo	Inner ear involvement
Otitis media in first year of life, previous myringotomy, or tympanostomy tube	Recurrent otitis media
Diabetes or immune compromise	Necrotizing otitis externa
Taking antibiotics	Resistant otitis media
Coronary artery disease risk factors	Myocardial infarction
Examination finding	**Concerning diagnosis**
Toxic appearance	Meningitis, sepsis
Age >50 years, ESR >50	Temporal arteritis
Posterior auricular erythema, swelling or tenderness; protrusion of auricle or loss of posterior auricular crease	Mastoiditis
Pain on insertion of otoscope speculum; pain with pulling on auricle or pushing on tragus	Otitis externa
Herpetiform vesicular eruption	Herpes zoster oticus
TM defect	TM perforation
Superior TM retraction pocket, otorrhea	Cholesteatoma
Facial paralysis (peripheral CN 7 palsy)	Mastoiditis, herpes zoster oticus, necrotizing otitis externa
Granulation tissue on the floor of the EAC	Necrotizing otitis externa

CN: cranial nerve; EAC: external auditory canal; ESR: erythrocyte sedimentation rate; TM: tympanic membrane.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 24.2).

History

Obtaining an accurate history from a young patient may be challenging. A parent or guardian may be required to describe the patient's symptoms.

How did the pain begin and how long have you had it?

Patients typically present with acute ear pain within 24 hours of onset. The ear pain may be moderate to severe, leading to difficulty sleeping and prompting an ED visit in the middle of the night. In one study, patients who delayed seeking initial care for OM were more likely to present with complications. Long-standing undiagnosed ear pain may represent an undiagnosed head or neck carcinoma.

Describe the pain?

Constant, sharp, stabbing pain with associated pressure or throbbing is typical of acute OM (AOM). The discomfort from OE may vary from itching to severe pain. Intermittent or variable pain is usually referred and may arise from the temporomandibular joint (TMJ) or teeth. Deep, unrelenting pain that keeps the patient awake at night is suggestive of necrotizing OE. Sharp, lancinating pain, arising in the tonsil and radiating to the ear, suggests trigeminal neuralgia. A sudden decrease in pain associated with discharge from the ear is typical of TM rupture. Pediatric patients may have increased fussiness and pull at their ears rather than complain of ear pain.

What makes the pain better or worse?

Pain exacerbated by eating or chewing may be referred from the TMJ or teeth. AOM typically worsens with recumbent position. The discomfort from OE is often aggravated by manipulation of the tragus or ear.

How is your hearing?

Patients may describe hearing loss, muffled hearing, or popping or crunching sounds. Hearing loss or changes may accompany OM, OE, foreign bodies (FBs) and cerumen impaction.

Any ear discharge?

Otorrhea (discharge from the ear) may occur with a ruptured TM, OE or FB. The discharge may be serosanguineous or purulent. Pain typically precedes otorrhea in OM, whereas it accompanies the drainage in OE. Chronic ear pain and drainage may represent mastoiditis or a cholesteatoma.

Any recent travel or trauma?

Diving or recent air travel could lead to TM perforation from barotrauma. A history of swimming or prior ear instrumentation often accompanies OE. A direct blow to the side of the head or noise trauma can also cause TM perforation. The use of a Q-tip to clean the ear canal can result in damage to the external auditory meatus or TM, as well as contribute to cerumen impaction. A whiplash injury or arthritis of the cervical spine can lead to referred otalgia.

Associated symptoms

General

Ask about fussiness, feeding, URI symptoms and fever. Although adults and older children can articulate ear pain, infants and toddlers may cry, fuss or refuse to eat or drink. Children with OM often have associated URIs and may present with nonspecific symptoms such as cough, vomiting and diarrhea. Very few patients with OM (only 4% in one series) have a fever >40°C. In these patients, consideration should be given to other etiologies of fever. In one prospective study, nearly all patients with OM had ear pain, decreased hearing and URI symptoms, but only 9% of these patients had fever.

Head and neck

Ask about headache, sinus problems, dizziness, bruxism, difficulty swallowing and changes in speech. As otalgia may be referred, a complete review of head and neck symptoms is imperative. Headache may occur with sinusitis, mastoiditis and necrotizing OE. Headache can also accompany complications of these conditions, such as meningitis, brain abscess and cavernous sinus thrombosis. The presence of dizziness or tinnitus suggests inner ear involvement. Patients who grind their teeth in the middle of the night (bruxism) are more likely to have TMJ syndrome or a dental problem. Difficulty swallowing and speaking suggests referred pain from a retropharyngeal or peritonsillar abscess, or possibly a pharyngeal or laryngeal tumor.

Past medical

Patients with cervicofacial pain syndromes such as myalgias, neuralgias or arthritis may have otalgia. Children diagnosed with AOM by 1 year of age are more likely to have recurrences, with 33% of patients getting five or more episodes by the age of 6 years. Previous myringotomy or tympanostomy tube placement usually indicates either a history of OM with a complication or frequent recurrences unresponsive to antibiotic therapy. Patients with allergies are at increased risk for both sinusitis and AOM; the same is true with craniofacial abnormalities seen in Down's syndrome or cleft palate. A history of sinus disease may suggest a source for referred otalgia. Immunocompromised patients and diabetics are at significant risk for developing necrotizing OE.

Medications

Patients taking medications containing acetaminophen or nonsteroidal antiinflammatory drugs (NSAIDs) for their ear pain may not exhibit a fever. Symptomatic patients with OM who are currently taking antibiotics may require a second-line agent due to penicillin-resistant *Streptococcus pneumoniae*. A careful review of a patient's medication list may identify immunocompromise or diabetes, both risk factors for necrotizing OE.

Social

There is a correlation between smoking and OM. Studies reveal a two- to four-fold increase in OM for children exposed to second-hand smoke. Smoking also increases the risk of sinusitis and head and neck cancers, well-known causes of referred otalgia. Children who attend group daycare are at a 2.5-fold increased risk for OM, whereas breastfed infants have 13% fewer ear infections. Like viruses and the common cold, ear infections are seasonal and tend to occur more frequently in the winter and early spring.

Physical examination

Although the history helps establish the problem, a careful physical examination usually makes the diagnosis.

General appearance

The importance of assessing the general appearance of a pediatric patient cannot be overemphasized. A toxic-appearing child with altered mental status or lethargy merits consideration of sepsis and meningitis, even if the examination reveals OM.

Vital signs

A fever can occur with AOM, but is seldom >40°C. Tachycardia is commonly due to fever and dehydration.

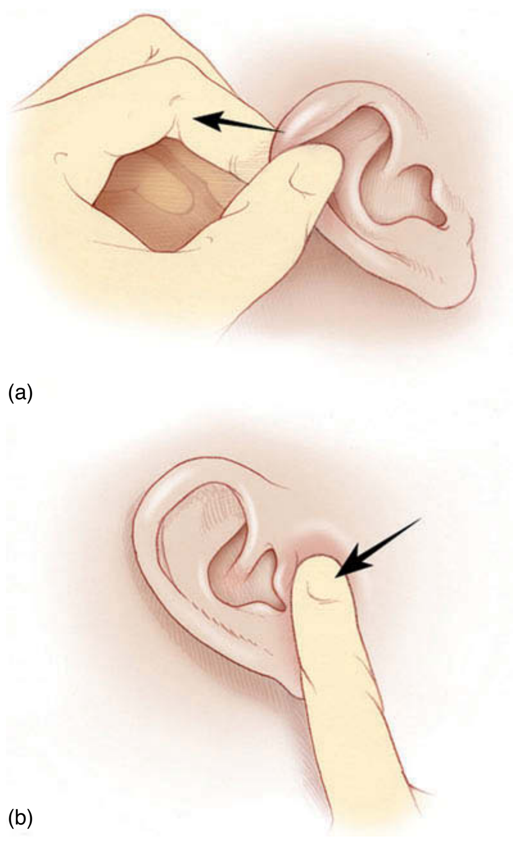

Figure 24.3
(a) Traction on auricle (b) pressure on tragus. © Chris Gralapp.

For every degree (in Celsius) of temperature elevation, expect an increase in the pulse of about 10 beats/minute.

Head, eyes, ears, nose and throat

A complete head, eye, ear, nose and throat (HEENT) examination is essential for the proper assessment of the patient with otalgia. Possible serious etiologies identified on examination include mastoiditis, ruptured TM with dislocation of the ossicles, retropharyngeal abscess, meningitis and necrotizing OE. A careful examination may also identify a head and neck malignancy, an important cause of referred otalgia.

Head and face

The sinuses should be examined to assess for possible sinusitis. The temporal artery should be palpated, as temporal arteritis is a treatable cause of referred otalgia. Periauricular lymphadenopathy may occur with scalp or neck infections. The submandibular, submaxillary and parotid glands should also be inspected and palpated. An infection, tumor or salivary stone affecting the parotid gland, which lies just anterior to the ear, could lead to otalgia.

Ears

External ear

The external ear and periauricular areas should be examined for signs of inflammation. Tenderness with manual traction of the auricle or applied pressure to the tragus suggests otitis externa (Figure 24.3). The presence of postauricular erythema, swelling and tenderness with protrusion of the auricle and loss of the postauricular crease suggests acute mastoiditis (Figure 24.4). In patients with necrotizing OE, the auricle may appear abnormal and grossly deformed.

Ear canal

Begin by selecting the correct speculum size to use with the otoscope. Pain on insertion of the speculum into the canal suggests OE (Figure 24.5). If the canal is occluded with cerumen, debris, or discharge, careful removal of the obstruction with an ear curette or irrigation may improve visibility. The EAC should be examined for signs of inflammation or the presence of a FB. Erythema or edema of the canal, with ear pain reproduced by pulling on the auricle or pressing on the tragus, signifies OE.

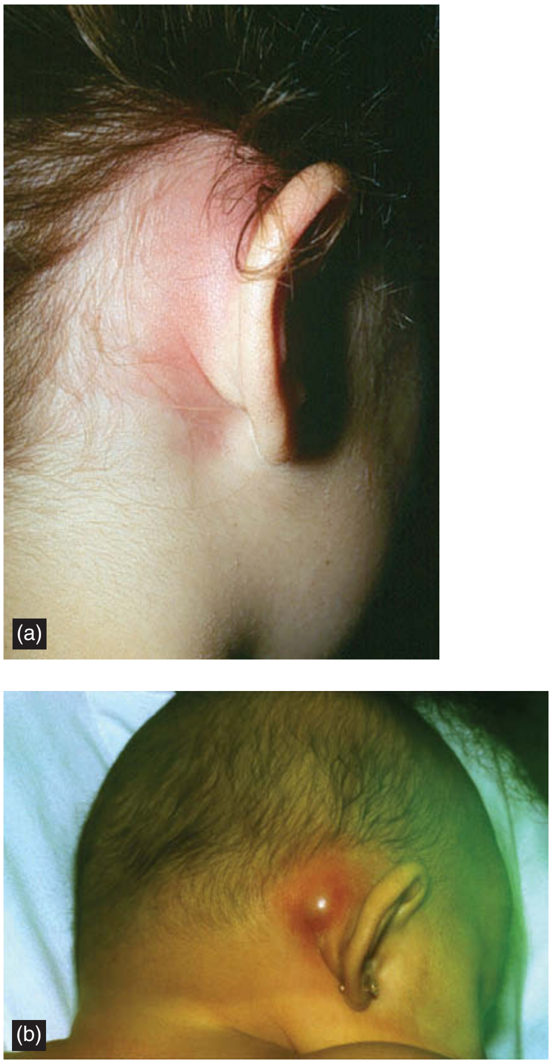

Figure 24.4
(a) Mastoiditis. Courtesy: Lawrence Stack, MD.
(b) Severe mastoiditis. Courtesy: Robert Jackler, MD.

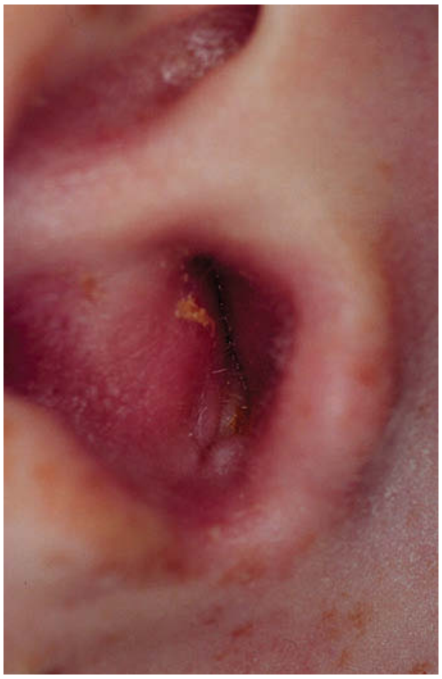

Figure 24.5
External otitis media. Note inflamed and erythematous external auditory canal. Courtesy: Lawrence Stack, MD.

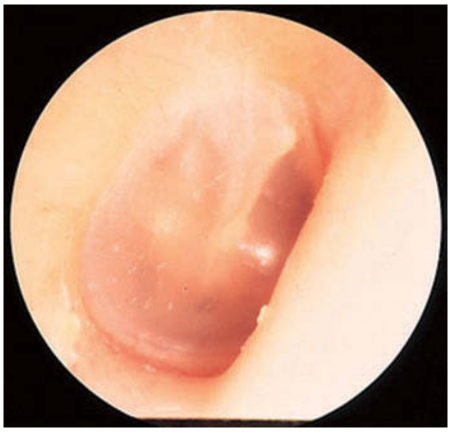

Figure 24.6
A normal tympanic membrane. The drum is thin and translucent, and the ossicles are readily visualized. It is neutrally positioned with no evidence of bulging or retraction. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

Tympanic membrane

Pulling the auricle posteriorly and superiorly straightens the EAC and facilitates visualization of the TM. The light reflex, color, translucency and bony landmarks of the TM should be noted. Comparison with the other ear may be helpful. The normal TM is shiny and translucent (Figure 24.6). Erythema may be present with OM, but also crying or fever. A retracted TM with prominence of the malleus may be found with OM with effusion. The development of bullae indicates bullous myringitis (Figure 24.7); vesicles suggest Ramsay Hunt syndrome (herpes zoster oticus) (Figure 24.8). The presence of a tympanostomy tube may lead to decreased TM mobility, altered landmarks, opacity and dullness, even in the absence of infection. A defect in the TM suggests perforation (Figure 24.9), whereas a white mass behind the TM may be a cholesteatoma.

Pneumatic otoscopy

Following visualization of the TM with the otoscope, air can be insufflated into the canal using a pneumatic bulb. The normal TM should be slightly mobile with

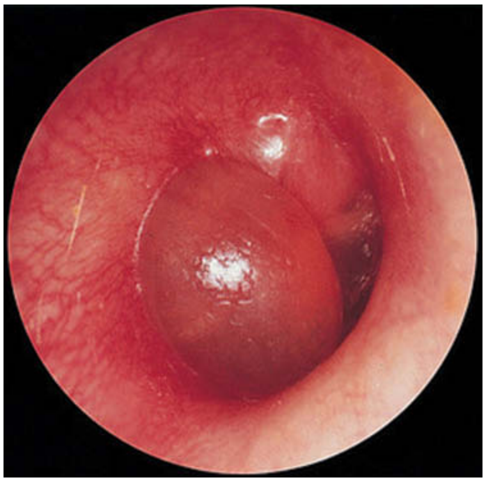

Figure 24.7
Acute otitis media with bullous myringitis. Otoscopy reveals an erythematous bullous lesion, obscuring much of the tympanic membrane. This phenomenon, called bullous myringitis, is caused by the usual pathogens of otitis media in childhood. The bullous lesion commonly ruptures spontaneously, providing immediate relief of pain. Reprinted Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

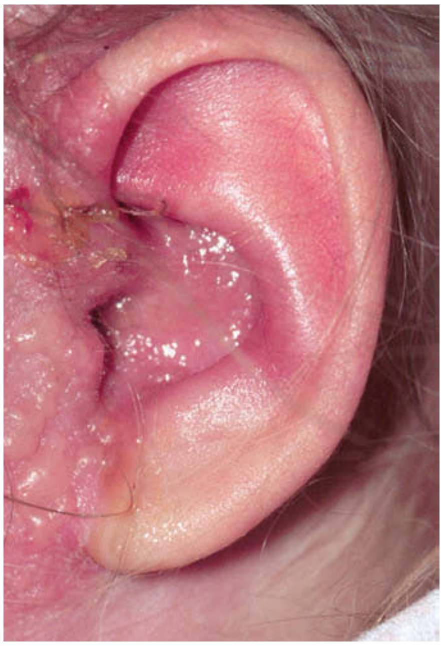

Figure 24.8
Herpes zoster oticus. Courtesy: Lawrence Stack, MD.

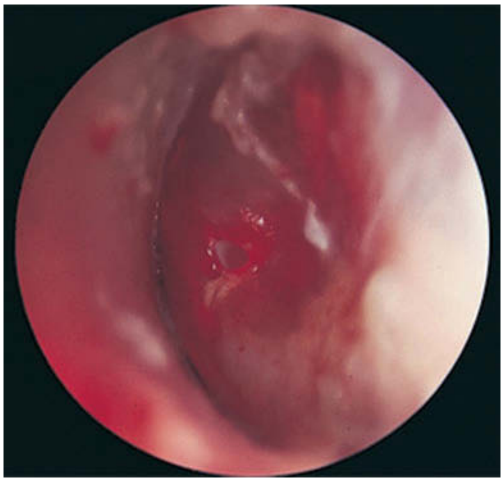

Figure 24.9
Acute otitis media with perforation. In this child, increased middle ear pressure with acute otitis resulted in perforation of the tympanic membrane. The drum is thickened, and the perforation is seen at the 3 o'clock position. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

insufflation. Diminished TM mobility highly suggests a middle ear effusion, though mobility may be reduced from middle ear adhesions, TM perforation or eustachian tube dysfunction. An immobile, bulging, erythematous eardrum that has lost its bony landmarks is predictive of AOM (Figure 24.10).

If the ear canal is too large to provide a good pneumatic seal, the patient is likely old enough to perform a modified Frenzel maneuver. In this procedure, the patient pinches the nose, gently blows without opening the mouth (cheeks puffing out is okay) and then swallows. Using this technique, the normal TM should move outward initially and then inward with swallowing.

Hearing

Hearing can be evaluated in a cooperative patient, but rarely in the young pediatric patient. Hearing can be measured grossly or assessed by the Weber and Rinne tests. The *Weber test* is performed by first placing the base of vibrating tuning fork on the middle of the forehead, equidistant from the ears. Hearing the tone louder in one ear suggests either conductive hearing loss in that ear or sensorineural hearing loss in the opposite ear. Plugging your ear with a finger (to simulate conductive hearing loss) and performing the test will demonstrate this finding. The *Rinne test* is performed by alternating placement of a vibrating tuning fork directly on the patient's mastoid process (bone conduction) and in front of the patient's ear (air conduction). Hearing the tone louder in front of the ear is a positive Rinne test, indicating normal hearing or sensorineural hearing loss. Hearing the tone louder with the tuning fork against the mastoid is a negative Rinne test, indicating conductive hearing loss in that ear.

Oropharynx

Since dental pain is the most common cause of referred otalgia, the gingiva and teeth should be carefully examined for dental caries, abscess, impacted molars or poorly fitting dentures. The gingiva should be palpated and the teeth percussed with a tongue-blade to assess for tenderness. Dental malocclusion resulting from TMJ dysfunction can cause referred ear pain from masticator muscle spasm. The TMJ should be assessed for clicking, popping and tenderness consistent with TMJ syndrome. Assess the oropharynx for pharyngitis, peritonsillar abscess, retropharyngeal abscess, or mass.

Neck

The neck should be evaluated for meningeal signs that must not be missed. Also assess the neck for musculoskeletal disorders, lymph node and thyroid enlargement, or other masses or tenderness. Movement of the neck may increase otalgia due to degenerative joint disease in the cervical spine.

Cranial nerves

Cranial nerve VII dysfunction and resulting facial paralysis may occur in patients with Ramsay Hunt syndrome, mastoiditis or necrotizing OE.

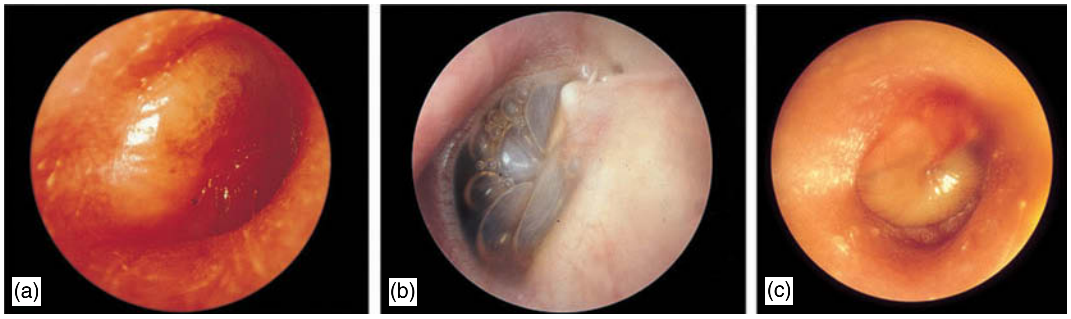

Figure 24.10
Acute otitis media. (a) An erythematous, opaque, bulging tympanic membrane with a reduced light reflex and partially obscuration of the landmarks. (b) The finding of both air and fluid-formed bubbles separated by gray-yellow menisci, combined with fever and otalgia, is consistent with acute infection (even though the drum is not injected). (c) The tympanic membrane is injected at the periphery and a yellow purulent effusion bulges outward from the inferior aspect. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

Differential diagnosis

Table 24.3 Differential diagnosis of ear pain

Diagnosis	Symptoms	Signs	Special work-up
Acute otitis media (Figure 24.10)	Ear pain; hearing loss; recent URI; pain worse at night; drainage from ear; children may have fever, fussiness, poor appetite, pulling at the affected ear	Temperature usually <40°C; TM findings: erythema, loss of light reflex, retraction or bulging, impaired mobility with pneumatic otoscopy; absence of pain with manipulation of pinna; drainage if TM ruptured	
Barotrauma	Pain onset during airplane descent or while scuba diving	TM hemorrhage; serous or hemorrhagic middle ear fluid	
Bullous myringitis (Figure 24.7)	Acute onset of ear pain, usually following a URI (like AOM); hearing loss; serosanguineous ear drainage	Serous or hemorrhagic blisters (bullae) on the TM; abnormal hearing	
Cholesteatoma	Unilateral hearing loss; malodorous ear drainage and pain	A cyst of desquamated epithelium or keratin that appears as a whitish area of the TM or polyp protruding through a TM defect	CT or MRI may reveal local bone erosion
Dental caries	Throbbing pain, sometimes localized; exacerbated by hot or cold foods, or lying supine	Poor dentition; pain with percussion of the affected tooth	
Foreign body	*Children*: ear pain; itching; discharge; foul odor *Adults*: Usually provide history of FB; may feel motion or buzzing with insect	Most FBs should be visible in the ear canal; reexamination after removal is important to exclude TM injury, or a retained or additional FB	
Herpes zoster oticus (Ramsay Hunt syndrome) (Figure 24.8)	Pain (may be out of proportion to physical findings); vesicular rash; facial paralysis; hearing loss, tinnitus and vertigo	Herpetiform vesicular eruption; vesicles may be seen on the pinna, EAC, TM, oral cavity, face and neck as far down as the shoulder; peripheral facial (CN VII) nerve palsy	
Necrotizing (malignant) otitis externa	Fever; severe pain; swelling of the pinna; purulent drainage; headache; facial paralysis; history of diabetes or immunocompromise; otorrhea unresponsive to topical measures	Classic finding is granulation tissue on the floor of the EAC at the bone–cartilage junction; tenderness of bony structures around the ear; cranial nerve involvement is a serious sign	ESR elevated; CT, technetium bone scan, Gallium scan
Mastoiditis (Figure 24.4)	Deep severe ear pain; headache; fever, chills, malaise; ear drainage; postauricular ear pain and swelling	Erythema, swelling and tenderness over the mastoid process; protrusion of the auricle; loss of the postauricular crease; fluctuance behind the ear; cranial nerve palsies	Radiographs may be negative; CT or MRI is more useful
Otitis externa (Figure 24.5)	Varies from itching to severe pain; serous to purulent discharge from the ear canal; systemic symptoms usually absent	Crusting and drainage in and around the EAC; erythema and edema of the EAC; manipulation of the auricle worsens the pain; ear canal may be swollen shut, leading to conductive hearing loss	
Pharyngitis, peritonsillar or retropharyngeal abscess	Sore throat; fever; absence of URI symptoms; headache; dysphonia; dysphagia; odynophagia; drooling	Fever; enlarged inflamed tonsils; exudates; trismus; displacement of the infected tonsil and deviation of the uvula with peritonsillar abscess; oropharyngeal fullness or tenderness with "rocking" the trachea with retropharyngeal abscess	A lateral neck radiograph or CT will demonstrate the retropharyngeal abscess
Sinusitis	Headache; facial pain; nasal congestion; purulent nasal drainage; persistent URI symptoms; maxillary toothache; pain exacerbated and relieved with changes in position	Tenderness with palpation of maxillary or frontal sinuses; mucosal erythema and edema; purulent nasal drainage; transillumination may reveal sinus opacification	Plain film accuracy best for maxillary sinusitis; diminished for other sinuses; CT is more sensitive and specific
Temporomandibular joint disorders	Intermittent pain or aural fullness; typically unilateral; worse at the start or end of the day; associated with chewing or bruxism (teeth grinding)	Otalgia with joint or masticatory muscle palpation; joint clicking and crepitus; trismus; excessive tooth wear	Radiographs are generally not helpful

AOM: acute otitis media; CN: cranial nerve; CT: computed tomography; EAC: external auditory canal; ESR: erythrocyte sedimentation rate; FB: foreign body; MRI: magnetic resonance imaging; TM: tympanic membrane; URI: upper respiratory infection.

Table 24.4 Uncommon causes of otalgia in patients with a normal ear examination

Angina, myocardial infarction
Aortic dissection
Bell's palsy
Carotidynia
Cervical adenopathy
Cricoarytenoid arthritis
Eagle's syndrome (elongation of the styloid process)
Gastroesophageal reflux
Head or neck malignancy
Myofascial pain, muscle spasm/inflammation
Neuralgia (e.g., trigeminal, glossopharyngeal)
Oral ulcers (involving the posterior third of the tongue, tonsils, or pharynx)
Psychogenic
Salivary gland disorders
Sinusitis
Temporal arteritis
Thyroiditis

Adapted from Ely JW, Hansen MR, Clark EC. Diagnosis of ear pain. *Am Fam Physician* 2008;77(5):621–8.

Diagnostic testing

Diagnostic testing is generally not indicated in the evaluation of otalgia. In patients with suspected necrotizing OE, cultures of the purulent discharge may aid pathogen identification and guide antibiotic therapy. Computed tomography (CT) and/or magnetic resonance imaging (MRI) are indicated to delineate the extent of infection. Patients without an obvious source for otalgia on physical examination may require CT of the head, face or neck to determine the etiology. Thyroid function tests or an erythrocyte sedimentation rate (ESR) may be helpful if thyroiditis or temporal arteritis is suspected. Panorex or dental X-rays may be helpful if there is concern for a mandibular or dental etiology.

General treatment principles

Pain relief

Although ibuprofen or acetaminophen may be adequate for some patients, others may require a short course of narcotic analgesics. A topical anesthetic like Auralgan (antipyrine/benzocaine) may be beneficial in patients with primary otalgia and an intact TM. Viscous lidocaine or Benadryl elixir can be gargled to anesthetize the throat and possibly localize referred otalgia to the oropharynx.

Otitis media

Antibiotics

Antibiotics are commonly used to treat AOM (Table 24.5). Since the advent of antibiotic therapy for AOM, the rate of complications and deaths from AOM has dropped dramatically. The first-line antimicrobial agent for AOM is typically amoxicillin, given that it has few side effects and reasonable efficacy. The use of higher dose amoxicillin (90 mg/kg/day) is more effective against drug-resistant pneumococcus; this therapy should be considered in children who have received antibiotics in the past 3 months, are less than 2 years of age, or are in daycare. In penicillin-allergic patients, consider using sulfa-containing agents (trimethoprim–sulfamethoxazole, erythromycin-sulfisoxazole) or macrolides (azithromycin or clarithromycin). Failure to improve after 72 hours of antibiotic therapy is considered a treatment failure. For patients who fail to respond to initial amoxicillin therapy, the Centers for Disease Control and Prevention (CDC) recommends amoxicillin/clavulanate, cefuroxime axetil or intramuscular ceftriaxone.

The need for antibiotic therapy in uncomplicated AOM is controversial. Over-prescribing antibiotics for AOM and the emergence of bacterial resistance to commonly used antibiotics has heightened the urgency to reduce unnecessary antibiotic use. Recent data suggest a lack of benefit to antibiotic treatment in children with uncomplicated OM (combined absence of fever, severe otalgia and vomiting). The practice of holding antibiotic therapy and "watchful waiting" is common in Europe and gaining acceptance in the United States. Reliable patients (>2 years of age) with adequate access to follow-up care are given a safety-net antibiotic prescription (SNAP) to be filled only if their symptoms do not resolve during the first 48 hours. Two-thirds of these patients recover without antibiotics, and their parents report a satisfaction score similar to those receiving antibiotics. During this period of observation, patients are managed with oral analgesics and topical otic anesthetic drops.

Other therapies

The use of antihistamines, decongestants or steroids provides no obvious benefit to patients with AOM.

Otitis media with tympanic membrane perforation

OM with TM perforation is treated similarly to AOM. Because the purulent discharge associated with perforation may cause an associated OE, patients are also commonly treated with topical eardrops containing a steroid–antibiotic suspension (not solution), such as Cortisporin-HC otic. Patients should avoid swimming and diving until the perforation has healed. During bathing, the placement of a petroleum jelly–impregnated cotton ball in the outer ear may prevent water entry into the EAC.

Table 24.5 Antibiotic choices for acute otitis media

First line	Dosage
Amoxicillin	Pediatrics: Age <3 months: 10–15 mg/kg PO BID for 10 days; Max: 30 mg/kg/day Age <3 months–2 years: 45 mg/kg PO BID for 10 days Age >2 years: 20–25 mg/kg PO BID for 10 days (Alt: 45 mg/kg PO BID for 5 days; Max: 875 mg/dose) Adults: 500–875 mg PO BID for 10 days
Trimethoprim-sulfamethoxazole	Pediatrics: Age >2 months: 0.5 mL susp/kg PO BID for 10 days; Max: 20 mL susp/dose Adults: 1 tablet PO BID for 7–14 days
Erythromycin–sulfisoxazole	Pediatrics: Age >2 months: 10–12 mg/kg (erythromycin component) PO QID for 7–14 days; Max: 2 g/day
Azithromycin	Pediatrics: Age ≥6 months: 10 mg/kg PO day 1 then 5 mg/kg/day PO QD days 2–5; Max: 500 mg/day Adults: 500 mg PO day 1; 250 mg PO QD days 2–5 (Alt: 500 mg PO QD for 3 days)
Clarithromycin	Pediatrics: 7.5 mg/kg PO BID for 10 days; Max: 1 g/day Adults: 500 mg PO bid for 7–14 days
Second line	**Dosage**
Amoxicillin–clavulanate	Dose based on amoxicillin component Pediatrics: Age <3 months: 15 mg/kg PO BID for 10 days; Max: 30 mg/kg/day Age 3 months–2 years: 40–45 mg/kg PO BID for 10 days; Max: 875 mg/dose Age >2 years: 20–25 mg/kg PO BID for 10 days; Max: 1800 mg/day Adults: 875 mg PO BID for 10 days
Cefuroxime axetil	Pediatrics: 10–15 mg/kg PO BID for 10 days; Max: 1 g/day Adults: 250–500 mg PO BID for 7–10 days
Ceftriaxone	Pediatrics: 50 mg/kg IM × 1–3 doses; Max: 1 g/dose

Alt: alternative; BID: twice a day; IM: intramuscular; Max: maximum; PO: per os; QD: daily; susp: suspension.

Table 24.6 Otic drops for otitis externa

Name	Components	Dosage
Vosol	Acetic acid and propylene glycol	5 gtts of 2% solution otic TID/QID for 7–10 days
Vosol HC	Acetic acid and hydrocortisone	3–5 gtts of 2% solution otic TID/QID for 7–10 days
Domeboro	Acetic acid and aluminum acetate	3–5 gtts of 2% solution otic every 2–4 hours for 7–10 days
Floxin	Ofloxacin	Age ≥1 year to 12 years: 5 gtts of 0.3% solution otic BID for 10–14 days
		Age >12 years: 10 gtts of 0.3% solution otic BID for 10–14 days
Cipro HC	Ciprofloxacin and hydrocortisone	Age ≥1 year: 3 gtts of 0.2% solution otic BID for 7 days
Cortisporin	Neomycin, hydrocortisone and polymyxin	3–4 gtts otic TID/QID for 7–10 days
Colimycin S	Neomycin	3–5 gtts otic TID/QID for 7–10 days
Otobiotic	Polymyxin and hydrocortisone	3–5 gtts otic TID/QID for 7–10 days

BID: twice a day; gtts: drops; HC: hydrocortisone; QID: four times a day; TID: three times a day.

Otitis externa

The treatment of OE begins by cleansing the external canal with gentle irrigation and suctioning. Though irrigation may be performed with tap water, saline or Burrow's solution, acetic acid has the added benefit of antifungal and antibacterial properties. Cleansing is followed by treatment with acetic acid or topical antibiotic–steroid otic drops (Table 24.6). In cases in which the canal is occluded by edema, the careful placement of a cotton wick facilitates the delivery of medicine throughout the entire ear canal. If the TM cannot be visualized and is at risk for perforation, a non-ototoxic, pH-balanced topical preparation should be used. Consider systemic antibiotics if cellulitis or systemic signs are present. Most patients experience a significant decrease in pain after 1 day of treatment.

Herpes zoster oticus

Treatment is aimed at shortening the outbreak and controlling symptoms. Systemic antivirals (e.g., acyclovir, famciclovir) and oral steroids are often used in combination. Though combination therapy may reduce the severity of postherpetic neuralgia, its efficacy for the reversal of facial nerve paralysis is controversial.

Mastoiditis

Treatment consists of broad-spectrum antibiotics and ENT consultation. Most cases resolve without the need for surgical drainage.

Foreign bodies

Approaches used for FB removal from the ear include irrigation, suction, direct instrumentation and cyanoacrylate (superglue). The preferred approach depends on the type of FB, available equipment and the physician's proficiency. Warm water irrigation is a simple, non-invasive approach for patients with an intact TM. Avoid irrigation if the suspected FB is made of organic material, as expansion of the object following contact with water may complicate its removal. For insects within the canal, mineral oil or viscous lidocaine is usually applied to immobilize and kill the insect. Lidocaine has the added benefit of anesthetizing the canal and TM, making extraction less painful. Following the removal of any FB, prophylactic antibiotics may be necessary to prevent OE. If removal of the FB cannot be achieved in the ED, then ear, nose and throat (ENT) referral within 24 hours is necessary.

Special patients

Immune compromised

Elderly diabetics and immunocompromised patients are at increased risk for necrotizing OE. More than 95% of cases are caused by *Pseudomonas aeruginosa*. Since the introduction of targeted systemic antibiotic therapy, mortality from necrotizing OE has decreased from 50% to 10%. Empiric treatment may be started with IV ciprofloxacin (400 mg IV q 8 hr) or an anti-pseudomonal beta-lactam agent if fluoroquinolone resistance is suspected. Antibiotics are usually administered for 4–8 weeks. Biopsy and surgical debridement may be required.

Disposition

Discharge

The vast majority of patients with otalgia are discharged home with an excellent prognosis. Patients who require subspecialty consultation include those suffering from necrotizing OE or mastoiditis, and those with worrisome complaints or findings such as severe pain, neurologic deficits, bloody discharge, hearing loss and vertigo.

Most cases of AOM and OE should improve within 48–72 hours. If symptoms persist, patients should be reevaluated for complications or possible treatment failure. Patients with an uncomplicated AOM should be reexamined in 2–3 weeks to ensure improvement of their middle ear effusion. Nearly 50% of patients have an effusion after 1 month, and 25% at 3 months. An asymptomatic patient with a middle ear effusion does not need additional antibiotic therapy. Follow-up with ENT should be arranged for patients with frequent ear infections, craniofacial abnormalities or multiple treatment failures. Patients with otalgia from an undetermined source need follow-up and further evaluation, as an occult malignancy may be responsible.

Pearls, pitfalls and myths

- The most common cause of otalgia is OM.
- Pain typically precedes otorrhea in OM; it accompanies the drainage in OE.
- Few patients with OM have very high temperatures (>40°C).
- An immobile, bulging red eardrum that has lost its bony landmarks is consistent with AOM.
- The presence of ear pain reproduced by pulling on the auricle or pressing on the tragus is likely caused by OE.
- Not all discharge from an ear canal is due to OE.
- Not all ear pain originates from the anatomic ear, especially if the ear examination is normal.
- Dental pain is the most common cause of referred otalgia.
- Not all patients with OM need immediate antibiotics.
- Placement of a wick may aid the treatment of a patient with canal occlusion from OE.
- OE in an immunocompromised host, especially with erythema and/or fever, should be considered necrotizing OE until proven otherwise.

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24B NOSEBLEED

Gregory H. Gilbert, MD

Scope of the problem

Nosebleeds (epistaxis) are frequently encountered in the emergency department (ED). There is typically a bimodal distribution, with patients commonly 2–10 or 50–80 years of age. Older patients tend to present with more significant epistaxis. Although a relatively small percentage (6–10%) of patients actually seek medical attention, epistaxis affects three out of every five persons in their lifetime, or 5–15% of the population per year. Surprisingly, one study showed that 66% of ED personnel lacked familiarity with basic first aid for epistaxis.

Anatomic essentials

Management of epistaxis requires a basic understanding of the nasal blood supply (Figure 24.11). The nasal circulation is derived from branches of the internal and external carotid arteries. The vascular nature of the nose is essential for its incredible heating and humidification requirements. To further facilitate this function, the vasculature runs just under the mucosa (not the squamous layer), leaving vessels more exposed and at risk for injury. These vessels spread out within this mucosal layer to form an anastomotic meshwork, artificially divided into anterior and posterior segments.

Anterior epistaxis typically originates from the anterior network of vessels located in the fleshy part of the nose, called Little's area or Kiesselbach's plexus. This collection of vessels receives blood from the following arteries: anterior ethmoidal, greater palatine, septal branch of the superior labial, and sphenopalatine. Ninety percent of nosebleeds originate from these vessels. Examining the nasal septum typically reveals the source of anterior nosebleeds.

Posterior epistaxis occurs in approximately 10% of nosebleeds. Posterior hemorrhage may not be directly visualized, and can be difficult to treat since it originates from a non-compressible part of the nose. A network of vessels called Woodruff's plexus, supplied by the sphenopalatine, posterior ethmoidal and nasopalatine arteries, is the most common site of posterior venous bleeds. Arterial bleeding is most likely from the sphenopalatine artery. Table 24.7 summarizes the findings of anterior and posterior epistaxis.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 24.8).

History

Management of the ABCs (airway, breathing, circulation) and hemorrhage control take precedence over obtaining a complete history. The following information should be obtained once the ABCs have been addressed and the bleeding controlled.

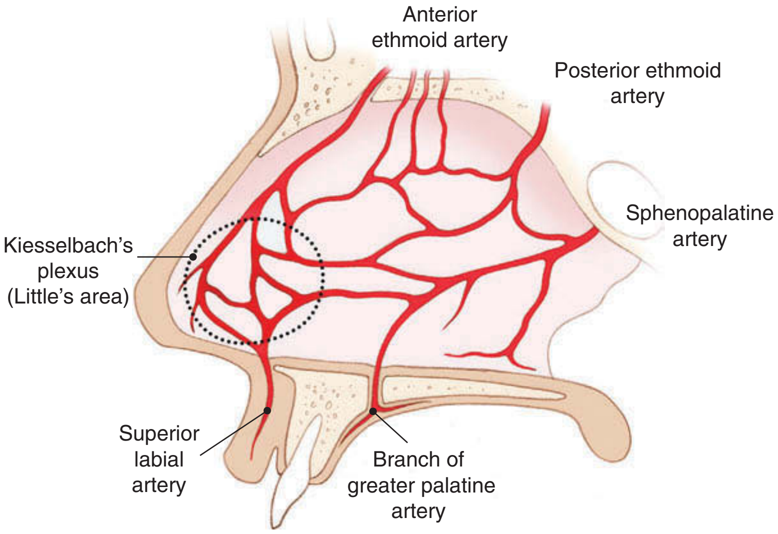

Figure 24.11
Blood supply of the nasal septum. © Chris Gralapp

Table 24.7 Historical and examination distinctions between anterior and posterior epistaxis

	Anterior	Posterior
History	• Presence of an inciting event • Recent use of agents that promote vasoconstriction of the nasal mucosa • Insertion of a foreign body • Recent cold, flu or allergies • Pediatric age group • Low-humidity environment • Unilateral	• Blood flowing down back of throat • Started in both nares (bilateral) • Seen more frequently in the elderly population • Tends to be more severe • Patient unable to control • History of angiofibroma in young males and squamous cell carcinoma in Asians
Physical examination	• Site of bleeding directly visualized • Bleeding from one nostril • Foreign body identified	• Cannot identify anterior site of bleeding • Bleeding from both nares • Blood continues to trickle down throat despite adequate anterior pack

Table 24.8 Epistaxis red flags

History	**Concerning diagnosis**
Trauma	Facial fracture, septal hematoma
Easy and recurrent bruising, bleeding	Coagulopathy or bleeding disorder
Recurrent unilateral epistaxis	Malignancy, foreign body
Child, institutionalized elderly, developmental delay	Foreign body
Examination finding	**Concerning diagnosis**
Abnormal vital signs, delayed capillary refill, pale, cool, diaphoretic	Significant blood loss
Ecchymoses, petechiae	Coagulopathy or bleeding disorder
Bleeding from both nares	Posterior source
Raccoon eyes, Battle sign, hemotympanum	Basilar skull fracture

How did the bleeding begin?

Attempt to determine what precipitated the epistaxis. Was it traumatic or spontaneous? The most common causes of epistaxis include nose picking (epistaxis digitorum), foreign bodies (FBs), and dry air and upper respiratory infections (URIs) during the winter months. Think of nasal FBs in children, institutionalized elderly and patients with developmental delay. Consult ear, nose and throat (ENT) specialists early if there has been recent nasal surgery. Traumatic epistaxis may be associated with other serious facial injuries.

Which side did it begin on?

Bleeding from one naris suggests an anterior nosebleed, whereas bleeding from both nares often indicates a posterior source. However, blood from a brisk anterior nosebleed can reflux into the unaffected side via the posterior choanae, simulating posterior epistaxis.

How severe has it been?

Try to determine how much blood loss has occurred. This is frequently embellished. Much more accurate predictors of blood loss are the patient's vital signs, symptoms and physical signs.

How did you attempt to stop the bleeding?

Patients may try direct pressure, oxymetazoline (Afrin) or pledgets before coming to the ED. Epistaxis that persists despite these efforts is generally more difficult to treat.

Have you ever had this before?

Recurrent nosebleeds should trigger questions about easy bruising or bleeding, which may suggest coagulopathy, and should raise concern for intranasal pathology, such as a deviated septum and primary or secondary tumors. Friable vessels and/or engorgement, which predisposes the patient to nosebleeds, may be seen with intranasal pathology, hypertension, congestive heart failure (CHF), pregnancy or frequent sneezing. Ask the patient about recent ED visits for bleeding and how the bleeding was treated.

Have you been coughing up or vomiting blood?

Massive epistaxis may initially be confused with hemoptysis or hematemesis. In cases of epistaxis without blood clearly dripping from the nose, visualization of bleeding from the posterior nasopharynx confirms the diagnosis.

Past medical

It is important to ask about underlying medical conditions such as bleeding disorders or blood dyscrasias (e.g., hemophilia, thrombocytopenia, von Willebrand's disease, or hereditary hemorrhagic telangiectasia [Osler–Weber–Rendu disease]). Ask about easy bruising or bleeding, human immunodeficiency virus (HIV), liver or kidney disease and cancer, as these patients may have thrombocytopenia, platelet disorders, splenomegaly, or medications predisposing them to bleeding.

Past surgical

Ask about prior nasal surgery, which might predispose a patient to bleeding or explain the source.

Medications

It is important to ask about medications that might promote bleeding or complicate therapy. These include platelet inhibitors like aspirin, dipyridamole and nonsteroidal antiinflammatory drugs (NSAIDs); alternative medications like garlic, ginkgo or ginseng; or, anticoagulants like warfarin, enoxaparin and heparin. Intranasal corticosteroids may induce bleeding, but spraying laterally may reduce the likelihood. Ask about alcohol abuse or cocaine insufflation, as both may contribute to or exacerbate bleeding.

Physical examination

First determine whether the patient is stable or ill. Then, a focused physical examination should look at the following items:

General appearance and skin

Pallor and diaphoresis are ominous findings. The patient has either lost a large amount of blood, or does not like the sight of it. In either case, placing the patient supine on a gurney will prevent serious injury should the patient lose consciousness. Ecchymosis, petechiae and spider angiomas suggest underlying bleeding disorders. Delayed capillary refill suggests significant blood loss.

Vital signs

It is important to check the blood pressure and pulse rate. Hypotension, tachycardia, or symptomatic blood pressure changes from supine to standing suggest significant blood loss and should prompt intravenous (IV) access and administration of fluid. Blood should be drawn for laboratory studies. Although hypertension has never been shown to cause epistaxis, it can worsen bleeding when present.

Head, eyes, ears, nose and throat

A complete head, eye, ear, nose and throat (HEENT) examination should be performed in all patients with epistaxis. Look for signs of basilar skull fracture (i.e., raccoon eyes, Battle sign, hemotympanum or cerebrospinal fluid [CSF] rhinorrhea), as devices introduced through the nares (e.g., intranasal balloon device) may perforate the cribriform plate and inadvertently enter the cranium. Assess for tenderness and stability of the maxilla and other facial bones to help identify Le Fort or orbital wall fractures.

Nose

The key to successful examination of the nose is preparation. Prior to the nasal examination, assemble the proper items for examination, stabilization and treatment of epistaxis (Table 24.9).

First, have the patient blow his or her nose to clear the nasopharynx, even if the bleeding has stopped. Then, place the patient in the sniffing position. A thorough nasal examination should be performed. A nasal speculum assists with this task (Figure 24.12); prior to insertion, orient the nasal speculum so that one blade moves superiorly and the other inferiorly. Attempt to locate the source of bleeding. Ninety percent of nosebleeds have a visible source, and careful examination of the nasal septum will reveal a friable vessel. If trauma preceded the bleeding, then examine the nasal septum (to exclude a septal hematoma) and the facial bones (to exclude a fracture). An untreated septal hematoma can lead to an abscess or avascular necrosis of the septum. If the bleeding source is not visible on nasal examination, it may be from the posterior circulation. Other signs of posterior epistaxis include bleeding from both nares and hemorrhage into the posterior pharynx. Controlling bleeding in these patients may be extremely difficult. A thorough nasal examination should identify nasal pathology, such as FBs, perforated or deviated septums, nasal masses, or engorged vessels.

Table 24.9 Suggested equipment for the evaluation and treatment of epistaxis

Examination	Stabilization	Treatment
Protective eyewear	Bayonet forceps	Silver nitrate sticks
Two gowns	Pledgets	Electrocautery
Nasal speculum	4% topical cocaine *or* 1% lidocaine with epinephrine *and* 4% topical lidocaine	Gelfoam, Surgicel
ENT headlamp or mirror	Afrin spray	Bacitracin
Yankauer and Frazier-tip suction		1/2” × 6” petroleum gauze
Emesis basin		16-Fr Foley or intranasal balloon
Balloon		Rapid Rhino or Merocel sponge
Kleenex or gauze		FloSeal, Avitene, Surgiflo

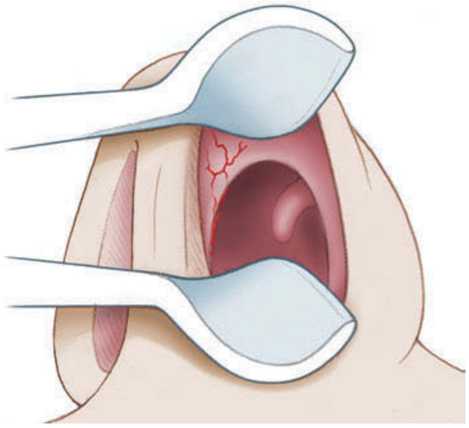

Figure 24.12
Use of a nasal speculum to examine the nose. © Chris Gralapp

Differential diagnosis

Table 24.10 provides numerous etiologies of epistaxis.

Diagnostic testing

Laboratory studies

Routine laboratory studies are not necessary in most cases of epistaxis.

Complete blood count (CBC), Type and Screen

If the patient complains of significant or prolonged blood loss, easy bruising, recurrent epistaxis, a history of platelet disorders, cancer or recent chemotherapy, a CBC should be checked. If the blood loss was significant enough to warrant a CBC, then a *type and screen* should also be considered.

Prothrombin time (PT), Partial thromboplastin time (PTT), International Normalized Ratio (INR)

These tests are helpful in anticoagulated patients and those with liver disease. Consider coagulopathy if epistaxis persists despite direct pressure, cautery and nasal packing.

Table 24.10 Etiologies of epistaxis

Traumatic or mechanical
Epistaxis digitorum (nose picking)
Congenital or acquired nasal defects
Direct blow (with or without fracture)
FB (demented, psychiatric, intentional, children)
Desiccation (low humidity household, winter, supplemental oxygen)
Infections/inflammation (allergic or atrophic rhinitis, URI, diphtheria, sinusitis, nasopharyngitis, nasopharyngeal mucormycosis, chlamydial rhinitis neonatorum)
Local irritants (cocaine abuse, chemical/environmental irritants, OTC nasal sprays)
Iatrogenic (nasal surgery, NG tube, nasopharyngeal airway, septal perforation, cautery)
Barotrauma (abrupt changes in pressure – diving or rapid altitude gain)
Venous congestion (CHF, mitral stenosis, sneezing, coughing, nose blowing, Valsalva, pregnancy)
Tumors (benign or malignant)
Primary (nasal polyps, juvenile angiofibroma, squamous cell, paranasal sinus tumors, metastatic)
Secondary (thrombocytopenia due to leukemia, lymphoma or chemotherapy)
Predisposing factors
Systemic toxins (rodenticide, plant poisoning, glycosides, coumarin, heavy metals)
Medications (salicylates, NSAIDs, warfarin, heparin, dipyridamole, ticlopidine, thrombolytics, garlic, ginkgo, ginseng)
Congenital (hemophilia A and B, von Willebrand's disease, inherited platelet disorders)
Hereditary hemorrhagic telangiectasia (Osler–Weber–Rendu syndrome)
Disease-mediated
Hypertension and atherosclerotic cardiovascular disease
Blood dyscrasias (polycythemia vera, granulocytosis)
Thrombocytopenia (drug-induced, chemotherapy, ITP, malignancy)
Vitamin deficiency (scurvy, folic acid, vitamin K)
Hepatic disease (alcoholism, hepatitis)
Renal disease (chronic nephritis, uremia, diabetes mellitus)
Disseminated intravascular coagulation, hypoprothrombinemia, hypofibrinogenemia
Other
Idiopathic (habitual, familial)
Migraine headache
Internal carotid artery aneurysm
Blood transfusion reactions
Endometriosis

CHF: congestive heart failure; FB: foreign body; ITP: idiopathic thrombocytopenic purpura; NG: nasogastric; NSAIDs: nonsteroidal antiinflammatory drugs; OTC: over-the-counter; URI: upper respiratory infection.

Bleeding time

This easy yet rarely performed test determines if a patient is able to clot normally. An abnormal bleeding time can occur even if the INR is normal and might explain difficulties controlling epistaxis.

Radiologic studies

Routine imaging is usually not necessary in most cases of epistaxis. If the patient presents with significant facial trauma, computed tomography (CT) of the facial bones may identify or exclude fractures.

General treatment principles

The ABCs are always the first priority. If severe bleeding compromises the airway and breathing, intubate the patient and then address the epistaxis. Although the majority of patients with nosebleeds are hemodynamically stable, those with significant blood loss should be placed on a gurney. Ask the patient to expel all clots from his nose to improve visualization (i.e., location and amount of bleeding) and to halt fibrinolysis of an existing clot, which can lead to continued bleeding.

Direct pressure

Direct pressure is the first step in controlling epistaxis. The fleshy part of the nose is squeezed between the thumb and a flexed index finger (Figure 24.13). It should look like the patient's nose is in a fist. Allow the patient to chose a comfortable head position, provided blood is not being aspirated or swallowed. With active bleeding, a forward sniffing position is preferred to allow expectoration, if necessary. Have the patient hold firm pressure for 10–15 minutes. During this time, gather the supplies mentioned previously (Table 24.9). Dress in appropriate attire, adhering to universal precautions; this includes a gown, eyewear, a facemask or shield, and gloves. A headlamp will help with visualization, and a basin should be placed below the patient's chin. Set up a suction device with a Yankauer or Frazier tip.

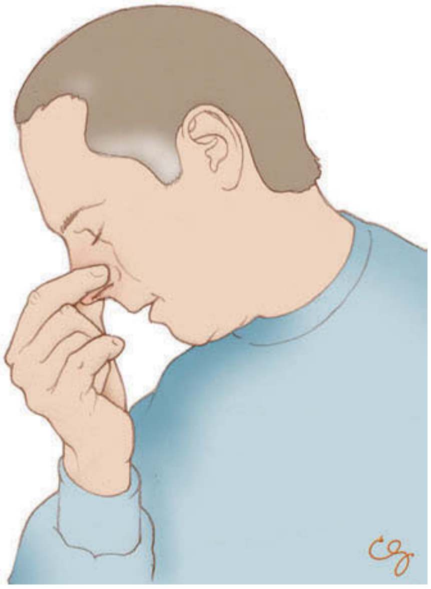

Figure 24.13
Direct pressure. © Chris Gralapp

If bleeding persists after withdrawing direct pressure, pledgets or sprays may arrest the bleeding. The pledget should first be soaked in lidocaine with epinephrine solution or cocaine, and then inserted into the nasal passage (Table 24.11). Be sure to reapply direct pressure. The use of vasoconstrictive agents without an anesthetic is inadequate, as interventions to halt bleeding will irritate the exquisitely sensitive nasal mucosa. Though oxymetazoline or phenylephrine spray may also be used, pledgets allow the nasal mucosa to absorb more agent than spraying alone. Heavy bleeding that persists after three attempts with direct pressure and pledget insertion requires nasal packing. However, if bleeding has slowed to an ooze or stopped, inspect the nasal cavity. The effects of these agents are temporary, so bleeding is likely to recur. Using the nasal speculum, headlamp and suction device, evacuate clots and attempt to identify a bleeding source.

Table 24.11 Vasoconstrictive and anesthetic agents used for epistaxis

Afrin or phenylephrine mixed with 4% lidocaine (toxic dose of lidocaine is 4 mg/kg)
Epinephrine 0.25 mL of 1:1,000 concentration mixed with 20 mL of 4% lidocaine
Cocaine (4%) (do not exceed 2–3 mg/kg in adults)

Note: 4% is equal to 40 mg/mL.

Cautery

Silver nitrate sticks are commonly used for cautery of the nasal mucosa in the absence of active bleeding. The sticks are applied to the vessel or friable mucosa for up to 10 seconds. Cauterize using a rolling motion peripherally to centrally, and superior to inferior, to avoid rendering the stick ineffective with blood. Beware of causing septal perforation with prolonged, bilateral, or overzealous use. Septal necrosis and perforation can also occur with multiple applications to both sides of the septum, so use with great care. Cautery has little value in trauma patients, and should not be attempted if the etiology of epistaxis is thought to be cancerous. Thermal or electrocautery is extremely difficult and fraught with iatrogenic injury; these modalities are best left to the ENT specialist. Laser cauterization has a limited role and is not used in acute epistaxis.

Packing

When medical management or cautery fails, packing is the next appropriate step. Packing may be classified as absorbable or non-absorbable, and anterior or posterior.

Anterior

Anterior packing with absorbable material tends to be better tolerated, is less painful, provides additional protection for the mucosa, and does not require removal or prophylactic antibiotics. Common absorbable materials include oxidized cellulose (Surgicel) and gelatin foam (Gelfoam). Other products (FloSeal, Avitene, Surgiflo) combine thrombin with gelatin to produce a slurry that fills the irregular contours of the nasal cavity, when squirted from a syringe. Though these products are expensive and may not effectively control brisk arterial bleeding, one study suggests they are superior to traditional non-absorbable packing materials.

Anterior packing with non-absorbable materials was traditionally performed with Vaseline gauze and forceps. The packing was placed along the floor of the nasal cavity, front to back, back to front, until the entire cavity was filled. This is a difficult, time-consuming process, but when done correctly, provides excellent hemostasis. Although very few patients (16/100,000) develop toxic shock syndrome, ENT specialists still recommend antibiotic prophylaxis for patients with non-absorbable nasal packing (Table 24.12).

Common non-absorbable devices include nasal tampons (Figure 24.14) or intranasal balloons. Tampons (Merocel) are typically lubricated with antibacterial ointment prior to insertion, although this routine practice has not been studied. Upon contact with fluid, the tampon softens and expands. Large nares may require two tampons. Another technique employs phenylephrine spray to induce tampon expansion by spraying either side. Intranasal balloon catheters (Rapid Rhino) should be inserted with water-based lubricants, as petroleum products can cause degradation of the balloon and possible rupture. There are two types of balloons: anterior and anterior/posterior. Tamponade should begin with the anterior balloon since placement of the anterior/posterior balloon often mandates hospital admission. Following packing, the oropharynx is inspected for continued bleeding, which implies either inadequate anterior packing or a posterior source.

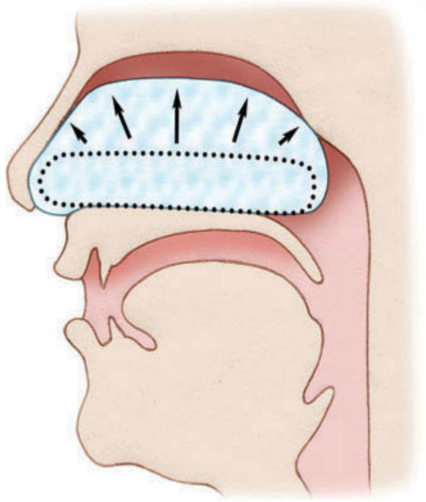

Figure 24.14
Merocel nasal tampons. © Chris Gralapp

Posterior

If anterior packing fails or bleeding persists, the source of bleeding is likely posterior. Traditionally, posterior packing was created using silk sutures attached to rolled gauze. This was drawn up through the mouth into the posterior pharynx and then bilateral anterior packs were placed. Faster, more comfortable posterior packing approaches include using a 12–16 French Foley catheter with a 30-mL balloon (the distal tip should be cut off for patient comfort) or the anterior/posterior nasal balloon (Figure 24.15). Both are inserted through the naris into the posterior nasal cavity. Following insertion, inflate the balloon about halfway with air or saline, and apply traction, securing it against the posterior aspect of the middle turbinate. Complete inflation of the balloon should stop the bleeding from the posterior pharynx. Then, the anterior balloon may be filled or an anterior pack placed using one of the aforementioned methods. The balloon should be checked for integrity prior to insertion, and care should be taken not to overfill the balloon, as pressure necrosis or septal damage may occur. Posterior packs may induce suppression of the respiratory drive and hypoxia. Due to the considerable morbidity and mortality and only a 70% success rate associated with posterior packs, early ENT consultation and admission are recommended. An ENT specialist can inject lidocaine with epinephrine via a transpalatal approach to

Table 24.12 Prophylactic antibiotic options for epistaxis with packing

Antibiotic 5-day course	Adult dose	Pediatric dose
First-line		
Cephalexin	250–500 mg PO QID	6.25–12.5 mg/kg PO QID; Max: 4000 mg/day
Augmentin	500–875 mg PO BID	15–20 mg/kg PO BID; Max: 1800 mg/day
Penicillin allergy		
Clindamycin	150–450 mg PO QID	3–10 mg/kg PO TID; max: 1800 mg/day
Trimethoprim/sulfamethoxazole	1 DS (160 mg TMP) tablet PO BID	>2 months: 4–5 mg/kg PO BID; Max: TMP 320 mg/day

BID: twice a day; DS: double strength; Max: maximum; PO: per os; QID: four times a day; susp: suspension; TID: three times a day; TMP: trimethoprim.

vasoconstrict the sphenopalatine artery. They can also perform endoscopic electric cauterization, embolization and surgical arterial ligation. Transnasal endoscopic sphenopalatine artery ligation (TESPAL) has an 87–100% success rate and does not require an inpatient stay. As for patients with posterior packing or significant anterior packing, antibiotic prophylaxis is recommended to reduce the risk of sinusitis and toxic shock syndrome (Table 24.12). Furthermore, appropriate analgesia should be considered for posterior packs, as these are often painful. Posterior packing can lead to alar, columellar, or palatal necrosis.

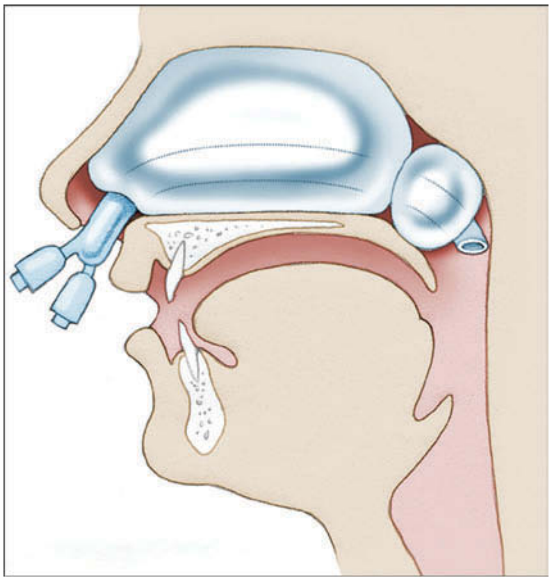

Figure 24.15
Anterior/posterior nasal balloon. © Chris Gralapp

Special patients

Elderly

Geriatric patients tend to have multiple medical problems; careful review of the patient's medical history and medications may reveal the cause of epistaxis. Liver or renal disease, CHF, hypertension, cancer, other coagulopathies, or the use of warfarin or aspirin may play a role in the patient's epistaxis and make it difficult to control.

Pediatric

Most pediatric patients require only direct pressure to control the bleeding. If packing is required, ENT consultation is recommended, as pediatric patients tend to be uncooperative and may need sedation in the operating room. It is especially important to consider the possibility of nasal FB as the cause of bleeding in this population.

Immune compromised

Patients with HIV may have thrombocytopenia, platelet disorders, splenomegaly or drug use predisposing them to bleeding. Universal precautions including goggles and a face shield are extremely important when caring for all patients with epistaxis, as blood can easily spray onto the practitioner.

Disposition

Ear, nose and throat consultation

Five to ten percent of ED cases of epistaxis require ENT consultation or admission, particularly if the clinician is unable to control the bleeding. Patients with posterior packing should be admitted to ENT due to increased morbidity and mortality as a result of hypoxia, apnea, and dysrhythmias. Although apnea and hypoxia have previously been ascribed to the nasopulmonary ("diving") reflex, they more likely result from obstructive sleep apnea. Pediatric patients who are uncooperative also require ENT consultation.

Discharge

The majority of patients presenting with epistaxis can be safely discharged. All patients with recurrent epistaxis or non-absorbable packing need ENT referral for removal of the packing and further evaluation of possible intranasal pathology. Although controversial, patients with high-risk (posterior or significant anterior) nasal packing should be placed on prophylactic antibiotics to prevent sinusitis and reduce the risk of toxic shock syndrome. Packs are generally left in for 2 to 3 days based on physician preference, response of the patient, risk factors, state of coagulopathy, and severity of initial bleeding. If bleeding recurs prior to the ENT evaluation, the patient should attempt direct pressure two or three times for 10–15 minutes each. Patients with bleeding around nasal packing should return to the ED. In dry or cold months, patients without packing may benefit from saline spray, humidifiers and petroleum jelly applied intranasally once or twice a day. A saline spray is recommended 24–48 hours after instillation of absorbable packing to promote degradation. Instruct patients to avoid blowing or picking their noses, straining, or participating in strenuous activities. They should sneeze with their mouths open. Patients should avoid aspirin and NSAIDs for 3–4 days. Educating patients about prevention and management of recurrences reduces morbidity, mortality and prevents unnecessary future visits.

Pearls, pitfalls and myths

- Direct pressure should be firmly held over the fleshy part of the nose, not the bridge, for at least 10–15 minutes.
- Ice on the bridge of a nose or in the mouth may help slow bleeding.
- If the patient can sit upright, the head should be maintained above the heart.
- Preparation is key to the successful treatment of epistaxis.

- Do not waste time inserting an anterior pack if a posterior source of epistaxis is suspected. Consult ENT early for these patients.
- Record the amount of fluid used to fill both the anterior and posterior intranasal balloons.
- Consider admitting all patients with posterior packing, as they may may become hypoxic and hypercarbic due to hypoventilation. They may also develop bradycardia or dysrhythmias.
- Patients with high-risk nasal packing should be started on antibiotics, due to an increased risk for sinusitis and toxic shock syndrome.
- Patients who start bleeding around anterior packing need to be reevaluated, repacked, and possibly have ENT consultation.
- Complications from anterior and posterior packs include ulcerations, pressure necrosis, septal perforation, sinusitis, synechiae, hypoxemia and dysrhythmias.
- Consider nasal FB in young children presenting with epistaxis.

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24C THROAT PAIN

Alice Chiao, MD and Michelle Huston, MD

Scope of the problem

Throat pain is one of the most common complaints seen by all health care providers, with pharyngitis being the most common cause of throat pain. Viruses are the most common cause of pharyngitis, accounting for approximately 40% of cases. Group A beta-hemolytic streptococcus (GABHS) accounts for up to 40% of pediatric pharyngitis, but less than 15% of adult cases.

Although most patients presenting with sore throat have a mild, self-limiting illness, throat pain may be the sign of a life-threatening condition. Recognizing and treating both common and serious causes of sore throat is an essential skill for emergency providers.

Anatomic essentials

The throat (pharynx) is divided into three areas extending from the base of the skull to the inlet of the esophagus (Figure 24.16): the *nasopharynx* (soft palate and posterior nasal cavity), *oropharynx* (posterior to the mouth down to the upper edge of the epiglottis) and *hypopharynx* (between the epiglottis and the cricoid cartilage). Sore throat may be caused by a disorder affecting any of these areas, as well as processes affecting the ears, tongue, esophagus and upper thorax. Throat pain is commonly associated with ear pain because cranial nerves IX and X provide sensory innervation to the pharynx and larynx as well as the ear.

Deep space infections of the lower face and neck may also cause sore throat. A polymicrobial cellulitis of the submandibular spaces of the head and neck causes Ludwig's angina. There are seven spaces in the neck that may also become infected: the peritonsillar, parapharyngeal, retropharyngeal, prevertebral, pretracheal, carotid and the "danger" space (between the prevertebral and retropharyngeal spaces). The supraglottic structures become infected in epiglottitis.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 24.13).

History

Life-threatening illnesses should be ruled out in all cases of sore throat. These include deep space infections, epiglottitis, foreign bodies (FBs), laryngeal trauma and

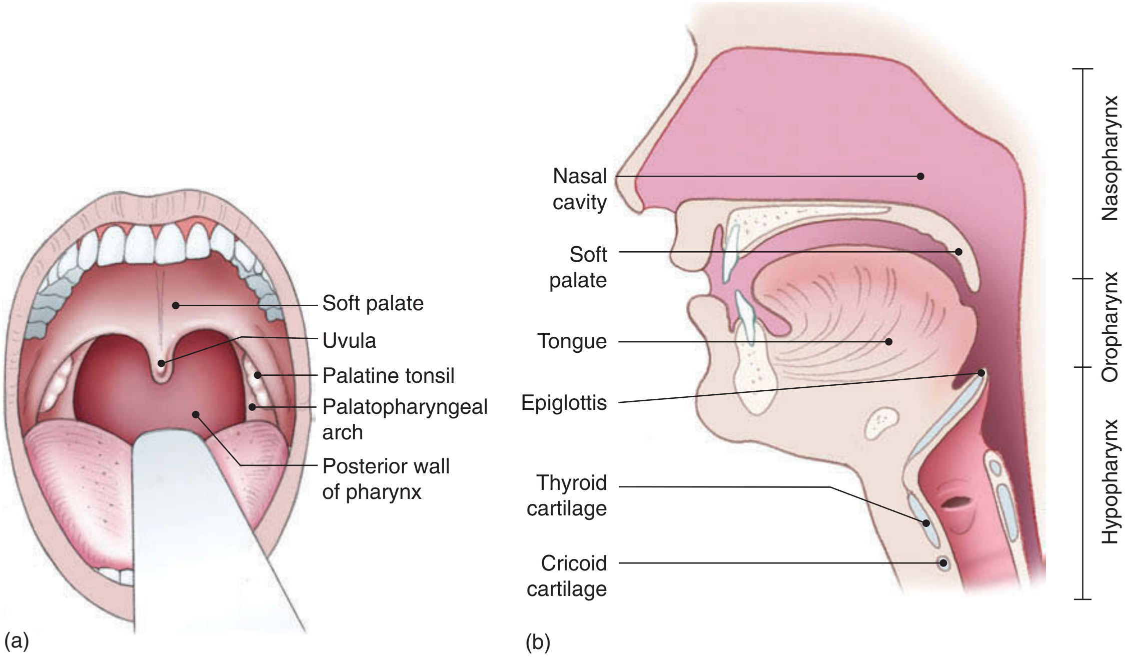

Figure 24.16
(a) Anatomy of the pharynx and (b) sagittal anatomy. © Chris Gralapp

Table 24.13 Throat pain red flags

History	Concerning diagnosis
Onset after age 50 years	Malignancy
Choking or difficulty swallowing	Mass, foreign body, airway compromise
Difficulty breathing	Mass, foreign body, abscess
Neck pain	Deep space infection, meningeal spread
Headache	Meningitis, mastoiditis, sinus thrombosis
Immunocompromised, chronic steroids	Infection
Tobacco/alcohol use, weight loss	Malignancy
Post-tonsillectomy patients	Post-procedural bleeding
Examination finding	**Concerning diagnosis**
Fever	Infection, epiglottitis
Stridor/respiratory distress	Airway compromise due to foreign body, mass, burns
Drooling	Potential airway obstruction
Muffled, “hot potato” voice	Peritonsillar abscess
Visible bulge in pharynx	Mass, peritonsillar abscess, malignancy
Crepitus	Infection, pneumothorax, laryngeal trauma
Pseudomembrane	Diphtheria, *Arcanobacterium hemolyticum*
Trismus, induration neck or submandibular region	Deep space infection, Ludwig’s angina
Rash	Scarlet fever, gonococcal, meningococcal infection
Splenomegaly, hepatomegaly	Epstein-Barr virus infection
High fever, toxic appearance	Epiglottitis, bacterial tracheitis

burns. All of these entities may cause sudden airway obstruction with asphyxia. Additionally, deep space infections may lead to carotid artery and jugular vein thrombosis or hemorrhage, mediastinitis, pericarditis, empyema and sepsis.

Where is the pain located?

Lateralization of symptoms is suggestive of peritonsillitis, cellulitis, or abscess of the peritonsillar space. Patients with retained FBs are often able to describe the exact location of the pain.

How long has the pain been present?

If the pain has been present less than 72 hours, it is unlikely that a deep space infection is present. A sore throat for more than 2 weeks in a patient over 40 years of age should warrant consideration and investigation for cancer.

How did the symptoms begin?

Sudden onset of pain during eating suggests a FB. The presence of a FB can be easily missed in children and those with mental illness or swallowing dysfunction, if these patients and their companions are not carefully questioned. Similarly, trauma is not always mentioned without the examiner specifically asking.

Have you had the pain before?

Many patients with GABHS pharyngitis have experienced their symptoms with previous episodes.

What is character of the pain (quality and severity)?

Throat pain ranges from a sensation of scratchiness to severe pain. The gradual onset of a scratchy sensation evolving into pain is consistent with a viral infection.

Any close contacts with similar symptoms?

A positive answer to this question supports either a viral or bacterial source of infection.

Are there any measures that make the discomfort worse or better?

Pain with swallowing (odynophagia), especially hot or acidic fluids, is seen with many causes of throat pain, including pharyngitis and cancer.

Associated symptoms

Potential airway obstruction

It is important to ask about swallowing function, drooling, voice change, trouble breathing and apprehension. Dysphagia (difficulty swallowing) and the inability to swallow must be distinguished from odynophagia (painful swallowing), which is present in almost all patients with sore throat. Drooling may represent the inability to swallow. Voice changes may range from mild hoarseness to a muffled voice to complete aphonia. A muffled or "hot potato" voice is often heard with deep space neck infections, epiglottitis, FB and trauma, as well as with severe pharyngitis. Dyspnea, tachypnea, "noisy breathing" and apprehension have been reported by patients with impending airway obstruction.

Trismus

Limitation of mouth opening is caused by inflammation of the muscles of mastication. Conditions that may cause trismus include peritonsillar abscess, deep space infections of the neck and Ludwig's angina.

Fever

Fever is associated with both viral and bacterial pharyngitis, epiglottitis and deep space infections. Patients with bacterial infections, including those with epiglottitis, typically have high fevers.

Upper respiratory infection

Symptoms of upper respiratory infection (URI) include rhinorrhea, nasal congestion, cough and coryza. The combination of these symptoms and sore throat is most commonly associated with viral infections.

Ear pain (otalgia)

Pain radiating to the ears is common with pharyngitis and other causes of throat pain, but does not point to a specific etiology of sore throat.

Tooth pain (odontalgia)

Dental pathology and procedures may precede the development of a parapharyngeal abscess, Ludwig's angina, or Vincent's angina.

Headache

Headache may be associated with GABHS pharyngitis. Although rare, the deep neck infections and GABHS pharyngitis may spread and cause mastoiditis, cavernous sinus thrombosis or meningitis, which are all serious etiologies of headache.

Neck pain

Posterior or lateral neck pain in the presence of sore throat should raise suspicion for deep space abscess and/or meningeal spread of infection. Anterior neck pain should raise suspicion for epiglottitis or laryngeal injury.

Abdominal pain

Abdominal pain may be associated with GABHS pharyngitis, particularly in children.

Rash

Scarlet fever, which is caused by GABHS, is diagnosed by the presence of a distinctive, diffuse sand-papery red rash. Gonococcal or meningococcal pharyngitis may lead to a disseminated rash.

Vaginal or penile discharge

Ask about a history of sexually transmitted infections (STIs) and orogenital sex in sexually active patients with sore throat. These patients are at risk for gonococcal and/or *Chlamydia trachomatis* pharyngitis.

Past medical

Pay special attention to systemic disorders (the immunocompromised host is at risk for opportunistic infections), medications, history of allergic reactions, tobacco and alcohol use (increased risk of cancer), and vaccination history. Ask about a prior history of "strep throat" diagnosed by laboratory measures, and any history of rheumatic fever or rheumatic heart disease. GABHS infection tends to recur in these patients. Surgical history, particularly previous head or neck surgery, recent intubation, gastric tube placement, or recent dental procedure may predispose to laryngeal trauma or retropharyngeal abscess. Be aware that many patients have already started antibiotics, which may mask the clinical picture. Diphtheria is now most commonly seen in adults who lack immunization and previous exposure. *Haemophilus influenzae* is a rare cause of epiglottitis in children since the initiation of vaccination programs in 1990.

Physical examination

Diagnosing the cause of sore throat depends on an accurate assessment of the oropharynx and, in some cases, the nasopharynx and hypopharynx. The physical examination should focus on the anatomic location of any lesions and potential complications, especially airway obstruction and systemic disease.

General appearance

Assess for toxicity, a general impression of how ill the patient appears. A patient who prefers to be sitting up or standing with the neck flexed, the head extended and the nose pointed toward the ceiling ("sniffing position") may be self-stenting their airway to avoid complete obstruction. A patient may have trouble providing a history due to voice alteration and difficulty swallowing with drooling. Other signs of toxicity that require immediate attention include stridor, cyanosis, dyspnea and tachypnea.

Stridor is a loud, harsh respiratory sound that results from obstruction of the trachea or larynx. Stridor is usually heard during inspiration; in severe cases of obstruction, it may also be heard during expiration.

Vital signs

Fever and tachycardia are nonspecific signs, but demonstrate systemic involvement of the illness.

Oropharynx

Inspection

In cases of suspected epiglottitis (Figure 24.17) or retropharyngeal abscess, a complete examination of the oropharynx should be done cautiously or deferred until reaching the operating room (OR), due to the risk of precipitating airway occlusion. Be cautious examining any patient assuming the sniffing position, in respiratory distress or with drooling. Inability to fully open the mouth may indicate trismus and limit the examination.

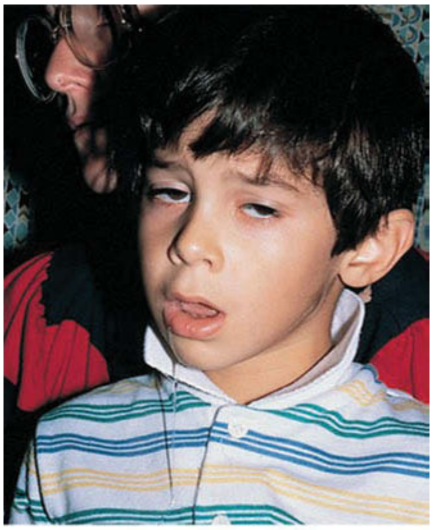

Figure 24.17
Epiglottitis. This 5-year-old, who had been symptomatic for several hours, holds his neck extended with head held forward, is mouth-breathing and drooling, and shows signs of tiring.

If the patient appears stable, examine the oral mucosa, hard and soft palates, oropharynx, tonsillar pillars and tonsils by holding the tongue down with a wooden blade. Prominent papillae on the tongue (strawberry tongue) may be seen with streptococcal infection. Look for erythema, exudates, pseudomembranes, swelling, petechiae (Figure 24.18), lesions (such as vesicles and ulcerations) and masses. A good light source is necessary. Local anesthetic sprays (e.g., Cetacaine) and having the patient assist by holding their own tongue down with a piece of gauze may allow a better examination in the patient with an overactive gag reflex. Having the patient say “ahhh” will also improve your view of the pharynx and tonsils by elevating the uvula and soft palate. Note the size, position and symmetry of the tonsils, looking especially at the degree of airway patency. Abnormal contours and bulges in the oropharyngeal wall may indicate a deep tissue infection. The oropharyngeal examination in a patient with peritonsillitis typically demonstrates unilateral soft palate swelling anterior and superior to the affected swollen tonsil, with loss of the line between the anterior tonsillar pillar and tonsil (Figure 24.19). The uvula is typically deviated to the opposite side. Unilateral bulging of the posterior pharyngeal wall may be seen with a retropharyngeal abscess. Bulging of the lateral wall of the oropharynx may be seen with a parapharyngeal abscess.

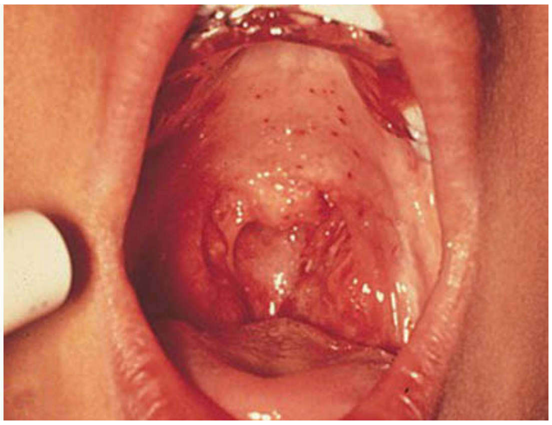

Figure 24.18
Palatal petechiae in a patient with group A beta-hemolytic streptococcal infection. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

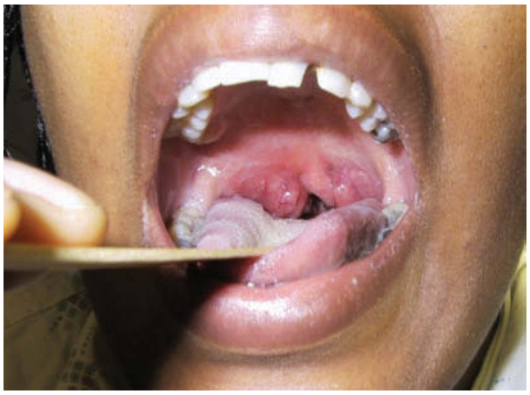

Figure 24.19
Right-sided peritonsillitis. Courtesy: S.V. Mahadeven, MD.

Palpation

Palpate and percuss the teeth and gums of any patient complaining of tooth pain. Palpation of swelling located on the soft palate or pharyngeal walls is not recommended due to the potential for disrupting an abscess.

Head, eyes, ears, nose and neck

Palpate the neck for evidence of enlarged or tender lymph nodes and for evidence of tumor or abscess. Gently palpate the hyoid bone, laryngeal and tracheal cartilages, and the thyroid. To examine the nasopharynx, use a headlight and nasal speculum. Inspect, palpate and percuss the sinuses for evidence of sinusitis or masses. Examine the ears for otitis media (OM), as it may manifest as throat pain, and pharyngitis may lead to OM.

Skin

Inspect the skin carefully for rashes or ulcers. Children with GABHS pharyngitis may develop a fine, diffuse papular erythroderma ("sandpaper rash") on the trunk that is worse in the groin and axillae. This scarlatiniform rash in the presence of pharyngitis is virtually diagnostic of GABHS infection with associated scarlet fever.

Cardiopulmonary

An examination of the heart and lungs, listening for murmurs or asymmetric and irregular breath sounds, should be performed in all patients with sore throat.

Abdomen

Palpate for tenderness and organomegaly. Splenomegaly and hepatomegaly may be seen with Epstein–Barr virus (EBV) infection. Abdominal tenderness with pharyngitis raises concern for splenic rupture in this setting.

Special signs/techniques

Unilateral enlargement of the pharynx or tonsil is associated with peritonsillitis, and less commonly neoplasms, vascular lesions and abscesses. *Exudates* are usually white or yellow spots on the tonsils (Figure 24.20). *Pseudomembranes* are usually gray-blue and tightly adherent to the posterior pharyngeal mucosa. When removed, a bleeding surface may be revealed.

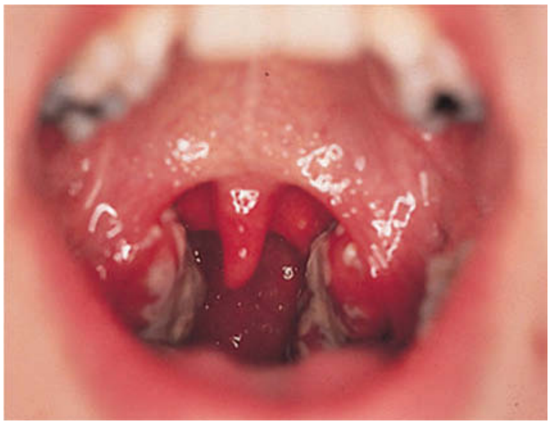

Figure 24.20
Exudative tonsillitis. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

Differential diagnosis

Table 24.14 provides a comprehensive list of causes of throat pain.

Diagnostic testing

Laboratory studies

White blood cell count

Ordering a white blood cell (WBC) count is of little value in most cases of sore throat. It may be useful if infectious mononucleosis (atypical lymphocytosis), serious bacterial infection, leukemia, or an immunocompromised state are concerns.

Blood cultures

Blood cultures should be obtained in patients with deep space infections (except most cases of peritonsillitis), immunocompromised states, sepsis and epiglottitis (once the patient's airway is secure).

Rapid diagnostic tests for group A beta-hemolytic streptococcus

GABHS is an important bacterial pathogen to detect, as appropriate antibiotic therapy can significantly reduce the duration of symptoms and possibly prevent development of acute rheumatic fever and suppurative complications. Rapid streptococcal antigen tests (RSATs) and throat cultures aid in the diagnosis of GABHS pharyngitis. The tonsils and posterior pharyngeal wall should be swabbed vigorously to obtain an accurate specimen for both RSATs and throat cultures. RSATs are generally considered to have good positive predictive values, but insufficient sensitivities to rule out GABHS infection (most being 79–95% sensitive). Specificities range from 31–100%, with most being 90–98% specific, depending on which commercial test is used. Results for RSATs usually return in 10–30 minutes. Both RSATs and cultures identify the Group A antigen, not active infection. Since 15–20% of the population are chronic carriers of GABHS, treating all positive RSATs or cultures with antibiotics results in overtreatment.

Throat cultures

All negative RSATs should be confirmed with culture; otherwise, a significant number of cases of GABHS pharyngitis will be missed. Culture for GABHS is 90–95% sensitive and 94–100% specific. About one-third of patients with infectious mononucleosis and diphtheria have positive GABHS cultures, which may lead to misdiagnoses. The true gold standard for determining GABHS infection is with acute and convalescent antistreptolysin-O (ASO) titers. However, ASO titers are not practical in an outpatient setting and are rarely done.

Table 24.14 Differential diagnosis of throat pain

Diagnosis	Symptoms	Signs	Work-up
Agranulocytosis	Sore throat; fever, malaise, nausea, vomiting; bleeding tendency	Rough-edged ulcers with gray-black membranes on gums, palate and possibly perianal area	CBC with differential showing low granulocytes; confirmatory bone marrow biopsy
Associated with flu-like illness (adenovirus, common cold and influenza)	Occur in epidemics; "scratchy" sore throat; absent or low-grade fever; cough, rhinorrhea, sneezing, myalgia and headache	Mild or absent erythema and edema of pharynx with normal tonsils; adenovirus may mimic GABHS; unilateral conjunctivitis, viral enanthemas, and stomatitis associated with adenovirus	Clinical diagnosis; point-of-care testing available for influenza
Associated with infectious mononucleosis-like illness (EBV, CMV and primary infection with HIV type 1)	Mainly affects 15- to 30-year age group; immunocompromised children at higher risk; often close contacts with same; risk factors seen with HIV; fluctuating fevers, malaise, anorexia, headache, myalgias and sore throat lasting weeks	EBV can lead to severely swollen tonsils with exudates and (rarely) airway obstruction; cervical adenopathy in 90% cases; painless splenomegaly and hepatomegaly in 50% EBV cases	Heterophil antibody test for EBV ("monospot"); other adjunctive tests for EBV include peripheral blood smear, CBC, and EBV antigen tests; HIV PCR testing
Associated with stomatitis (coxsackie and herpes infections)	Affects mainly toddler and school age children; HSV-2 pharyngitis mainly affects young adults; fever precedes oral lesions	Vesicles and/or ulcers on posterior pharynx with herpangina; on pharynx, lips, tongue, and buccal mucosa with HSV gingivostomatitis, and throughout oral cavity and on hands, feet, buttocks with HFM disease. Pharyngeal exudates and tender adenopathy with HSV-2 pharyngitis	Clinical diagnosis for herpangina and HFM disease (coxsackie viruses) and gingivostomatitis (HSV-1); viral throat culture and cytopathologic scrapings of lesions for HSV-2 pharyngitis
Bacterial pharyngitis	Fever; odynophagia and dysphagia; associated headache, abdominal pain, nausea, and vomiting (especially children with GABHS); dysuria, genital discharge, rash and arthralgias may be reported with disseminated gonorrhea	Fever typically >38.3°C; exudates, tonsillar swelling, palatal petechiae; tender cervical adenopathy (severe with diphtheria); pseudomembrane with diphtheria and *Arcanobacterium hemolyticum*. Stridor, myocarditis, and neuropathy may be seen with diphtheria; scarlatiniform rash may be seen with GABHS	Controversy surrounding the work-up for GABHS: RSAT and throat culture (if RSAT negative) versus clinical diagnosis. Laboratory needs notification when diphtheria, *Arcanobacterium hemolyticum,* gonorrhea or *Chlamydia trachomatis* suspected. Genital cultures or urine probes for suspected gonorrhea or *Chlamydia trachomatis*
Bacterial tracheitis	Similar to epiglottitis except longer viral prodrome; often initially mistaken for croup	High fever and toxic-appearance; similar to epiglottitis	Lateral neck radiograph useful for excluding epiglottitis; laryngoscopy is gold standard
Burns (chemical and thermal)	Hot or caustic liquid exposure by ingestion or inhalation; symptoms may take up to 5 hours to develop; some combination of throat pain, dysphagia, odynophagia, chest, back, or abdominal pain; vomiting, hematemesis and respiratory complaints present; injury from hot liquids may cause epiglottitis	Findings variable; possible mucosal and tongue erythema, swelling, and ulceration; may have signs of upper airway obstruction (stridor, drooling, muffled voice); absence of oropharyngeal lesions does not exclude tracheal, esophageal, or gastric injury	Neck and chest radiographs may demonstrate positive findings; laryngoscopy useful
Candidal pharyngitis	*Risk factors*: immunocompromised, pregnancy, infancy, decreased salivary flow, dentures; burning sore throat, dysphagia, odynophagia	Pharyngeal erythema and edema; white plaques when scraped off reveal superficial erythematous ulcer	Clinical diagnosis; yeast seen on KOH preparation of throat swabs
Cancer (laryngeal, tongue, tonsil and soft palate)	Heavy tobacco with or without alcohol. Persistent (>2 weeks) throat pain, hoarseness, dysphagia, cough and/or dyspnea; may have sensation of "lump in throat"	Normal pharyngeal examination; *tongue cancer:* raised white lesion or ulcer usually on posterolateral border of tongue; *tonsil and soft palate cancer:* superficial ulcer which may contain impacted food debris	Urgent referral to ENT for biopsy
Croup (parainfluenza virus, influenza virus and RSV)	Affects infants and toddlers; peaks in spring and fall; barking cough worse at night; often 2–3 day common cold prodrome	Inspiratory stridor; hoarseness; expiratory rhonchi; no dysphagia or drooling	Clinical diagnosis; lateral neck radiograph not necessary, but may see "steeple sign"
Epiglottitis	Most common in African Americans, males and smokers. Classically rapid onset severe sore throat, odynophagia, and dysphagia; may	Classically toxic-appearing with high fever, stridor, tongue protrusion, muffled voice, and assuming the "sniffing" or "tripod" position; may be more subtle	Lateral neck radiograph shows "thumbprint" sign; gold standard is laryngoscopy; blood cultures positive in 80–90% of bacterial cases

(*continued*)

Table 24.14 Differential diagnosis of throat pain (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
	have 1–2 day prodrome of cold symptoms; atypical presentations increasingly reported	with absence of fever (up to 50%) and pain out of proportion to examination; often coexisting pharyngitis. Tenderness to palpation of anterior neck over hyoid and with moving larynx or upper trachea is a reliable finding	
Foreign body	Peanuts and popcorn in children; dentures, meat and bones in adults. Choking episode, dyspnea, throat pain, dysphagia, chest pain, vomiting and unexplained cough; pain may persist after FB dislodged	May cause high-pitched inspiratory stridor, barking cough, focal wheezing, dysphonia and drooling; FB may be seen lodged near tonsil on oropharyngeal examination	Plain radiographs only helpful if FB radiopaque; fiberoptic scope examination frequently reveals FB or abrasion in lingual or palatine tonsils or pyriform sinus
Laryngeal trauma	Rare, but many cases unrecognized; occurs after motor vehicle crashes, assaults and sports injuries; may be asymptomatic initially; earliest symptom may be subtle voice change. *Other:* throat pain, dysphagia, dyspnea, cough, hemoptysis	May see swelling, bruising, seatbelt mark, laryngeal/tracheal tenderness, and crepitus; signs often absent	Plain neck and chest radiographs may show air in soft tissues; CT will demonstrate fractures and dislocations; indirect laryngoscopy also useful
Laryngitis	Mild sore throat; hoarseness predominant; viral URI symptoms	Hoarseness; otherwise normal oropharyngeal examination	Clinical diagnosis
Lingual tonsillitis	Rare, but seen in patients without palatine tonsils; may be acute or chronic; may cause sleep apnea; throat pain (above hyoid bone) worse with tongue motion; sensation of throat swelling; dysphagia	May have muffled voice; normal-appearing pharynx; cervical adenopathy	Indirect laryngoscopy
Ludwig's angina	Usually preceded by dental procedure or infection; ≥48 hours of symptoms; progressive throat pain, odynophagia, dysphagia, anterior neck pain and swelling; alteration in voice, drooling, and halitosis	Usually toxic-appearing with high fever and dehydration; lymphadenopathy; stridor if severe; bilateral submandibular swelling ("bull neck") with marked tenderness (may have "woodiness" or crepitus on palpation); elevation of floor of mouth with tongue protrusion	Lateral neck radiograph shows swelling of submandibular tissues; CT of face and neck with IV contrast for confirmation and surgical planning
Parapharyngeal abscess	Rare; spread from dental infection (30%); ≥48 hours of symptoms; fever, lateral neck pain and swelling	Usually toxic-appearing with fever and dehydration; lymphadenopathy; examination may be limited by trismus; stridor when supine if severe; lateral neck swelling and mass below angle of mandible	Lateral neck radiograph of limited use; CT of neck and mediastinum with IV contrast for confirmation and surgical planning
Peritonsillitis/ peritonsillar abscess	Most common deep space infection; adolescents and young adults; increased risk in diabetes, immunocompromise; preceded by pharyngitis; ≥48 hours of symptoms often despite antibiotics; fever, progressive throat pain, odynophagia, dysphagia, alteration in voice, drooling and halitosis	Toxic-appearing with high fever and dehydration; lymphadenopathy; trismus may limit examination in severe cases; stridor in supine position if severe; unilateral swelling anterior and superior to tonsil with loss of line between anterior tonsillar pillar. Tonsil and uvula deviation to contralateral side	Clinical diagnosis confirmed by needle aspiration; aspirated pus sent for Gram stain and culture; if diagnosis suspected and needle aspiration negative, CT with IV contrast or US
Retropharyngeal abscess	≥48 hours of symptoms; fever, drooling, poor feeding, and irritability in infants; neck pain, dysphagia in older children and adults	Classically toxic-appearing with fever and dehydration; lymphadenopathy; stridor in supine position if severe; unilateral bulging of lateral or posterior wall of oropharynx; meningismus and torticollis may be present	Lateral neck radiograph is a screening measure; CT of neck and mediastinum with IV contrast for confirmation and surgical planning
Vincent's angina (ANUG)	Poor dental hygiene; abrupt onset severe throat pain, odynophagia and foul taste; fever, malaise	Gray exudates over gums and tonsils; gingival ulcers; submandibular adenopathy	Clinical diagnosis
Uvulitis	Throat pain and/or FB sensation	Uvula red and swollen	Clinical diagnosis. RSAT and throat culture may reveal GABHS as etiology

ANUG: acute necrotizing ulcerative gingivitis; CBC: complete blood count; CMV: cytomegalovirus; CT: computed tomography; EBV: Epstein–Barr virus; ENT: ear, nose, throat; FB: foreign body; GABHS: group A beta-hemolytic streptococcus; HFM: hand-foot-mouth; HIV: human immunodeficiency virus; HSV: herpes simplex virus; IV: intravenous; RSAT: rapid streptococcal antigen test; RSV: respiratory syncytial virus; URI: upper respiratory infection; US: ultrasound.

Table 24.15 lists clinical indications for throat cultures.

Table 24.15 Clinical indications for throat cultures

Evidence of epiglottitis, peritonsillitis, or retropharyngeal abscess (once airway secured)
Presence of a pharyngeal membrane: culture for *Arcanobacterium hemolyticum* and *Corynebacterium diphtheriae* (laboratory should be notified)
History of or suspected immunocompromised state (including history of splenectomy)
History of possible gonorrhea (laboratory should be notified)
History of prolonged and/or severe pharyngitis: consider obtaining cultures for *Yersinia*, *Arcanobacterium hemolyticum, and Corynebacterium diphtheriae,* and a monospot test
Pediatric patients (controversial)

Heterophil antibody test (monospot test)

The monospot test, used to detect EBV infectious mononucleosis, may not be positive until 1–2 weeks of illness. The test's sensitivity declines as the patient's age decreases, with 95% sensitivity in adults but only 30% sensitivity in those less than 20 months of age. This test is almost always negative in persons of Japanese ancestry (for unknown reasons). False positives may occur with some systemic illnesses, such as leukemia.

Electrocardiogram

An electrocardiogram (ECG) is indicated in the patient with throat discomfort, a negative pharyngeal examination, and a history concerning for acute coronary syndrome (ACS).

Radiologic studies

Plain films

A soft tissue lateral view of the neck is useful in the work-up of croup, epiglottitis, lingual tonsillitis, retropharyngeal abscess, Ludwig's angina, laryngeal trauma and suspected FBs. However, plain radiographs may be normal despite the presence of these illnesses. Any patient who appears unstable should not leave the emergency department (ED) for radiographs.

The "steeple sign" (narrowing of the airway) due to glottic and subglottic edema is a reliable finding of croup, although an X-ray is not required to make the diagnosis. A soft tissue lateral radiograph of the neck is abnormal in 90% cases of epiglottitis. Positive findings include an enlarged, misshapen epiglottis ("thumbprint" sign) and swelling of the retropharyngeal soft tissues (Figure 24.21). Plain films are useful for excluding epiglottitis in cases of bacterial tracheitis and croup.

Abnormal retropharyngeal soft tissue swelling may also be seen with retropharyngeal abscess (Figure 24.22). Nonspecific soft tissue swelling may be seen with parapharyngeal abscess. Patients with Ludwig's angina may have swelling of the submandibular soft tissues, airway narrowing and gas collections on plain film. Air in the soft tissues may also be seen on plain film in patients with laryngeal trauma or burns. If a FB is radiopaque, it may be seen.

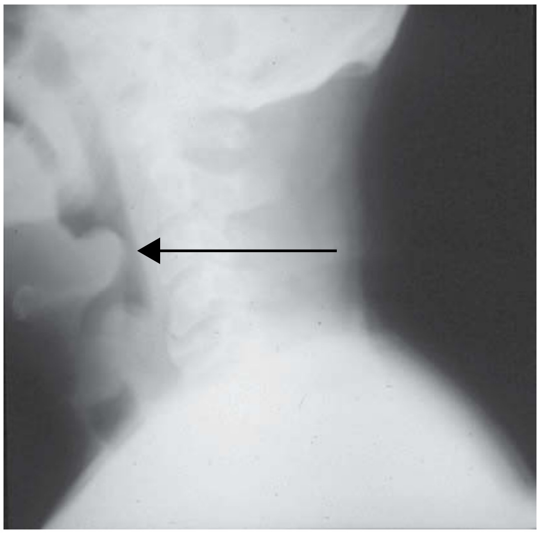

Figure 24.21
Epiglottitis. Lateral view of the cervical soft tissues demonstrating marked swelling of the epiglottis *(thumbprint sign)* with obliteration of the vallecula. Courtesy: Edward Damrose, MD.

Ultrasound

Ultrasound (US) is useful in the evaluation of deep space infections when the goal is to distinguish between cellulitis and abscess. US is preferred over computed tomography (CT) in critically ill patients, who should not be transported from the ED.

Computed tomography

CT with intravenous (IV) contrast is useful in the evaluation of throat pain with a suspected neck mass or laryngeal trauma. CT may also help distinguish abscess from cellulitis, and assist in surgical planning for deep space infections. CT will demonstrate fractures of the hyoid, cricoid and thyroid cartilages, and dislocation of the cricoarytenoid joints. The patient's airway must be stable prior to transport to CT.

Laryngoscopy

Patients with drooling, inability to swallow, FB sensation, dysphonia and/or laryngeal neck pain require complete visualization of the pharynx if history, physical and diagnostic imaging do not identify the etiology of the illness. Laryngoscopy is used to definitively diagnose epiglottitis, bacterial tracheitis, lingual tonsillitis, FB, injury from laryngeal trauma, and chemical and thermal burns. Visualization of a swollen epiglottis ("cherry red") is seen in epiglottitis (Figure 24.23). Erythematous, swollen lingual tonsils covered with exudates are seen with lingual

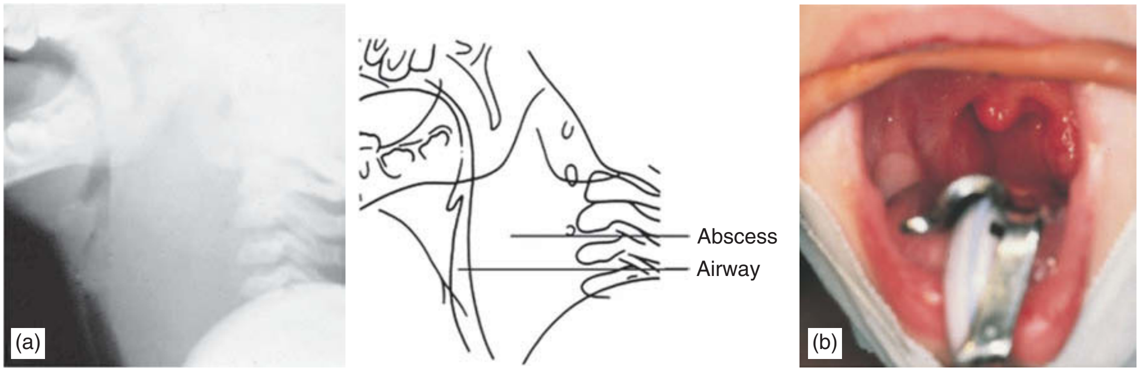

Figure 24.22
Retropharyngeal abscess. (a) A lateral neck radiograph and associated line diagram reveal prominent prevertebral soft tissue swelling that displaces the trachea forward. (b) Pharyngeal examination in the operating room revealed an intensely erythematous, unilateral swelling of the posterior pharyngeal wall. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

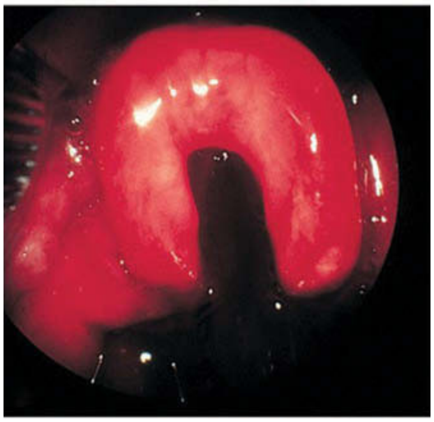

Figure 24.23
In the operating room, the epiglottis can be visualized and appears intensely red and swollen. It may retain its omega shape. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

tonsillitis. An FB or abrasion in the lingual or palatine tonsils or pyriform sinus may be seen. Laryngoscopy may show mucosal tears, cartilaginous fractures, or dislocations in patients with trauma. Edema, burned tissue, erythema and ulcerations may be seen on laryngoscopy in a patient with a chemical or thermal burn.

General treatment principles

Most management decisions relevant to the patient with sore throat concern antibiotic use and palliative measures. More serious considerations involve airway management and emergency anesthesiology or ENT consultation for procedures.

Airway management

Patients with illnesses associated with upper airway involvement (e.g., epiglottitis, deep space infections, trauma, burns and FBs) should be handled carefully to avoid precipitating sudden complete airway obstruction. Allow patients to maintain the position in which they are most comfortable. Pediatric IV lines should not be established in the ED unless the child is already in extremis. Never leave these patients alone. Difficult airway equipment should be ready at the bedside. Definitive airway management is best accomplished by an otolaryngologist and anesthesiologist in the OR with the neck prepped for a tracheostomy.

Volume repletion

Patients who are dehydrated or not tolerating sufficient oral hydration should be given IV crystalloid fluids.

Pain relief

Anesthetic lozenges, throat sprays and saltwater gargles may soothe mild to moderate discomfort. Viscous lidocaine or Xylocaine may be used for acute temporary relief, but should not be used frequently or chronically because they may mask an underlying disorder and cause toxicity. These agents also decrease the gag reflex and may lead to aspiration. Gargling with Benadryl elixir is another option, although the pain relief is brief. Patients with mild to moderate pain may do well with acetaminophen or ibuprofen alone. Elixirs (even in adults) may be better tolerated than tablets. Patients with severe pain may require oral or IV narcotics. Other palliative measures include air humidification and voice rest.

Antibiotics

Antibiotics are indicated in patients with suspected bacterial infections. Despite patients' misconceptions, antibiotics will not help most sore throats. The disadvantages of overtreating with antibiotics include increased bacterial drug resistance, decreased immune response, disruption of natural microbial ecology, antibiotic-associated side effects and patients' expectations for antibiotics with repeated episodes of sore throat. GABHS pharyngitis generally resolves spontaneously in 3–5 days without antibiotics. However, untreated GABHS infection may result in significant sequelae, including rheumatic fever, peritonsillar abscess and glomerulonephritis. A recent resurgence in invasive streptococcal infections (e.g., scarlet fever and streptococcal toxic shock syndrome) has influenced the aggressive treatment of pharyngitis. Early antibiotic treatment has been shown to shorten the course and severity of illness, and decrease transmission of GABHS. In a study of GABHS with severe symptoms, symptoms resolved 2.5 days earlier in patients treated with antibiotics.

The selection of patients with pharyngitis for laboratory testing and antibiotic treatment is controversial. One popular strategy is based on the adult scoring system for GABHS, also known as the *Centor criteria*. The presence of three or more risk factors (Table 24.16) indicates a 50–60% probability of disease; thus, empiric oral antibiotic therapy has been previously recommended for these patients. However, this approach leads to high rates of unnecessary antibiotic exposure. The general consensus is that patients with a history of rheumatic fever or a family member with a history of rheumatic fever (or documented GABHS infection), evidence of scarlet fever and/or partially treated pharyngitis should be empirically treated with antibiotics. Many experts recommend empiric treatment for GABHS pharyngitis in the midst of a GABHS, rheumatic fever, or glomerulonephritis outbreak. Some believe that those who will be unavailable for follow-up (noncompliant patients) should be empirically treated for GABHS pharyngitis, since cultures take 24–48 hours to return. Patients with fewer than two risk factors may undergo RSAT and throat culture if RSAT negative, and only be treated if either result is positive. Despite the fact that many providers empirically prescribe antibiotics for the above-mentioned situations, the American Academy of Pediatrics (AAP), the American Heart Association (AHA) and the Infectious Disease Society of America (IDSA) all recommend doing at least one laboratory test before deciding whether to administer antibiotics. The best strategy for an individual clinician depends on the prevalence of streptococcal disease, the ease of follow-up, and the availability and accuracy of the specific RSAT used.

Antibiotic therapy for GABHS pharyngitis should be initiated within 9 days of symptom onset to prevent acute rheumatic fever. The treatment of choice for GABHS infection remains penicillin. A one-time dose of parenteral benzathine penicillin should be given to patients who cannot tolerate per os (PO) or in whom poor compliance is suspected.

Table 24.16 Adult scoring system for GABHS

Risk factors
Pharyngeal exudates
Tender anterior cervical adenopathy
Fever >38°C
Absence of cough and coryza

GABHS: Group A beta-hemolytic streptococcus.

Modified from Centor RM, Witherspoon JM, Dalton HP Brody CE, Link K. The diagnosis of strep throat in the emergency room. *Med Decis Making* 1981;**1**:239–46.

Increased GABHS treatment failure with penicillin has been reported. Erythromycin is one alternative to penicillin, although several other regimens exist. Current guidelines recommend first-generation cephalosporins for persons with penicillin allergy; some experts advocate the use of cephalosporins in all nonallergic patients because of better GABHS eradication and greater efficacy against chronic GABHS carriage.

Antibiotic treatment is also indicated for lingual tonsillitis, gonococcal and chlamydial pharyngitis, diphtheria (which also requires treatment with antitoxin), *Arcanobacterium hemolyticum* pharyngitis, Vincent's angina, epiglottitis and deep space infections. Candidal pharyngitis should be treated with oral fluconazole or itraconazole. Patients with evidence of herpes pharyngitis should be treated with acyclovir or famciclovir.

Steroids

Steroids may be useful with severe bilateral tonsillar swelling in infectious mononucleosis and some cases of lingual tonsillitis. Research has demonstrated that steroids slightly reduce time to resolution of pain in severe or exudative cases of pharyngitis. Steroid use for epiglottitis, Ludwig's angina and caustic ingestion is controversial. A single dose of steroids is useful in the treatment of croup.

Racemic epinephrine

Racemic epinephrine is useful in reducing airway edema in moderate and severe croup. It has reportedly been used for epiglottitis and lingual tonsillitis. Evidence-based studies are needed prior to recommending its use in these circumstances.

Needle aspiration or incision and drainage

Until recently, incision and drainage or immediate tonsillectomy was the recommended treatment for peritonsillitis caused by an abscess. Currently, needle aspiration by either a physician skilled in this procedure or an otolaryngologist is recommended. This has been shown to be equally effective, safer, and less painful compared with incision and drainage. Patients with severe trismus or those who cannot cooperate (young children) are best served by having this procedure or a tonsillectomy done in the OR by an otolaryngologist. Surgical drainage for Ludwig's angina is reserved for patients with crepitance and abscess, and may be done to eradicate dental infections as well. Most cases of retropharyngeal abscess require surgical drainage.

Special patients

Elderly

The incidence of infectious pharyngitis declines with age. Persistent sore throat without obvious physical findings in an elderly patient should prompt a search for a neoplasm, particularly if there is a history of tobacco use.

Pediatric

Children with GABHS pharyngitis should receive antibiotics for 24 hours prior to returning to school. Gonococcal pharyngitis may be seen in sexually abused children and sexually active adolescents.

Immune compromised

Any immunocompromised patient with pharyngitis, who is going to be discharged, needs to be followed very closely. Asplenic patients are at risk for developing streptococcal sepsis and should be admitted. Leukopenic patients should only be discharged if they have an adequate granulocyte count. Candidal infection is the most common type of pharyngitis in patients with acquired immunodeficiency syndrome (AIDS). A patient with a candidal infection without an obvious underlying risk factor should be evaluated for potential neoplasm or an immunocompromised state.

Infectious mononucleosis

Patients should be informed that infectious mononucleosis may persist for weeks to months. Steroids may help reduce severe tonsillar edema. Any patient with infectious mononucleosis and abdominal pain should undergo immediate US or CT to detect splenic rupture, which typically occurs after 4–6 weeks of illness. If given amoxicillin or ampicillin, 90% of patients with EBV infection will develop a diffuse macular rash, often mistaken for an allergic reaction. All patients with infectious mononucleosis should be seen by their primary care physician within 1 week of their diagnosis and instructed to avoid contact sports.

Post-tonsillectomy

Up to 6% of patients will present with bleeding 5–10 days after tonsillectomy. The majority of these patients are 21 to 30 years of age, and have minor bleeding from the tonsillar veins that can be controlled with direct pressure. About 1% of patients presenting with post-tonsillectomy bleeding have major bleeding, which requires emergent airway control and massive transfusion. ENT should be consulted emergently for all patients with post-tonsillectomy bleeding.

Disposition

Emergent ENT consultation and admission

The following are admission criteria for patients with throat pain:

1. Evidence of or at risk for airway compromise (includes all suspected cases of epiglottitis, retropharyngeal abscess, Ludwig's angina and diphtheria).
2. Cannot maintain hydration or swallow.
3. Require IV antibiotics.
4. Patients whose pain is intolerable despite maximal oral analgesia.
5. Controversy still exists concerning whether the patient with a peritonsillar abscess should be treated in the ED and discharged or hospitalized. This depends not only upon the appearance of the patient but the preference of the ENT consultant.
6. Evidence of disseminated infection.
7. Evidence of deep neck space infection.
8. Evidence of or significant risk for sepsis (often in immunocompromised patients).
9. Post-tonsillectomy patients with bleeding.

Any patient with a chronic sore throat or evidence of carcinoma of the oropharynx should be referred to ENT to be seen within 3–5 days for further work-up of a potential neoplasm.

Observation/serial evaluation

Patients with peritonsillitis may benefit from observation over a several hour period, during which time they receive IV hydration, antibiotics and a PO challenge. All of these patients must have close follow-up within 24 hours with an otolaryngologist to check for abscess formation.

Discharge

Most patients with sore throat can be safely discharged. If antibiotic treatment is planned pending culture results, it is important to establish a detailed plan for follow-up.

Pearls, pitfalls and myths

- Recognize the signs of impending airway obstruction: sniffing position, apprehension, tachypnea, drooling, voice alteration and stridor. Patients with these signs should be allowed to assume the position in which they are most comfortable.
- ENT and anesthesia should be consulted emergently and the OR prepared for patients who appear to have impending or actual airway obstruction.
- Always be prepared for complete airway obstruction and other catastrophic complications (sepsis, carotid artery hemorrhage) in any patient with a deep space infection or epiglottitis.
- Epiglottitis may be overlooked (especially in adults), resulting in fatal consequences. Consider this diagnosis in those with rapid onset of sore throat, throat pain out of proportion to examination, respiratory symptoms accompanying the sore throat, or the sensation of a "lump" in the throat.
- Do not fail to recognize an abscess or impending abscess in the potential spaces of the head and neck.
- Plain radiographs of the neck may be useful for detecting retropharyngeal abscess and epiglottitis. Advanced imaging in a stable patient with a secure airway is useful for further diagnosis, distinguishing abscess from cellulitis, and surgical planning.

- Antibiotics should be tailored to the specific disease process suspected. Understand the rationale and criteria for testing for and empirically treating pharyngitis.
- Needle aspiration of a suspected peritonsillar abscess should be attempted only by physicians skilled in this procedure, because significant complications (puncture of major vasculature in the neck) are possible.
- A patient with a chronic sore throat (especially one with an alcohol or tobacco history) needs prompt referral to ENT for work-up of a potential cancer.

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25 Extremity trauma

Dan Garza, MD and Gregory W. Hendey, MD

Scope of the problem

Trauma to an extremity is a common reason for a patient to present to the emergency department (ED). According to the 2006 National Hospital Ambulatory Medical Care Survey, there were over 17 million visits to the ED in the year for fractures, sprains, strains and diseases of the musculoskeletal system. The most common sites of injury were the wrist and hand, followed by the ankle and shoulder. It is important to perform a thorough yet efficient history and physical examination in order to accurately diagnose and provide initial treatment for these injuries. When improperly treated, extremity injuries may lead to long-term pain and disability.

Anatomic essentials

Each extremity can be viewed as a group of individual bones held together by a musculoligamentous apparatus. Careful attention must be paid to the vascular and nerve supply to each extremity; injury to these structures may be overlooked when fractures are present. Each extremity is encased in soft tissue that is often subdivided into fascial compartments. The clinician should become familiar with the normal anatomy and pathology of an extremity in this context: bones and ligaments, muscles and tendons, nerves and vessels, and soft tissue (compartments). The examination is complete only when all of these structures in the relevant area have been assessed (Tables 25.1 and 25.2).

Sensory and motor innervation of the extremities can be rapidly assessed. When evaluating sensorimotor function due to an extremity injury, the examiner should focus on peripheral nerves rather than nerve roots and dermatomal distribution, as is the case with vertebral injury (Table 25.3).

Each extremity is divided into compartments by longitudinal fascia. Best seen on cross-section, these compartments are named according to their anatomic position. For example, the compartments of the leg and the structures they contain are shown in Table 25.4 and Figure 25.9.

Table 25.1 Bones, ligaments, arteries and nerves of the upper extremity

	Bones	Ligaments	Arteries	Nerves
Shoulder (Figure 25.1)	Scapula Humerus Clavicle	Acromioclavicular Coracoclavicular Coracoacromial Coracohumeral Capsular ligaments Transverse ligaments of humerus	Axillary Anterior circumflex humeral Posterior circumflex humeral	Axillary Musculocutaneous
Elbow (Figure 25.2)	Humerus Radius Ulna	Annular Ulnar collateral Radial collateral	Brachial Inferior ulnar collateral Superior ulnar collateral Radial collateral	Median Radial Ulnar
Wrist (Figure 25.3)	Radius Ulna Carpals	Ulnar collateral Radial collateral Palmar radiocarpal Dorsal radiocarpal	Radial Ulnar	Median Radial Ulnar
Hand (Figures 25.3 and 25.4)	Carpals • Scaphoid • Lunate • Triquetral • Pisiform • Trapezium • Trapezoid • Capitate • Hamate Metacarpals	Intercarpal ligaments Palmar carpometacarpal Dorsal carpometacarpal Palmar and collateral metacarpophalangeal ligaments Deep transverse metacarpal Superficial transverse metacarpal	Deep palmar arch Superficial palmar arch Common palmar digital	Median • Muscular branch • Common palmar digital Ulnar • Superficial branch • Deep branch • Common palmar digital
Digits (Figure 25.4)	Proximal phalanges Intermediate phalanges (except thumb) Distal phalanges	Palmar and collateral ligaments of proximal interphalangeal joints and distal interphalangeal joints	Palmar digital	Palmar digital

Red flags

Emergency clinicians must be adept at recognizing “red flags” (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 25.5).

History

How did the injury occur?

The nature, magnitude and direction of forces applied to the extremity help determine the likely resulting injury. Crush injury may predispose to compartment syndrome

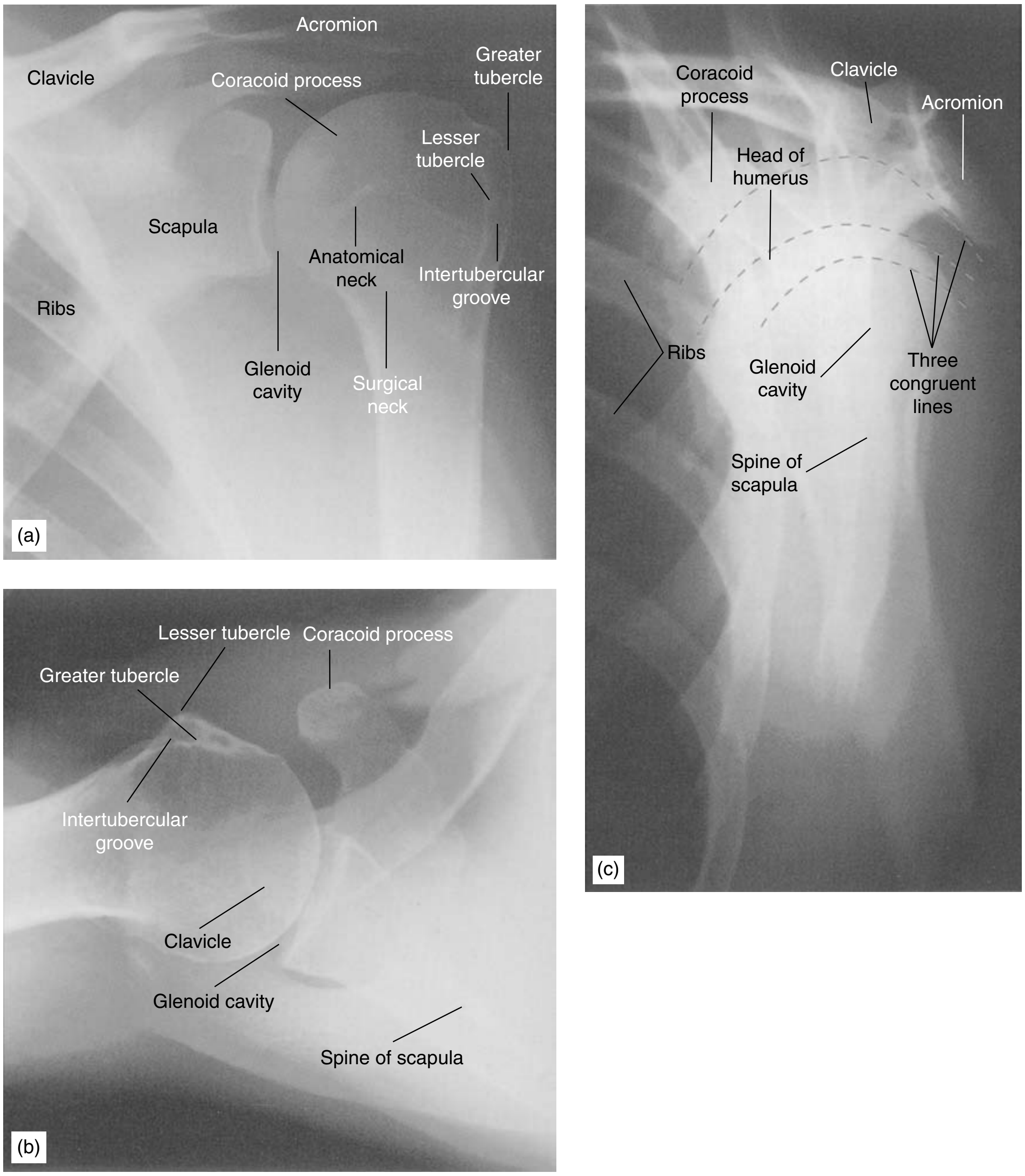

Acromion
Clavicle
Coracoid process
Greater tubercle
Lesser tubercle
Scapula
Anatomical neck
Intertubercular groove
Ribs
Glenoid cavity
Surgical neck
(a)
Lesser tubercle
Coracoid process
Greater tubercle
Intertubercular groove
Clavicle
Glenoid cavity
Spine of scapula
(b)
Coracoid process
Clavicle
Acromion
Head of humerus
Ribs
Glenoid cavity
Three congruent lines
Spine of scapula
(c)

Figure 25.1
(a) Anteroposterior, (b) axillary, and (c) lateral radiographic projections of the shoulder. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

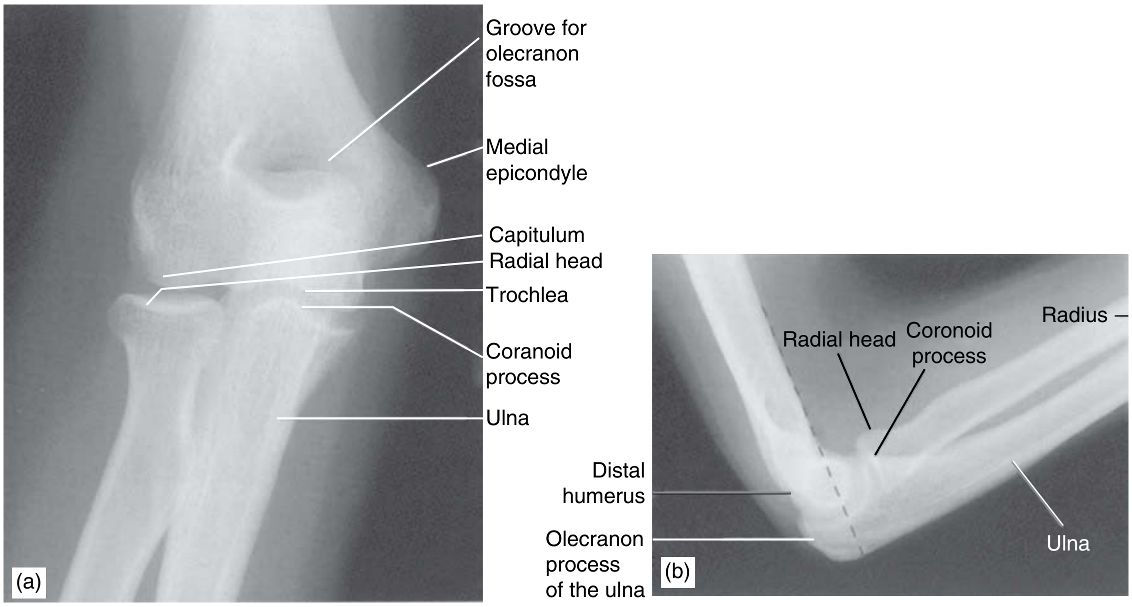

Groove for olecranon fossa
Medial epicondyle
Capitulum
Radial head
Trochlea
Coranoid process
Ulna
(a)
Radial head
Coronoid process
Radius
Distal humerus
Olecranon process of the ulna
Ulna
(b)

Figure 25.2
(a) Anteroposterior and (b) lateral radiographs of the elbow. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

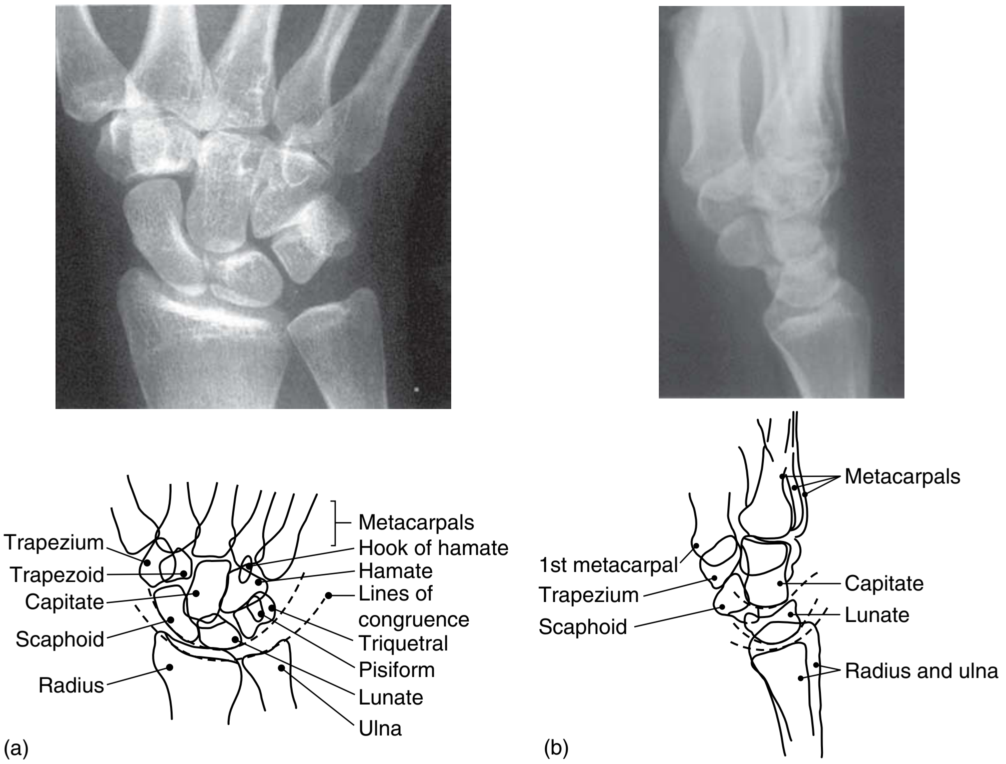

Trapezium
Trapezoid
Capitate
Scaphoid
Radius
Metacarpals
Hook of hamate
Hamate
Lines of congruence
Triquetral
Pisiform
Lunate
Ulna
(a)
Metacarpals
1st metacarpal
Trapezium
Scaphoid
Capitate
Lunate
Radius and ulna
(b)

Figure 25.3
(a) Anteroposterior and (b) lateral radiographs of the wrist. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

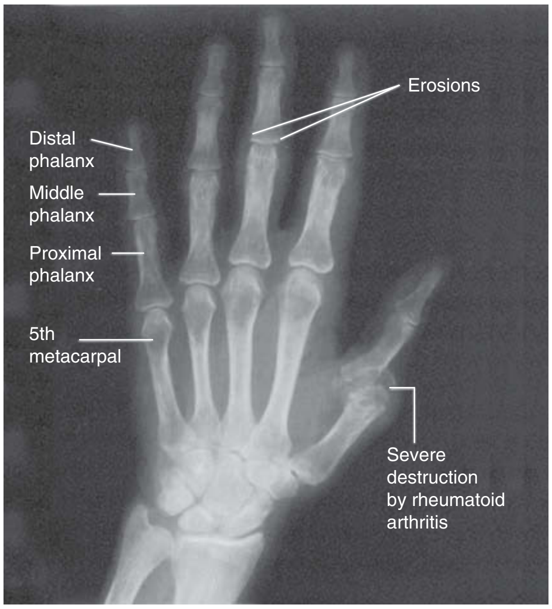


Figure 25.4
Radiograph of the hand. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

Table 25.2 Bones, ligaments, arteries and nerves of the lower extremity

	Bones	Ligaments	Arteries	Nerves
Hip (Figure 25.5)	Pelvis Femur	Iliofemoral Pubofemoral Ischiofemoral Round	Femoral Profunda femoris Medial femoral circumflex Lateral femoral circumflex Inferior gluteal	Femoral Obturator Sciatic
Knee (Figure 25.6)	Femur Tibia Fibula	Anterior cruciate Posterior cruciate Medial collateral Lateral collateral Anterior and posterior ligaments of head of fibula	Popliteal Anterior tibial Descending genicular Medial and lateral superior genicular Medial and lateral inferior genicular Descending branch of lateral femoral circumflex Anterior tibial recurrent Circumflex fibular	Tibial Common peroneal Medial and lateral sural cutaneous Saphenous
Ankle (Figure 25.7)	Tibia Fibula Talus	Lateral • Anterior tibiofibular • Anterior talofibular • Posterior talofibular • Calcaneofibular Medial • Deltoid (talar, calcaneal, navicular)	Anterior tibial Posterior tibial	Superficial peroneal Deep peroneal Saphenous Tibial Sural
Foot (Figure 25.8)	Calcaneus Talus Navicular Cuboid Cuneiforms • Medial • Intermediate • Lateral Metatarsals Phalanges	Bifurcate • Calcaneocuboid • Calcaneonavicular Interosseus talocalcaneal Plantar calcaneonavicular Plantar calcaneocuboid Long plantar	Medial plantar Lateral plantar Plantar arch Dorsalis pedis Arcuate Medial and lateral tarsal Dorsal metatarsal Plantar metatarsal Common plantar digital Proper plantar digital Dorsal digital	Medial plantar Lateral plantar Deep peroneal Common plantar digital Proper plantar digital Dorsal digital

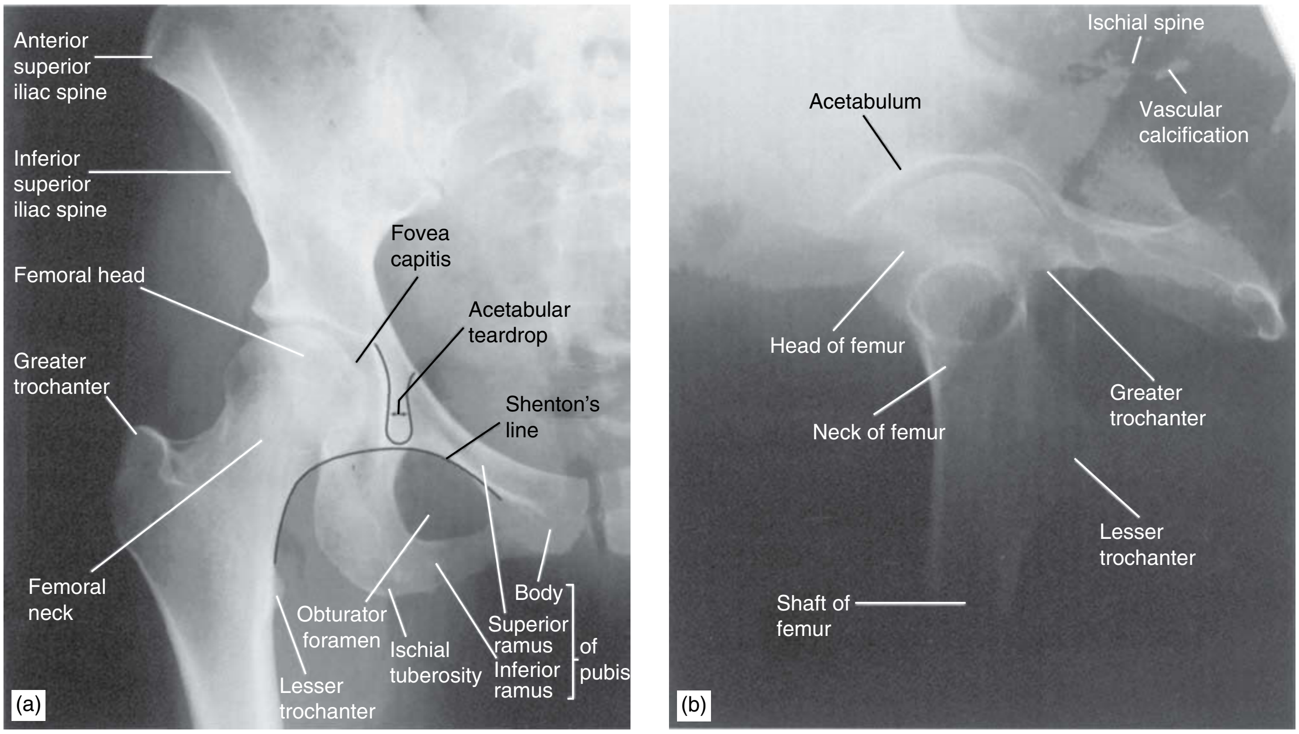

Anterior superior iliac spine
Inferior superior iliac spine
Femoral head
Greater trochanter
Fovea capitis
Acetabular teardrop
Shenton's line
Femoral neck
Obturator foramen
Ischial tuberosity
Lesser trochanter
Body
Superior ramus
Inferior ramus
of pubis
(a)
Ischial spine
Vascular calcification
Acetabulum
Head of femur
Neck of femur
Greater trochanter
Lesser trochanter
Shaft of femur
(b)

Figure 25.5
(a) Anteroposterior and (b) lateral radiographs of the hip. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

or rhabdomyolysis. A shearing force onto gravel or dirt raises suspicion for foreign bodies that increase the risk of wound infection or osteomyelitis. Falls from a height, significant collisions, or loading of a patient's entire weight on a single joint increases the likelihood of fracture. Trauma that involves force imparted across the transverse axis of a bone raises the possibility of a transverse fracture, whereas a force along the long axis will more likely lead to compression or impaction fractures. Table 25.6 summarizes classic injuries resulting from common mechanisms.

When did the injury occur?

Depending on the nature of the injury, the time elapsed since its onset may be important. As the amount of time between injury and definitive care for a laceration or open fracture increases, the risk of infection also increases. Depending on location, lacerations may need to undergo delayed closure if more than 6–12 hours have passed. In the case of vascular injury, blood flow must be restored within 6 hours for a meaningful chance of limb salvage.

Is the patient right- or left-handed? What is the patient's occupation?

It is appropriate to assess the relative importance of an affected upper extremity to a patient's quality of life. Although all patients should receive optimal care, an injury to the dominant hand of a professional illustrator may be treated more aggressively by a consultant.

What is the patient's tetanus status?

Although rare, the potentially fatal consequences of tetanus can be easily avoided with appropriate prophylaxis (Table C.3, Appendix C). The individuals most likely to have inadequate prior immunization are those older than 60 years and immigrants. If a patient's tetanus status is unknown or uncertain, he or she should receive the complete series.

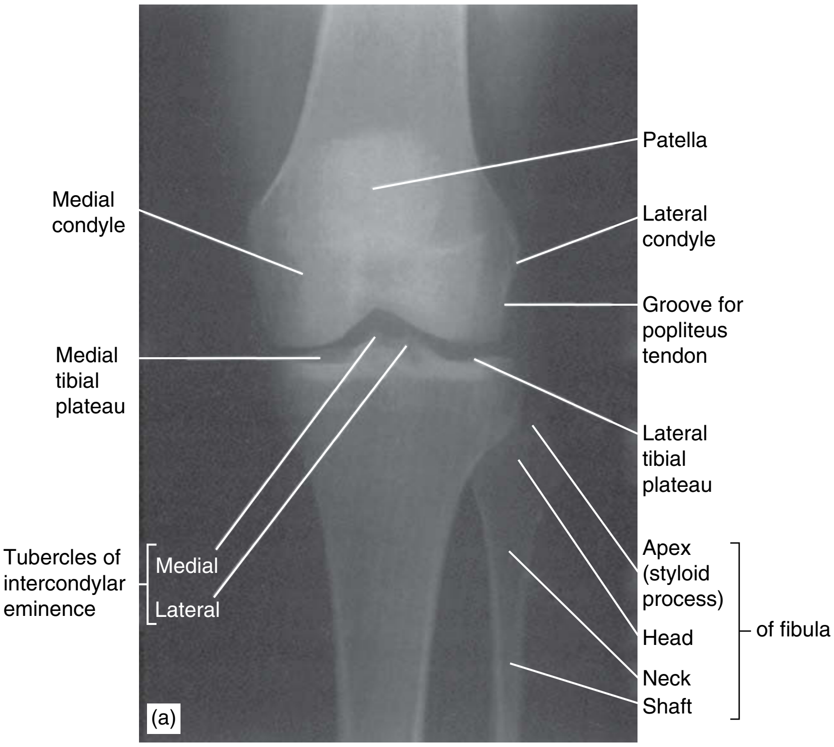
When was the patient's last meal?

The patient's injury may require reduction under procedural sedation or general anesthesia; assessing the risk of aspiration requires knowledge of the time since the patient's last meal. Acceptable limits vary according to institution and injury.

Associated symptoms

Is the extremity weak, cold, or numb?

These symptoms might indicate a nerve or vessel injury in the affected extremity. The clinician must perform a thorough neurovascular examination distal to the injury. An obvious bony deformity or joint dislocation should be reduced promptly in an attempt to restore any neurovascular deficit.


Patella
Medial condyle
Lateral condyle
Groove for popliteus tendon
Medial tibial plateau
Lateral tibial plateau
Tubercles of intercondylar eminence
Medial
Lateral
Apex (styloid process)
Head
Neck
Shaft
of fibula
(a)

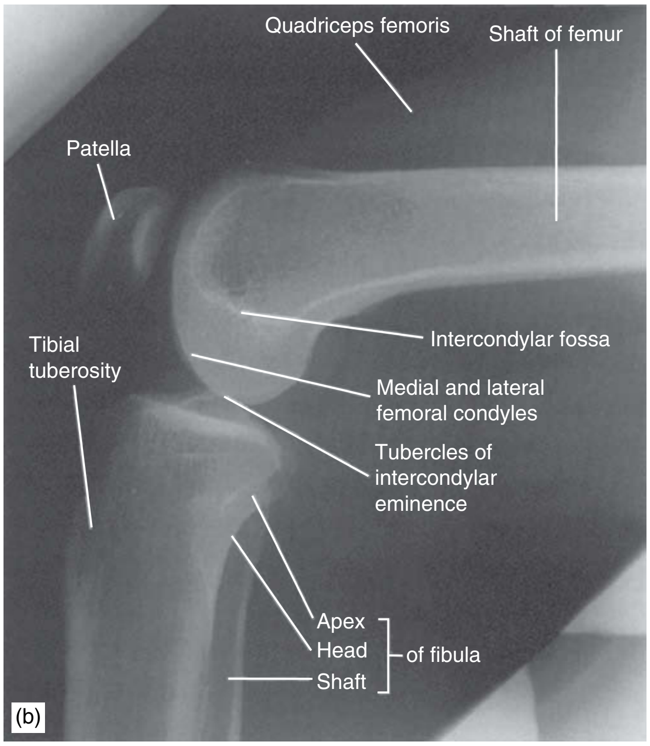

Quadriceps femoris
Shaft of femur
Patella
Intercondylar fossa
Tibial tuberosity
Medial and lateral femoral condyles
Tubercles of intercondylar eminence
Apex
Head
Shaft
of fibula
(b)

Figure 25.6
(a) Anteroposterior and (b) lateral radiographs of the knee. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

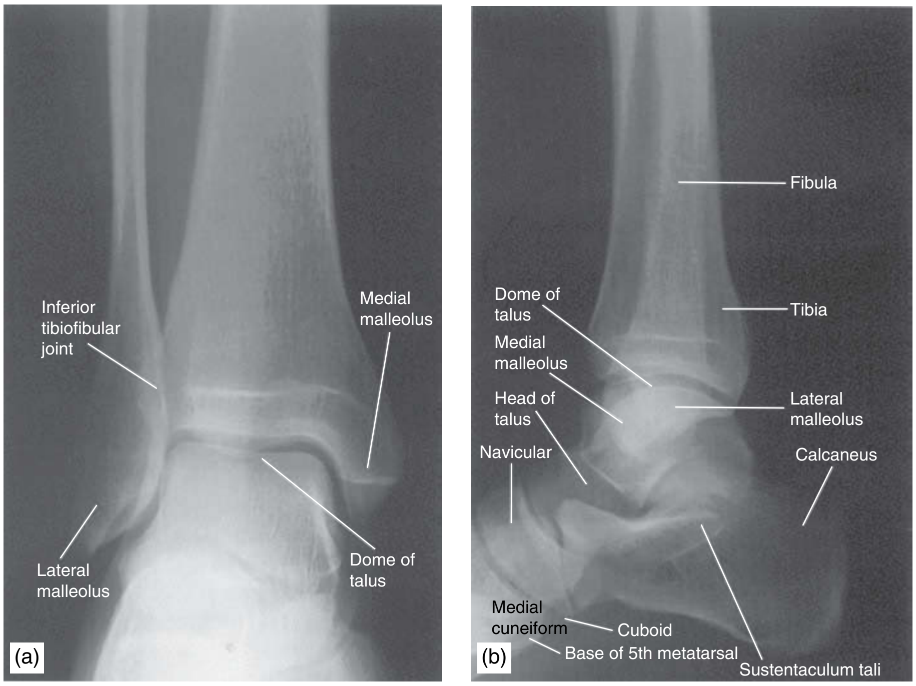

Inferior
tibiofibular
joint
Medial
malleolus
Lateral
malleolus
Dome of
talus
(a)
Fibula
Dome of
talus
Tibia
Medial
malleolus
Head of
talus
Lateral
malleolus
Navicular
Calcaneus
Medial
cuneiform
Cuboid
Base of 5th metatarsal
Sustentaculum tali
(b)

Figure 25.7
(a) Anteroposterior and (b) lateral radiographs of the ankle. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

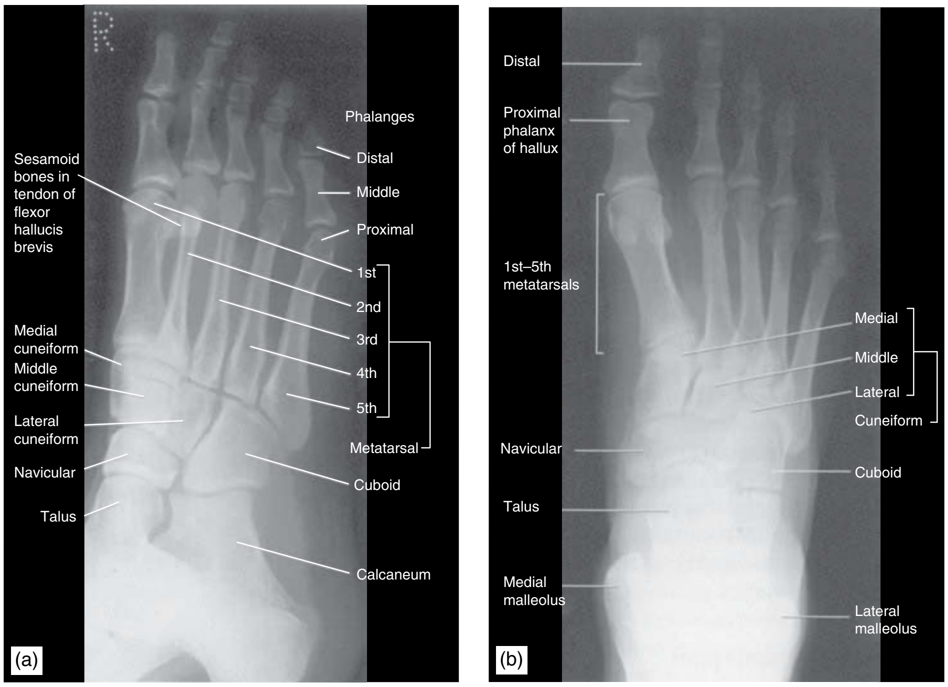

Phalanges
Distal
Middle
Proximal
Sesamoid
bones in
tendon of
flexor
hallucis
brevis
1st
2nd
3rd
4th
5th
Metatarsal
Medial
cuneiform
Middle
cuneiform
Lateral
cuneiform
Navicular
Cuboid
Talus
Calcaneum
(a)
Distal
Proximal
phalanx
of hallux
1st–5th
metatarsals
Medial
Middle
Lateral
Cuneiform
Navicular
Cuboid
Talus
Medial
malleolus
Lateral
malleolus
(b)

Figure 25.8
(a) Oblique and (b) anteroposterior radiographs of the foot. Reproduced from Butler P, Mitchell AWM, Ellis H, *Applied Radiological Anatomy*, Cambridge University Press, Cambridge, 1997.

Table 25.3 Peripheral nerves: sensory and motor function

Nerve	Sensory	Motor
Axillary (C5,6)	Lateral aspect of deltoid	Shoulder abduction
Median (C6–8)	Lateral palmar aspect of hand (including lateral palmar half of ring finger)	Abduction of thumb
Radial (C6–8)	Lateral dorsum of hand	Thumb/wrist extension
Ulnar (C8, T1)	Medial palmar aspect of hand (including medial palmar half or ring ringer)	Finger abduction
Femoral (L2–4)	Anterior aspect of thigh	Knee extension
Saphenous (L2–4)	Medial aspect of leg and foot	
Sciatic (L4–S3)	Posterior aspect of thigh	Knee flexion
Tibial (L4–S3)	Sole of foot	Plantar flexion (posterior compartment)
Common peroneal (L4–S2)	Posterior aspect of lower leg	
Superficial peroneal (L4–S2)	Lateral aspect of lower leg and dorsum of foot	Foot eversion (lateral compartment)
Deep peroneal (L4–S2)	First toe web space	Dorsiflexion (anterior compartment)

Table 25.4 Compartments of the leg

Compartment	Contents
Anterior	Muscles • Tibialis anterior • Extensor digitorum longus • Extensor hallucis longus • Peroneus tertius Anterior tibial artery Deep peroneal nerve
Lateral	Muscles • Peroneus brevis • Peroneus longus Superficial peroneal nerve
Deep posterior	Muscles • Tibialis posterior • Flexor digitorum longus • Flexor hallucis longus • Posterior tibial artery Peroneal artery Posterior tibial nerve
Superficial posterior	Muscles • Gastrocnemius • Plantaris • Soleus Sural nerve

Past medical

Of particular concern is the patient taking warfarin or presenting with coagulopathy, in whom blood loss and hematoma formation may be exaggerated. Patients on chronic steroid therapy are more prone to fractures. Other considerations include allergies to analgesics or anesthetics, and a history of prior surgeries or surgical hardware in the affected extremity.

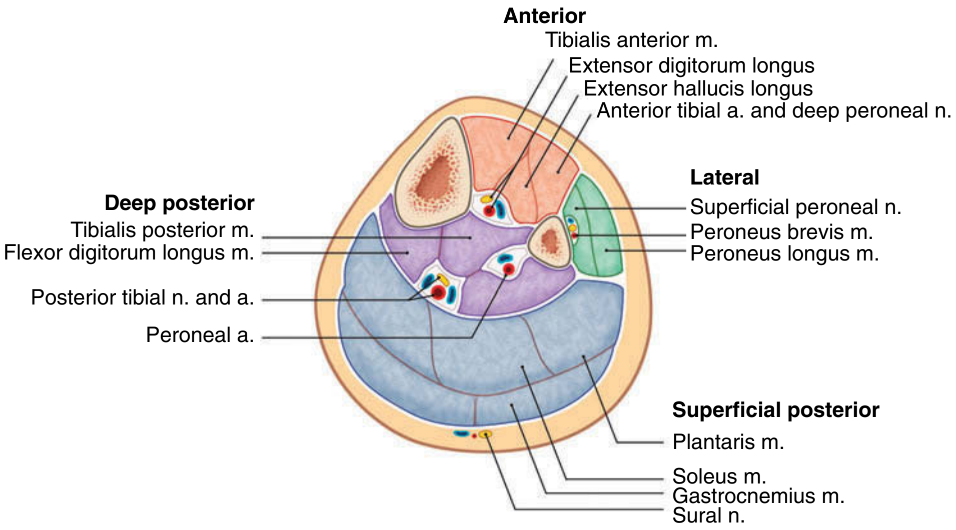

Anterior
Tibialis anterior m.
Extensor digitorum longus
Extensor hallucis longus
Anterior tibial a. and deep peroneal n.
Lateral
Superficial peroneal n.
Peroneus brevis m.
Peroneus longus m.
Deep posterior
Tibialis posterior m.
Flexor digitorum longus m.
Posterior tibial n. and a.
Peroneal a.
Superficial posterior
Plantaris m.
Soleus m.
Gastrocnemius m.
Sural n.

Figure 25.9
Compartments of the leg at its mid-length. © Chris Gralapp.

Table 25.5 Extremity trauma red flags

History	Concerning diagnosis
Blood or bleeding at the scene or on clothing/dressing	Open fracture or open fracture-dislocation
Pain out of proportion to mechanism or findings	Compartment syndrome
Elderly patient with hip pain, especially with weightbearing (negative plain films)	Occult hip fracture (may need MRI, CT, or bone scan), pubic ramus or acetabular fracture
Significant or specific mechanism of injury	Multiple fractures at different sites, fracture-dislocation, or combination fractures (Monteggia, Galeazzi, Maisonneuve)
Jumping from a height	Calcaneous fracture (Lover's fracture) with vertebral body compression fracture
Elderly, history of malignancy, mechanism does not "fit" fracture	Pathologic fracture
Grossly deformed extremity now "normal" after manipulation in the field	Dislocation reduced at the scene
Examination finding	**Concerning diagnosis**
Blood or bleeding	Open fracture
Marked swelling or tenseness; decreased sensation, temperature, or pulses	Compartment syndrome
Numbness, decreased sensation, decreased or absent distal pulses	Neurovascular injury
Tenderness on palpation of anatomic snuffbox (esp. with negative plain films)	Scaphoid fracture – splint with follow-up evaluation (may need CT or MRI)
Inability to reduce a dislocation	Consider fracture-dislocation, tendon or fracture fragment interfering with reduction and joint space, incorrect technique, inadequate sedation/relaxation
Grossly swollen knee	Posterior knee dislocation with possible vascular injury, tibial plateau fracture
Large hematoma or hemarthrosis	Coagulopathy, bleeding disorder

CT: computed tomography; MRI: magnetic resonance imaging.

Table 25.6 Common injuries with associated mechanisms

Mechanism	Possible injury
Fall onto shoulder	Acromioclavicular joint separation Shoulder dislocation Humerus fracture
Seizure Electrical injury	Posterior shoulder dislocation
FOOSH	Radial head fracture Colles fracture Scaphoid fracture
Pulling child's arm	Radial head subluxation (Nursemaid's elbow)
Striking knee against dashboard in high-speed collision	Posterior hip dislocation Femur fracture
Landing on feet after fall from height	Calcaneus fracture Tibial plateau fracture Vertebral compression fracture
Ankle inversion	Malleolus fracture Fracture of base of fifth metatarsal
Rotary ankle force	Malleolus fracture Maisonneuve injury
Inversion, medial or lateral stress to midfoot	Midfoot dislocation (Lisfranc injury)

FOOSH: fall on outstretched hand.

Modified from Tintinalli JE (ed). *Tintinalli's Emergency Medicine: A Comprehensive Study Guide,* 7th ed. McGraw-Hill, New York, 2011.

Physical examination

The physical examination should begin with adequate exposure. Patients often present with various bandages or splints applied, which must be carefully removed. Although the tendency to "just order an X-ray" may seem efficient, the few minutes required to carefully examine the injury may save unnecessary radiographs or reveal unexpected findings that demand immediate attention. In addition, jewelry and clothing that may form a tourniquet due to swelling should be removed immediately. Analgesia should not be withheld pending a definitive diagnosis. In fact, the use of parenteral, regional, or local anesthesia may assist the clinician by making it easier for the patient to comply with the physical examination. It is recommended to perform a sensory examination prior to applying a local or regional nerve block.

Vital sign abnormalities (tachycardia, hypertension) usually represent a normal response to pain. The failure of tachycardia to resolve with adequate analgesia should raise suspicion for blood loss.

The general approach to the assessment of extremity injuries includes evaluation of the following:

Bones and ligaments

Bony deformities are often obvious, but more subtle clues to fractures include crepitus, marked swelling, point tenderness and ecchymosis.

Sprains or ligamentous injuries may be characterized as first-, second-, or third-degree. First-degree sprains are tears of only a few fibers and result in minimal swelling, point tenderness, normal joint motion and stability. Second-degree sprains are more significant tears of the ligament, although not complete disruptions. Signs include more significant swelling, tenderness and functional loss, although joint motion and stability remain normal. Third-degree sprains are complete disruptions of the ligament with marked swelling, tenderness, functional loss, abnormal motion and laxity at the joint.

Muscles and tendons

Rupture of tendons may result from repetitive stress or excessive loading, or from deep lacerations that directly disrupt the tendon. Tendon function should be carefully tested on physical examination. If a tendon is visible in a skin laceration, the tendon should be directly visualized through its full range of motion. *Strains* (injuries to muscle fibers) have a similar classification to sprains. First-degree strains are disruptions of a few fibers and are characterized by mild localized pain exacerbated by stretch. Second-degree strains are more significant, although not complete disruptions, with more marked tenderness and ecchymosis. Third-degree strains are complete disruptions with significant tenderness, ecchymosis and loss of function. Larger muscles, such as the biceps, may display obvious deformities when ruptured.

Nerves and vessels

Care must be taken to assess the neurovascular status distal to an injured extremity. Neurovascular damage may result from direct trauma, from disruption due to a severely displaced fracture or dislocation, or from fracture fragments. Injuries to nerves are more common than vascular injury, and range in severity from *neuropraxia* (secondary to contusion), which results in eventual recovery, to complete disruption or destruction. Complete assessment should include sensory, motor and deep tendon reflex (DTR) examinations. Table 25.7 lists common injuries associated with possible nerve deficits.

Although not as common, vascular injuries are potentially devastating. Complete assessment involves capillary refill time, palpating pulses, and noting color and temperature changes. If pulses are not palpable, a Doppler should be used to confirm flow. Table 25.8 lists common injuries associated with possible vascular deficits.

Table 25.7 Extremity injuries and associated nerve deficits

Injury	Possible nerve deficit
Anterior shoulder dislocation/ fracture	Axillary nerve Musculocutaneous nerve
Humeral shaft fracture	Radial nerve
Fracture of distal third of radius	Radial nerve
Supracondylar fracture of humerus	Median nerve Radial nerve Ulnar nerve
Posterior elbow dislocation	Median nerve Ulnar nerve
Wrist fracture/dislocation	Median nerve
Posterior hip dislocation	Sciatic nerve
Anterior hip dislocation	Femoral nerve
Knee fracture/dislocation	Peroneal nerve Tibial nerve
Proximal fibula fracture	Peroneal nerve

Table 25.8 Extremity injuries and associated vascular deficits

Injury	Possible vascular deficit
Anterior shoulder dislocation/ fracture	Axillary artery
Supracondylar fracture of humerus	Brachial artery
Posterior elbow dislocation	Brachial artery
Knee dislocation	Popliteal artery

Soft tissue (compartments)

Bound by stiff fascial walls, limb compartments are susceptible to dangerously high pressures when there is an increase in volume. When trauma results in muscle swelling or extravasation of blood, there is little room within the compartment to expand. As intracompartmental pressure rises, blood flow to the nerves and muscles decreases and, if unrelieved, muscle necrosis occurs. This process represents *compartment syndrome* and is classically characterized by the five Ps:

- *P*ain
- *P*allor
- *P*aralysis
- *P*ulselessness
- *P*aresthesias

Unfortunately, by the time all of these signs and symptoms are present, permanent damage has usually occurred. The key is to maintain a high index of clinical suspicion. Certain fractures are more commonly associated with compartment syndromes; tibial fracture with anterior tibial artery involvement or supracondylar fracture of the humerus with brachial artery involvement are two examples. The earliest manifestation is pain in the affected extremity followed by paresthesias. Pain is often exacerbated by passive extension of the fingers or passive flexion of the toes.

Regional

Shoulder

Examination of the shoulder begins with inspection and palpation of the clavicle, acromioclavicular and sternoclavicular joints. Deformity, swelling, or tenderness of

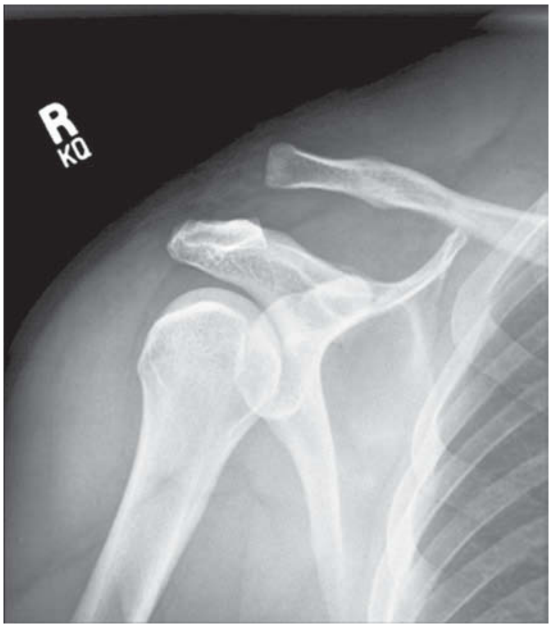


Figure 25.10
Complete (grade 3) acromioclavicular separation. AP radiograph of the right shoulder showing diastasis of the AC joint, with superior displacement of the distal clavicle and widening of the coracoclavicular distance. Courtesy: S.V. Mahadevan, MD.

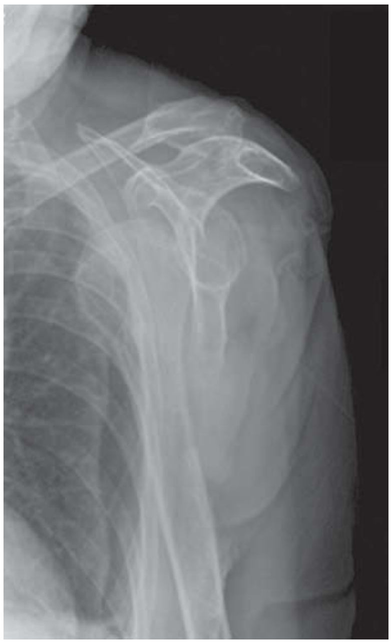


Figure 25.11
Anterior shoulder dislocation. Trans-scapular Y-view of the left shoulder showing the humeral head positioned anterior and inferior to the glenoid. Courtesy: S.V. Mahadevan, MD.

the clavicle may represent a fracture. Most clavicle fractures occur in the middle third of the bone. Superior displacement or prominence of the lateral clavicle is seen with complete (grade 3) acromioclavicular separations (Figure 25.10), whereas incomplete separations (grades 1 and 2) often present only with point tenderness at the joint. Anterior dislocation of the sternoclavicular joint may present with a prominent and easily palpable medial clavicle, whereas posterior dislocation may have a sulcus sign from depression of the clavicle. Tenderness, swelling, or bruising over the proximal humerus may represent a fracture.

The integrity of the glenohumeral joint should be evaluated. Anterior shoulder dislocations (Figure 25.11), which are far more common than posterior dislocations, present with the patient holding the arm fully adducted. There is a loss of the normal rounded contour of the lateral aspect of the shoulder. A simple method to rule out a shoulder dislocation requires the examiner to gently internally and externally rotate the shoulder, followed by asking the patient to place the hand of the injured extremity across his chest on the opposite shoulder. Free rotation of the humeral head is painful and difficult in the presence of a shoulder dislocation, and the ability to perform these maneuvers virtually rules out a dislocation. The musculocutaneous branch of the axillary nerve may be injured in anterior shoulder dislocations, resulting in weakness in shoulder abduction and diminished sensation over the lateral aspect of the shoulder. Patients who have suffered a shoulder subluxation should still demonstrate instability and/or apprehension with examination. Anterior subluxation can be evaluated by placing the affected shoulder at 90 degrees abduction and external rotation. Further external rotation applied by the examiner will elicit an “apprehension sign,” or sensation that the joint is about to dislocate. Posterior instability can be assessed by having the patient lie supine on a table with the shoulder in 90 degrees abduction and just off the side; a force is directed posteriorly along the humerus with the hand or knee, and instability and apprehension is elicited. Inferior instability can be assessed by pulling downward on a relaxed and extended arm, revealing a sulcus sign at the joint.

Examination of the scapula requires palpation along its entire surface. Because significant force is required to fracture the scapula, the mechanism is usually a direct blow or fall from height. Fractures of the scapula may be associated with other serious injuries such as pneumothorax, rib fractures and vertebral compression fractures. As abduction beyond 90 degrees involves scapular rotation, this motion should produce pain in a scapular fracture.

Soft tissue injuries can be acute or chronic, and frequently involve rotator cuff tears. These are disruptions of the muscles that permit shoulder abduction and rotation: subscapularis, infraspinatus, supraspinatus and teres minor. Consequently, patients present with weak and painful active abduction and external rotation, as well as tenderness over the greater tuberosity (the insertion site of supraspinatus). Passive range of motion may be pain-free. In the *drop arm test,* the patient abducts the shoulder to 90 degrees and then is asked to slowly lower the arm. In the presence of a rotator cuff tear, the patient is unable to lower the arm slowly and smoothly.

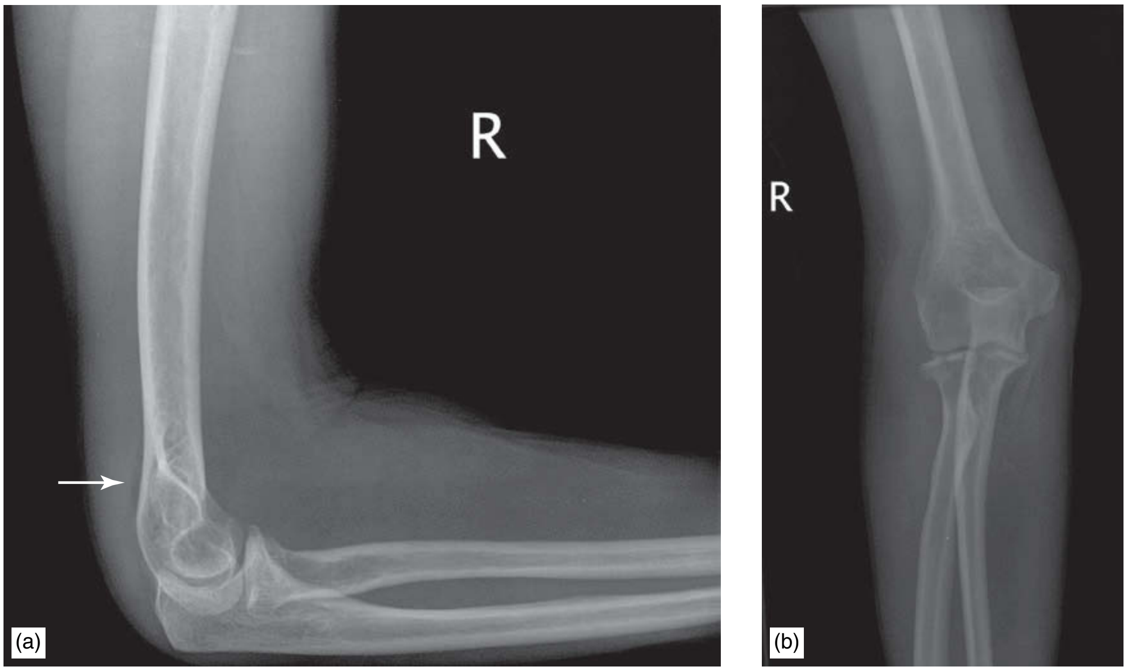


Figure 25.12
Radial head fracture. Lateral (a) and AP (b) X-rays of the right elbow showing a posterior fat pad sign (arrow) indicative of a joint effusion, and a fracture of the radial head. Courtesy: S.V. Mahadevan, MD.

Ruptures of the pectoralis major occur most frequently when weightlifting (e.g., during a bench press). Most occur as disruption away from the insertion at the humeral head. Findings include ecchymosis at the humeral insertion site, loss of the anterior axillary fold, and weakness or pain with shoulder adduction.

Elbow

Deformity at the elbow may represent a fracture or dislocation, and radiographs are needed to differentiate the two. Important clues include tenting of the posterior aspect of the elbow by the olecranon in a posterior dislocation, isolated tenderness of the proximal radius in a radial head fracture, or point tenderness and swelling of the olecranon in olecranon fractures. Any effusion identified either clinically or radiographically in the setting of trauma is concerning for fracture (Figure 25.12).

Supracondylar fractures (Figure 25.13) occur most commonly in children who have fallen on an outstretched hand. Displacement of the distal humeral fracture fragment posteriorly may cause injury to the brachial artery or median, radial and ulnar nerves. It is therefore important to document distal neurovascular findings in patients with a supracondylar fracture. Patients with supracondylar fractures are at risk of compartment syndrome of the forearm, leading to muscle necrosis and contractures of flexor muscles (Volkmann's ischemic contractures). Orthopedic consultation for appropriate disposition is mandatory, with hospitalization, reduction and surgery if the fracture is significantly displaced.

Nursemaid's elbow is a subluxation of the radial head that results from longitudinal traction applied along the radius. This usually occurs when a child's arm is pulled to prevent him from falling or to redirect his path. The child is usually less than 5 years old, and presents with the arm held in passive pronation and dangling to the side. Patients typically refuse to use the affected limb. Nursemaid's elbow is a clinical diagnosis; routine radiographs are not indicated unless a fracture is suspected.

Soft tissue injuries of the elbow most commonly occur at the collateral ligaments. The medial ulnar collateral ligament is injured with a valgus load and is associated with pain along the course of the ligament. The valgus stress test reveals pain and laxity as compared with the unaffected side. It is performed with the patient seated, the elbow flexed at 30 degrees to unlock the olecranon from its fossa, and valgus stress applied by the examiner. The ulnar collateral ligament follows a course from the lateral epicondyle of the humerus posteriorly to the ulna, so injuries result in posterolateral instability. Physical examination must direct a posterolateral force to be accurate, rather than a simple varus stress. One simple test is the "stand-up test." The patient is instructed to stand up from a seated position by pushing against the chair with both arms fully supinated. Pain or instability with this maneuver is a positive test.

Wrist

Examination of the distal radius and ulna may reveal characteristic deformities on inspection. Dorsal angulation of the radius after a fall on an outstretched hand is the typical presentation of a Colles fracture (Figure 25.14), whereas volar angulation represents a Smith fracture. Minimally displaced fractures of either the radius or ulna may present with minimal swelling and point tenderness. A thorough examination of all bony landmarks is essential.

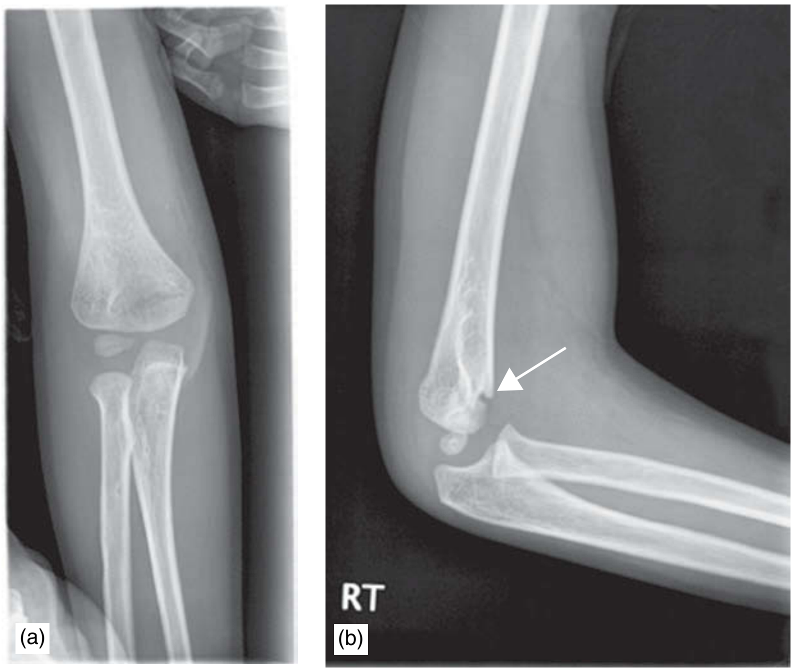


Figure 25.13
Supracondylar fracture. AP (a) and lateral (b) radiographs of the right elbow of a child demonstrating a supracondylar fracture (arrow). Courtesy: S.V. Mahadevan, MD.

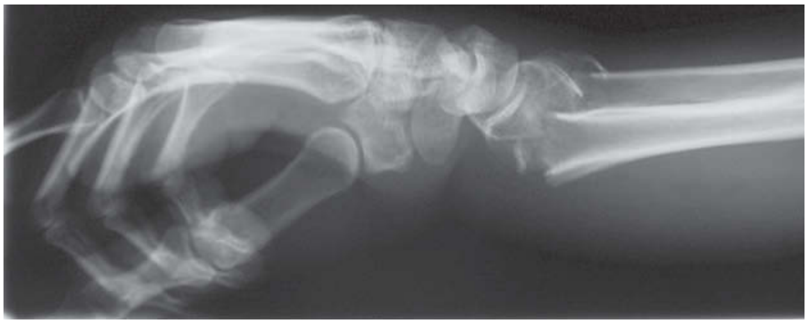


Figure 25.14
Colles fracture. Lateral radiograph showing a fracture of the distal radius, with significant dorsal displacement of the distal fragment. Courtesy: S.V. Mahadevan, MD.

Examination of the carpal bones requires careful palpation of each bone, as fractures may not be immediately apparent on radiographs. This is typical of scaphoid fractures (Figure 25.15), the most common carpal fracture. As missed scaphoid fractures significantly increase the likelihood of avascular necrosis, the physical examination is more important than radiographs. Sensitive signs for a scaphoid fracture include tenderness in the anatomic snuffbox, pain with axial loading of the thumb, or tenderness of the scaphoid tubercle. Clinical suspicion based on either of these findings and a history of falling on an outstretched hand (FOOSH) mandates appropriate splinting and follow-up in 7–10 days for repeat films.

Triquetral fractures are the second most common carpal fracture, often the result of a FOOSH mechanism. These may often only be visualized on a lateral radiograph (Figure 25.16). Patients present with tenderness just distal to the ulnar styloid. Lunate fractures, although less common, are potentially devastating because, like the scaphoid, they carry a significant risk of avascular necrosis (Kienbock's disease). Patients generally complain of centrally located wrist pain after a fall. On examination, tenderness is just distal to Lister's tubercle. As with scaphoid fractures, clinical suspicion alone mandates splinting and orthopedic follow-up.

Ligamentous disruption can also occur between carpal bones, the most common of which causes scapholunate dissociation. Particular attention should be paid for tenderness at the joint, which is immediately ulnar to the anatomic snuffbox. With perilunate (Figure 25.17) and lunate (Figure 25.18) dislocations, the wrist appears deformed and median nerve sensation may be diminished. *Watson's test* for scapholunate instability may reveal pain or subluxation when the patient deviates the wrist in an ulnar to radial direction while the examiner palpates the scaphoid tubercle.

Vascular integrity of the radial and ulnar arteries can be assessed by the *Allen test*. The examiner applies pressure to both arteries and asks the patient to elevate his hand and repeatedly pump his fist. The examiner then releases the radial artery and determines the time required for the blanched hand to return to its normal color. The process is repeated for the ulnar artery. Significant differences in refill time between the two

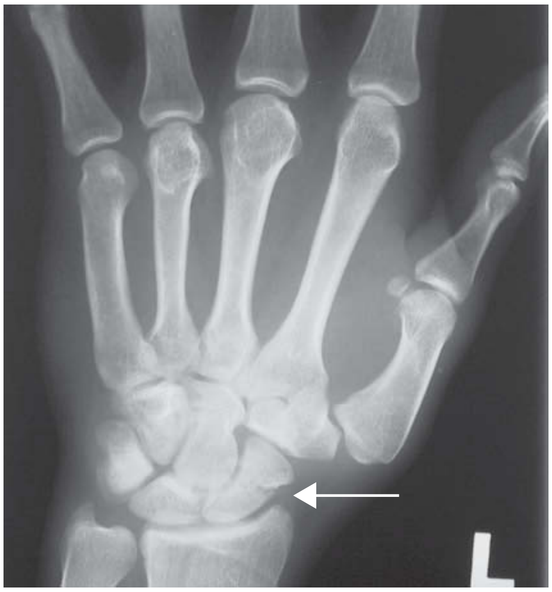


Figure 25.15
Scaphoid fracture. AP X-ray of the left hand showing a fracture through the waist of the scaphoid. Courtesy: S.V. Mahadevan, MD.

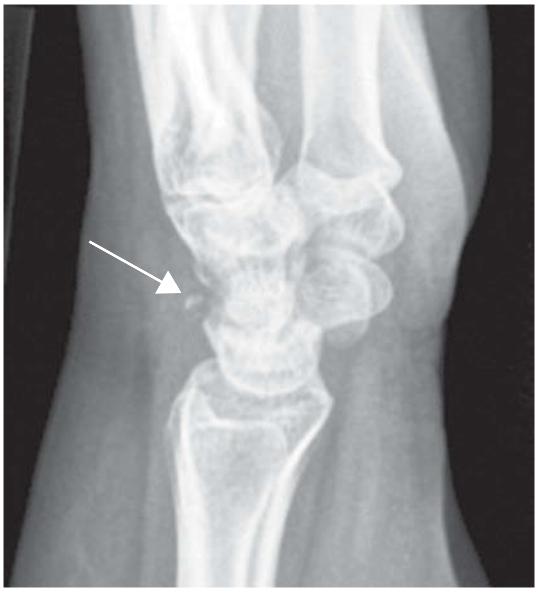


Figure 25.16
Triquetral fracture. Lateral X-ray of the wrist revealing avulsion fracture of the triquetral carpal bone. Courtesy: S.V. Mahadevan, MD.

arteries or between the affected and unaffected hand suggest vascular injury and require consultation.

Hand

Bony deformities are often obvious in fractures and dislocations of the hand. It is important that not only angulation and displacement be noted, but also rotational deformities. All three factors must be addressed in an adequate reduction. Palpation of each metacarpal and phalanx may reveal point tenderness suspicious for a fracture (Figure 25.19).

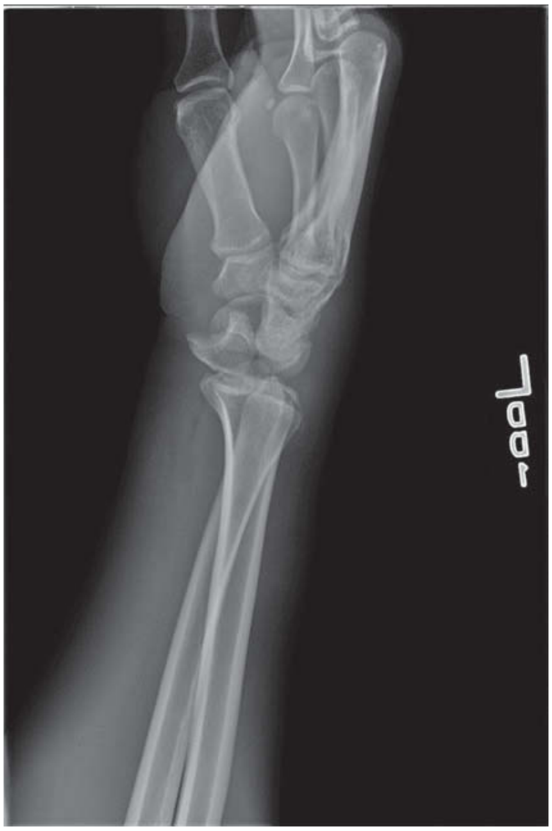


Figure 25.17
Perilunate dislocation. Lateral X-ray of the wrist demonstrates a perilunate dislocation. The lunate appropriately articulates with the distal radius. Courtesy: Gus M. Garmel, MD.

Any injury to the hand should prompt an examination of the sensorimotor function of the median, radial and ulnar nerves (Table 25.3). The sensory examination of the hand is best assessed by two-point discrimination. The patient should be able to distinguish between two discrete blunt points at a minimum distance of 5 mm at the fingertips and 10 mm at the base of the palm. This examination can be performed with a paper clip whose ends have been separated 5 mm.

Tendon injuries may be apparent on initial inspection. Flexor tendon injuries may result in the finger held in relative extension compared with other digits, whereas extensor tendon injuries result in relative flexion (Figure 25.20). Deficits or pain on active range of motion indicate injury to the tendon being assessed. Care must be taken to assess the function of flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) tendons separately. To test FDP function, have the patient flex the distal interphalangeal (DIP) joint. When assessing the FDS, the examiner must isolate the digit by holding all other fingers in extension. Otherwise, adjacent FDS tendons may assist in flexion of the proximal interphalangeal (PIP) joint and mimic normal function.

Hip

The position in which the affected leg is held on presentation can be a significant clue to underlying pathology. Anterior dislocations and femoral neck fractures (Figure 25.21) present

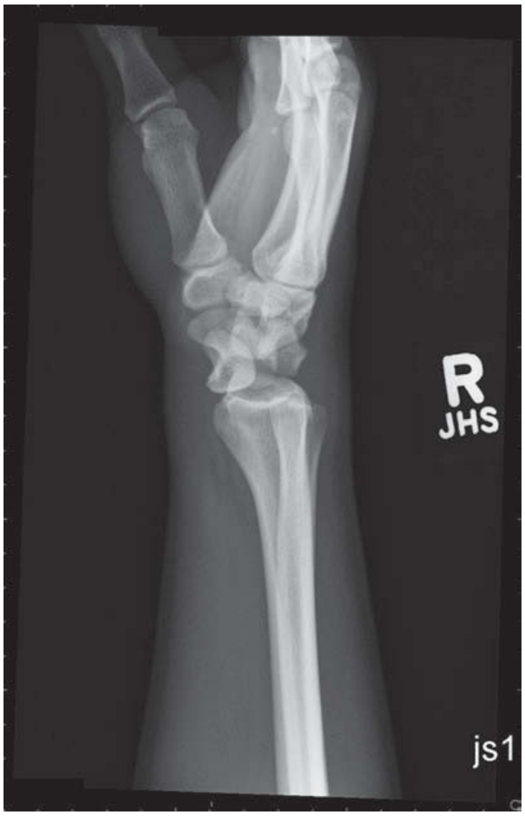


Figure 25.18
Lunate dislocation. Lateral X-ray of the wrist demonstrates anterior (volar) dislocation of the lunate bone. Courtesy: Gus M. Garmel, MD.

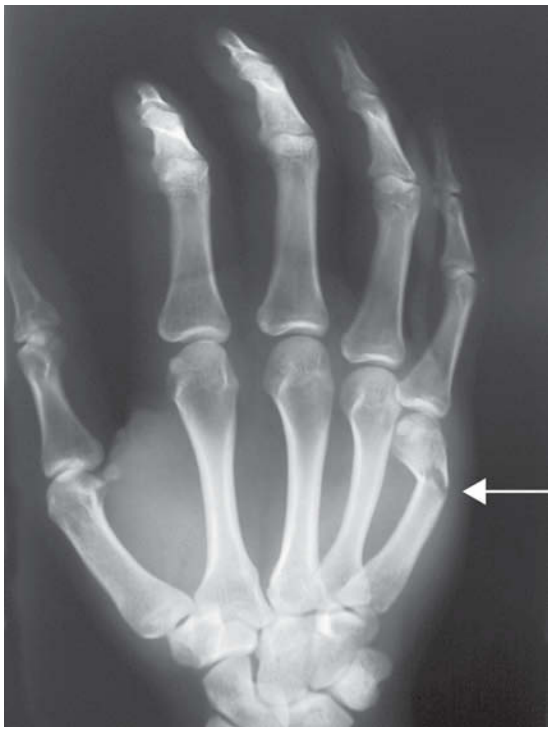


Figure 25.19
Boxer's fracture. AP radiograph showing an angulated fracture through the neck of the fifth metacarpal. Courtesy: S.V. Mahadevan, MD.

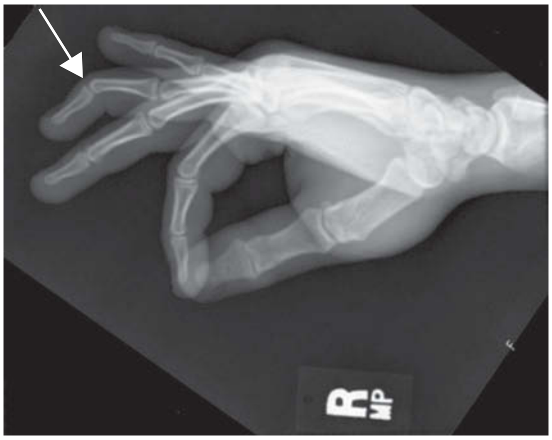


Figure 25.20
Mallet finger. Lateral radiograph of the right hand with a fixed flexion deformity of the distal interphalangeal joint. Courtesy: S.V. Mahadevan, MD.

with the leg abducted, externally rotated, and, in impacted fractures, shortened. Posterior dislocations present with the leg shortened, adducted, and internally rotated. Palpation may reveal tenderness at the site of fracture or a dislocated femoral head. It is important to assess range of motion and stability with respect to flexion/extension, abduction/adduction, and internal/external rotation.

The hip is also a site for possible acute chondral injury from a fall, particularly in younger patients who are at lower risk for fracture. Patients with vague joint pain and an unremarkable examination should be assured follow-up for persistent hip pain, as the loss of articular cartilage can lead to disabling hip pain.

Soft tissue injuries at the hip are most commonly strains or ruptures of the myotendinous units: adductors, flexors, or hamstrings. The clinician should examine for ecchymosis, deformity, weakness or pain with muscle activation to suggest the site of injury.

Knee

Asymmetry of the knees, particularly loss of the peripatellar groove, can indicate a joint effusion resulting from meniscal or ligamentous disruption. Palpation for joint effusion includes:

1. Testing for a fluid wave by tapping the lateral aspect of the knee while simultaneously compressing the medial and superior aspects
2. Ballottement of a patella "floating" in an effusion by pressing against the femoral condyle and eliciting a tapping sensation

Additional landmarks important for palpation are the patella, fibular head (tenderness indicates suspicion for fracture), and the joint line (tenderness is suspicious for meniscal or collateral ligament injury). Although an effusion may distort the anatomy of the affected knee, the position of the patella should be compared with that of the unaffected knee to rule out patellar dislocation, which almost always occurs laterally, or patellar fracture (Figure 25.22).

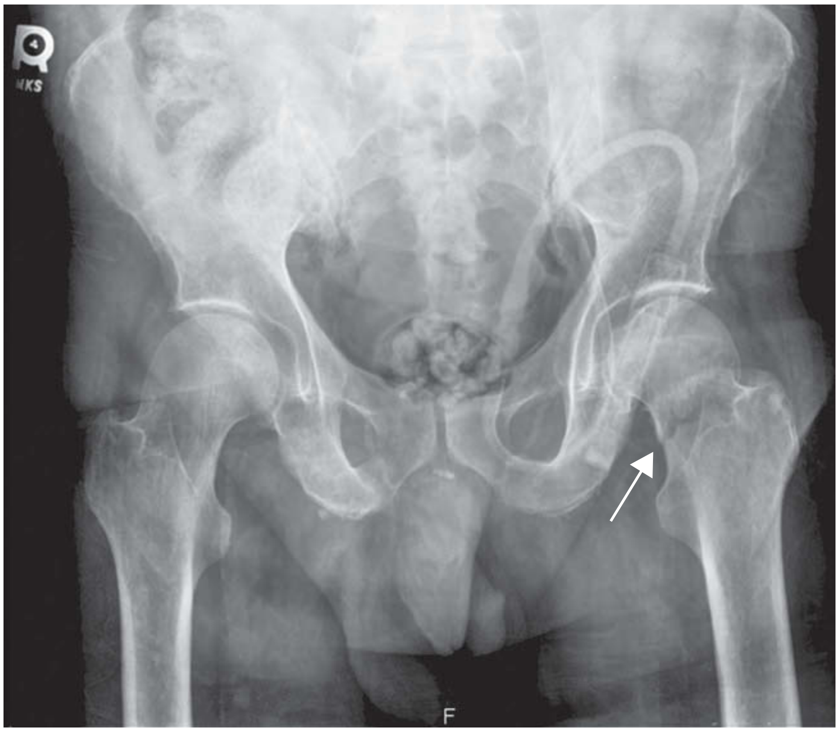


Figure 25.21
Left femoral neck fracture. AP X-ray of the pelvis demonstrating a transcervical fracture of the left femoral neck. Courtesy: S.V. Mahadevan, MD.

The anterior cruciate ligament (ACL) is commonly injured when the foot is planted and a lateral force or torsion is applied to the knee. It can be readily assessed by two maneuvers. The *Lachman test* (Figure 25.23) is performed with the patient supine and the knee flexed 20–30 degrees. The examiner grasps the distal femur with one hand and the proximal tibia with the other hand. The lower leg is given a brisk forward tug in an attempt to identify a discrete end point. A positive test (indicative of injury) occurs when no end point is appreciated or there is increased anterior translation of the tibia relative to the unaffected side. The *anterior drawer test* (Figure 25.24) is performed with the patient supine and the knee flexed to 90 degrees. Both hands are placed around the proximal tibia with the thumbs approximated at the anterior tibial plateau. The examiner quickly pulls anteriorly, without rotation, and feels for a discrete end point. The test is positive if no discrete end point is reached, especially compared with the unaffected side.

Posterior cruciate ligament (PCL) stability is commonly assessed by the *posterior drawer test*. This test is performed with the patient supine and the knee flexed to 90 degrees. There are two different ways it may be performed. The first is the opposite of the anterior drawer test. Absence of a discrete end point with posterior force applied to the tibia is considered positive. The second approach is positive if anterior force applied to the tibia corrects posterior subluxation or "sag" of the affected knee.

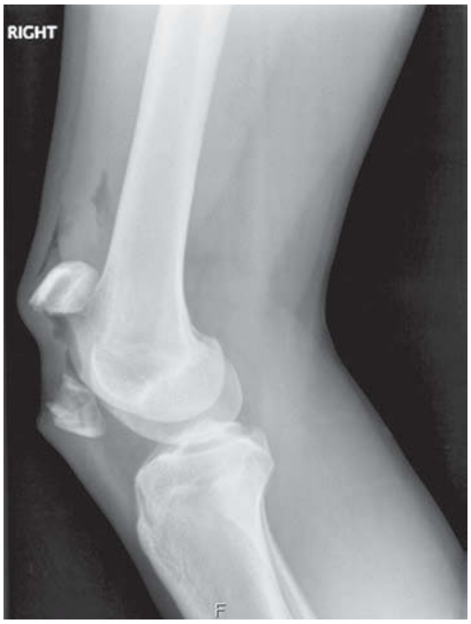


Figure 25.22
Patella fracture. Lateral X-ray of the knee with a transverse fracture of the patella and significant retraction of the fracture fragments. Courtesy: S.V. Mahadevan, MD.

Integrity of the menisci is assessed by the McMurray and Apley compression tests. The *McMurray test* is performed with the patient supine and the examiner grasping the medial aspect of the affected knee with one hand and the patient's heel with the other hand. A valgus force is generated and the tibia internally rotated as the knee is moved from a fully flexed position to full extension. The test is repeated while externally rotating the tibia. Any

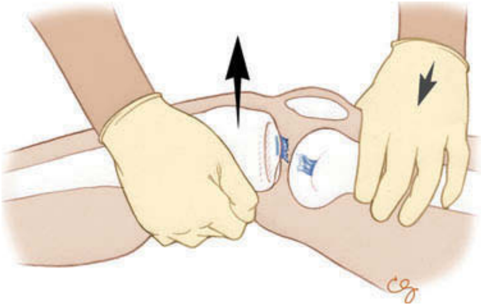


Figure 25.23
Positive Lachman test. © Chris Gralapp.

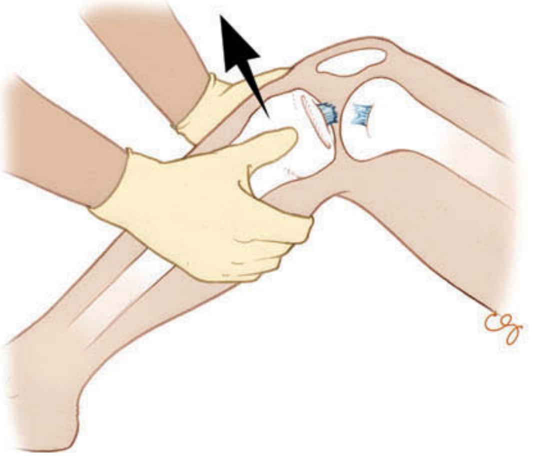


Figure 25.24
Positive anterior drawer test. © Chris Gralapp.

"popping" or pain along the joint line is considered a positive test. The *Apley compression test* (Figure 25.25) is performed with the patient prone and the knee flexed to 90 degrees. A downward force is generated along the long axis of the tibia while simultaneously externally rotating it. If pain is increased, the test is positive.

Injuries to the collateral ligaments are assessed by stress tests. The *valgus stress test* is performed with the patient supine and the knee in 20 degrees of flexion. With one hand on the lateral aspect of the knee and the other on the foot, the examiner gently abducts and externally rotates the lower leg. Increased laxity compared with the unaffected side is considered a positive test for medial collateral ligament (MCL) injury. In the *varus stress test,* the examiner adducts and internally rotates the lower leg to assess the stability of the lateral collateral ligament (LCL).

Regardless of the maneuver attempted to diagnose ligamentous injury, pain and effusion may make adequate examination of the knee impossible. Analgesia prior to examination is important. If an adequate examination cannot be performed, the patient should be treated conservatively with a knee immobilizer and/or crutches. Expeditious follow-up for re-examination (once the swelling has decreased) should be arranged.

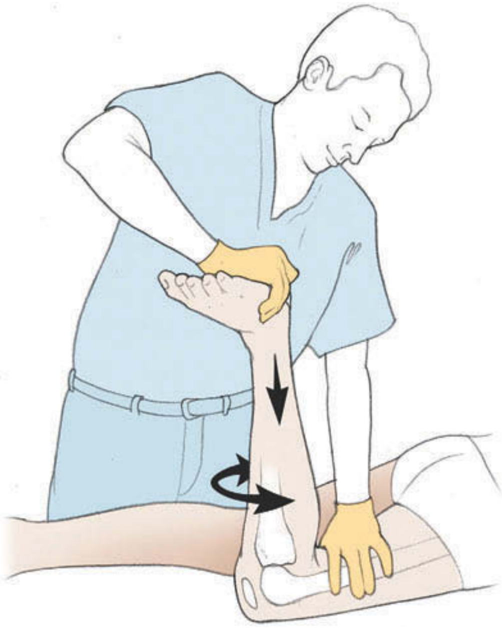


Figure 25.25
Apley compression test. © Chris Gralapp.

Ankle

Sprains and fractures of the ankle may present with marked swelling and point tenderness. The major ligaments of the ankle are assessed by placing stress on them in an attempt to elicit instability. These maneuvers are frequently limited by soft tissue swelling and pain. Therefore, adequate analgesia and immediate application of rest, ice, compression and elevation (RICE) assists in the ankle examination. If the examination is questionable or inadequate, and orthopedic consultation is not indicated, then conservative treatment and follow-up for repeat assessment (once swelling and pain has subsided) is appropriate.

The *anterior drawer test* (Figure 25.26) assesses the stability of the anterior talofibular ligament. The examiner exerts a downward force on the tibia while simultaneously attempting to "lift up" the foot while grasping behind the heel. A significant difference from the unaffected side (>2 mm) or dimpling of the anterior skin (suction sign) is considered positive.

The *talar tilt test* (Figure 25.27) may also be used to assess the integrity of the anterior talofibular ligament. The examiner plantar flexes and inverts the patient's ankle; an increase in laxity compared with the unaffected side is considered a positive test.

Any tenderness over the medial aspect of the ankle warrants an examination of the proximal fibular head to assess for a possible Maisonneuve fracture. A result of external rotation of the ankle, the Maisonneuve fracture is a spiral fracture of the fibular head found in association with fractures of the medial malleolus or deltoid ligament injury (Figure 25.28).

Integrity of the Achilles tendon is assessed by the *Thompson test*. The examiner squeezes the calf with the patient prone. If the foot does not plantar flex, the test is positive for Achilles tendon disruption.

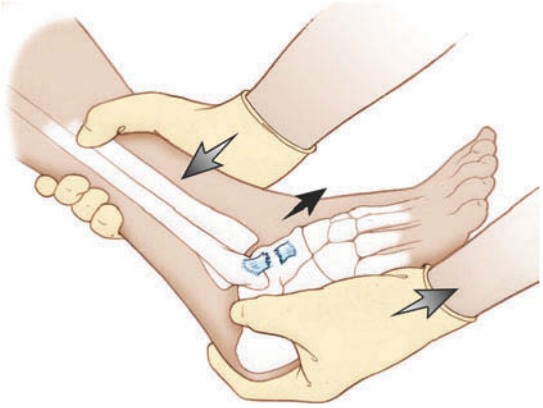


Figure 25.26
Positive anterior drawer test. © Chris Gralapp.

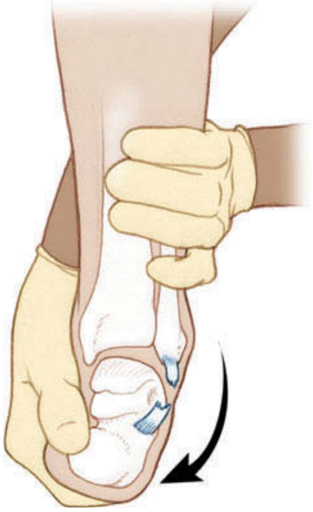


Figure 25.27
Positive talar tilt test. © Chris Gralapp.

Foot

Care must be taken to assess the foot separately from the ankle. Although the deformity of dislocated metatarsophalangeal or interphalangeal joints may be obvious, fractures may present with only minimal swelling and point tenderness. Stress fractures occur most commonly in the second and third metatarsals and often cannot be seen on initial radiographs. Injury to the base of the fifth metatarsal may be seen with inversion injuries of the ankle (Figure 25.29). Injuries to the calcaneus are associated with a fall onto the feet or with a severe twisting mechanism, resulting in heel pain and soft tissue swelling. In falls that produce axial loading, a careful search for coincident lower extremity injuries and compression fractures of the thoracolumbar spine should occur. The Lisfranc injury (Figure 25.30) is a tarsometatarsal dislocation that occurs when

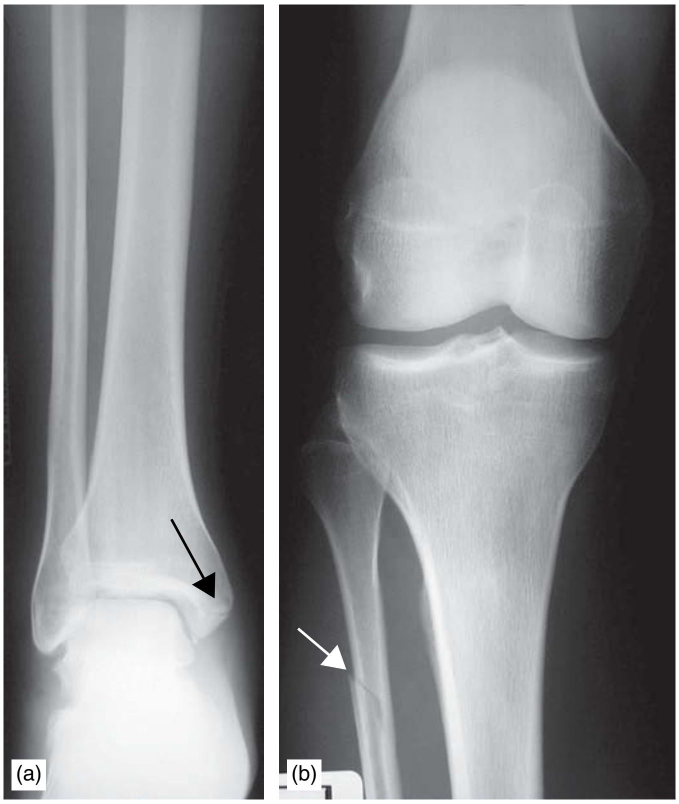


Figure 25.28
Maisonneuve fracture. (a) AP radiograph of the ankle showing a small avulsion fracture of the medial malleolus, with widening of the mortise joint and (b) spiral fracture of the proximal fibula in the same patient. Courtesy: Kathryn Stevens, MD.

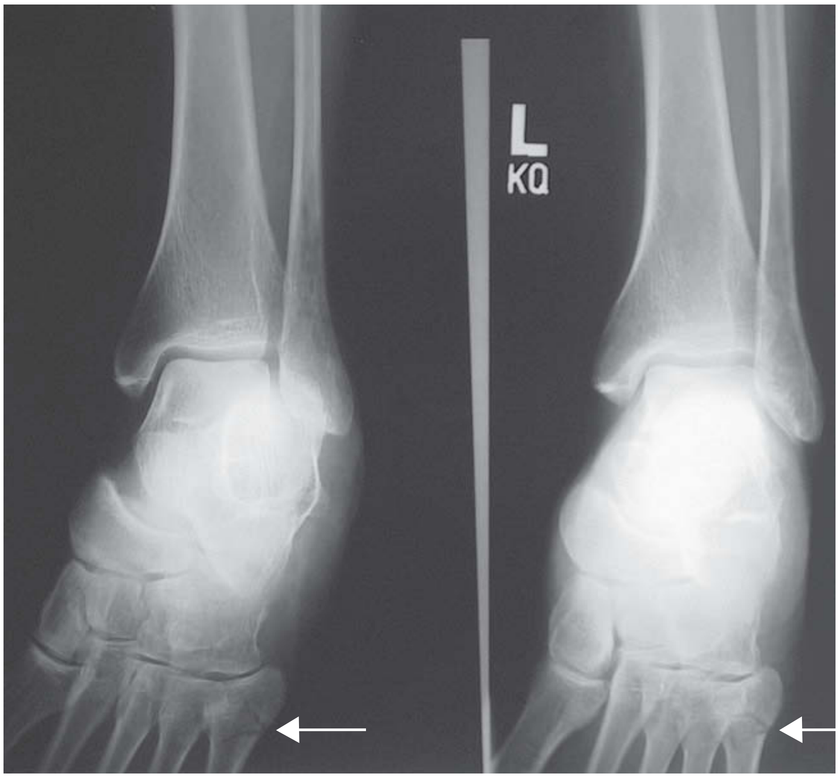


Figure 25.29
Fifth metatarsal fracture. AP views of the left ankle showing a fracture of the base of the fifth metatarsal. Courtesy: S.V. Mahadevan, MD.

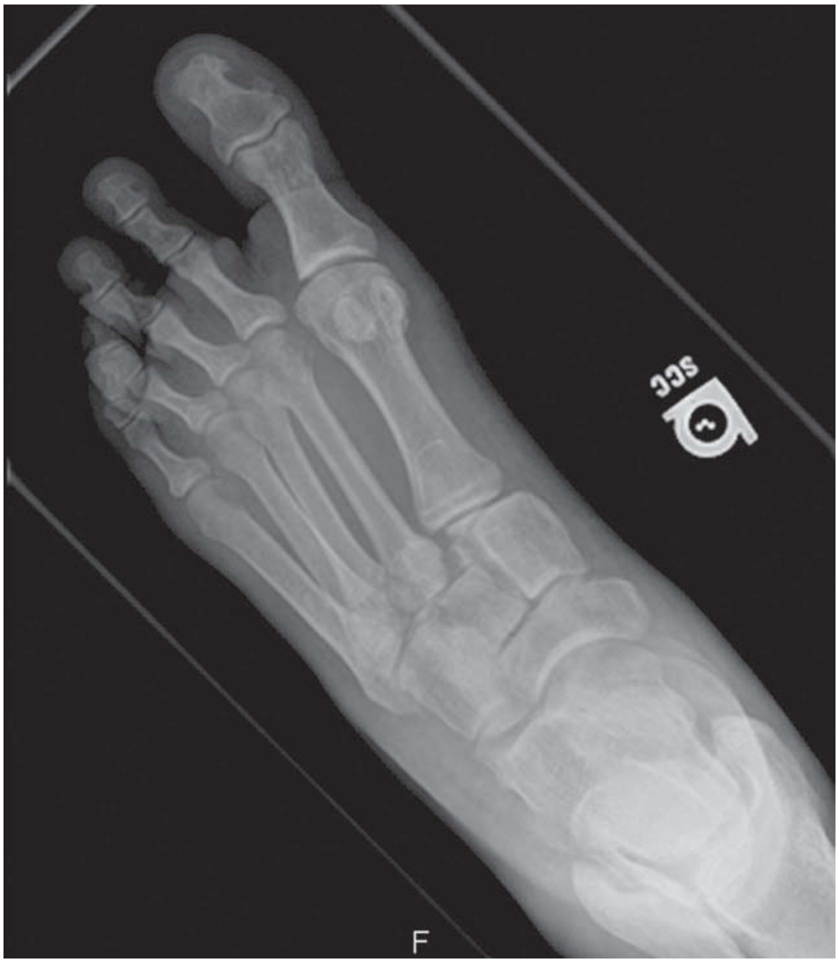


Figure 25.30
Lisfranc fracture-dislocation. AP oblique radiograph of the left foot showing fractures through the bases of the second and third metatarsals, and lateral dislocation of the metatarsals. Courtesy: Kathryn Stevens, MD.

there is a direct axial load on a foot that is plantar flexed. Radiographs are often negative, although a fracture of the base of the second metatarsal is pathognomonic. Any examination revealing tenderness at the tarsometatarsal joint or base of the second metatarsal should prompt the clinician to consider a Lisfranc injury.

Diagnostic testing

Radiographs

Radiographs are important in evaluating extremity trauma. By revealing the nature of a fracture or dislocation, they assist in decisions regarding the need for reduction or operative repair, the length of time required for immobilization, or potential complications during rehabilitation. However, indiscriminate use of radiographs for extremity pain leads to higher health care costs, unnecessary radiation exposure and increased length of stay in the ED. Therefore, the challenge for the emergency physician is to order radiographs *when appropriate.* Research on judicious film utilization in patients with extremity injuries continues to evolve. The Ottawa ankle, foot and knee rules (Table 25.9) represent guidelines derived from high-quality evidence in large studies.

Once the decision is made to order radiographs, the clinician should follow several important principles. First, it is important to formulate a presumptive diagnosis based on history and examination. Some injuries, such as a scaphoid fracture or posterior shoulder dislocation, may require special visualization techniques not included in a normal series. Also, injuries such as a scaphoid fracture, non-displaced radial head fracture, and metatarsal stress fracture may not be apparent on initial films. If these injuries are suspected, patients should be treated as though they have such an injury despite negative radiographs, and appropriate follow-up for repeat imaging should be arranged. The use of weight-bearing films to diagnose a Lisfranc injury or acromioclavicular (AC) separation is often not feasible in a busy ED; conservative treatment and follow-up radiographs are appropriate for these patients.

The clinician should also ensure that the radiographs taken are adequate to visualize structures of concern. At least two views, taken perpendicular to one another, are necessary for most bones and joints. Sometimes a third (oblique) view may be necessary. A fracture of a long bone is often associated with a nearby dislocation or additional fracture along the shaft; therefore, films should include the joints above and below the injury. Finally, if any reduction has been attempted, post-reduction films should be ordered after the extremity is splinted to assess adequacy of reduction and identify small fractures that might have been initially obscured.

Table 25.9 Ottawa rules for extremity radiographs

Ottawa ankle rules
Order ankle radiographs only if the patient has ankle pain and either of the following:

1. Inability to bear weight for four steps, both immediately and in the ED
2. Bone tenderness at the posterior edge or distal 6 cm of either medial or lateral malleolus

Ottawa foot rules
Order foot radiographs only if the patient has foot pain and either of the following:

1. Inability to bear weight for four steps, both immediately and in the ED
2. Bone tenderness at the navicular or base of the fifth metatarsal

Ottawa knee rules
Order knee radiographs only if the patient has knee pain and any of the following:

1. Age ≥ 55 years
2. Isolated tenderness of the patella
3. Tenderness over the head of the fibula
4. Inability to flex knee to 90 degrees
5. Inability to bear weight for four steps, both immediately and in the ED

Description of fractures

Once a fracture is visualized on the radiograph, it is essential that the emergency physician communicate to a consultant the location and nature of the injury. An accurate verbal description will enable the consultant to make an informed decision regarding disposition and indications for operative repair. The terms below are commonly used by orthopedists in fracture description and therefore facilitate communication.

Exposure

Perhaps the most important description, and therefore the first that should be mentioned, is whether the fracture is open or closed. An *open fracture* is exposed to the environment and often requires parenteral antibiotics as well as operative repair. The bone may be obviously protruding through the skin, or there may be only a small laceration overlying the fracture. It is therefore important to clean and thoroughly examine the extremity for skin integrity. A *closed fracture* is present when the skin overlying a fracture is intact.

Location

Description of fracture location can involve both general anatomic terms and landmarks specific to a particular bone. Fractures of long bones can be described as being either *midshaft* or in the *proximal or distal thirds.* Describing the length of the fracture from either the proximal or distal end provides additional information.

Specific anatomic descriptions exist for a variety of fractures; often this nomenclature is used because these fractures may require operative repair or because of specific associated complications. Examples include *intertrochanteric* and *femoral neck* fractures of the femur, and *supracondylar* fractures of the humerus.

Orientation

Figure 25.31 illustrates nomenclature based on the direction of the fracture line. A *transverse fracture* runs perpendicular to the long axis of the bone. *Oblique* fractures run at an angle to the long axis of the bone, usually

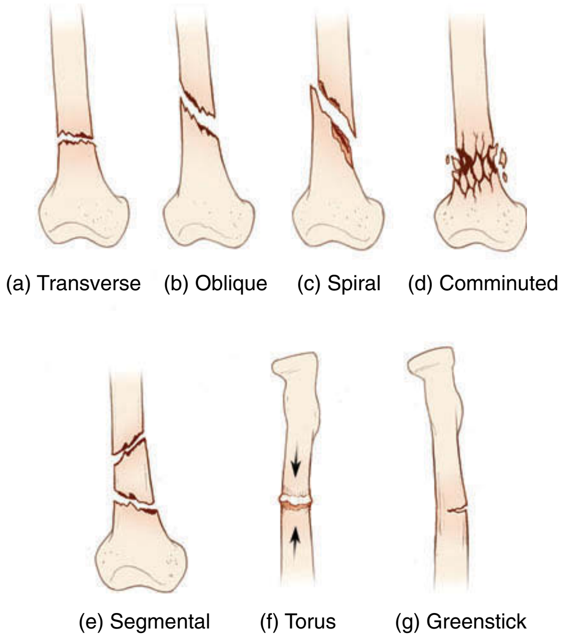


Figure 25.31
Fracture orientation. © Chris Gralapp.

between 45 and 60 degrees. A *spiral* fracture results from torsion on the bone. A *comminuted* fracture consists of more than two fracture fragments, often in a "shattered" pattern. A *segmental* fracture consists of a single free-floating fracture fragment between two fracture lines.

Two fracture patterns found in pediatric populations deserve special mention. A *torus* fracture is demonstrated by "buckling" of the bone cortex, whereas a *greenstick* fracture is an incomplete fracture with disruption of only one cortical aspect on the radiograph.

Displacement

Displacement refers to the amount of offset of a fracture fragment expressed in millimeters or percent. It may be modified by the anatomic direction in which the distal fragment is displaced. For example, in Figure 25.32a, the tibia fracture is 50% laterally displaced.

Separation/shortening

Separation refers to the distance by which two fragments have been pulled apart. Shortening refers to the distance by which the bone's length has been reduced. Shortening occurs as a result of impaction of one fragment into another, or as a result of complete displacement allowing one fragment to "slide over" the other. It is measured in millimeters or centimeters.

Angulation

Angulation describes the relationship between the long axes of the respective fracture fragments. The direction of angulation is determined by the apex formed by these two axes; Figure 25.32b demonstrates a fracture with 30 degrees of lateral angulation. Figure 25.32c demonstrates a fracture with 30 degrees of medial angulation, 100% displacement, and shortening of an undetermined length.

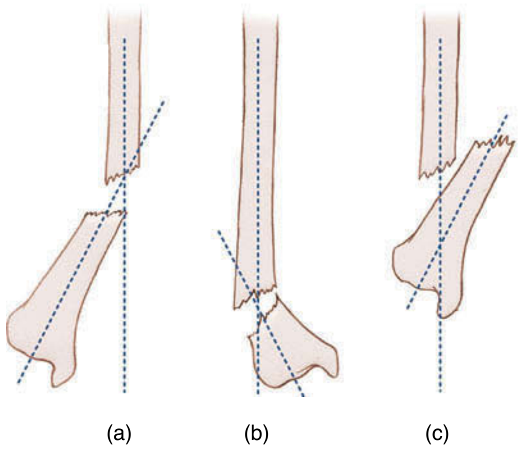


Figure 25.32
Fracture displacement and angulation. © Chris Gralapp.

Joint involvement

The clinician must pay particular attention to fractures located near any joint. A fracture that enters the joint is termed *intra-articular* and deserves special attention by the consultant. In addition, fractures near a joint may be associated with a dislocation. Such fracture–dislocations often require operative repair; therefore, joint alignment should always be assessed prior to communicating with a consultant.

General treatment principles

As with any patient presenting to the ED, initial assessment of extremity trauma begins with the ABCs (airway, breathing, circulation). In isolated extremity trauma, circulation deficits, indicated by diminished or absent pulses, may be the most worrisome initial finding. If an obvious deformity is present, immediate reduction with appropriate analgesia should be performed in an attempt to restore circulation. Once a patient can answer questions reliably and clinical suspicion for other injuries "masked" by the pain of the extremity injury is sufficiently low, treatment can be focused on the affected limb.

Analgesia should be administered as soon as possible to patients who are hemodynamically stable. Options include local anesthesia, regional nerve blocks and parenteral analgesia. In cases requiring extremely painful maneuvers such as reduction, procedural sedation should be used. Oral analgesia should never be given until the need for immediate operative repair or procedural sedation is ruled out.

Fractures and dislocations can undergo reduction by the emergency physician, depending on individual proficiency. Most emergency physicians are comfortable reducing the majority of dislocations, but any reduction with which the clinician is not familiar or that is unsuccessful requires orthopedic consultation. *Fractures or dislocations with signs of neurovascular compromise should undergo emergent reduction in the ED,* even before radiographs are obtained.

The majority of extremity injuries require immobilization, especially following reduction. Appropriate immobilization reduces pain by decreasing movement, inflammation, and the likelihood of bleeding. Casts are rarely applied in the ED, as patients generally present after an acute injury, and continued swelling confined by a hard cast may lead to compartment syndrome. Therefore, the preferred method of immobilization in the ED is non-circumferential splinting. Individual injuries require specific splinting techniques; a summary of common injuries and appropriate immobilization is listed in Table 25.10.

Several general principles apply to patients with extremity trauma. RICE (rest, ice, compression, elevation) remains effective in reducing swelling and discomfort for the patient, and should be initiated in the ED. Ice should never be applied to exposed skin; a towel placed underneath the ice pack prevents skin damage. The extremity should be elevated (above the level of the heart, if possible), and the use of gentle compression with an elastic bandage augments venous and lymphatic drainage.

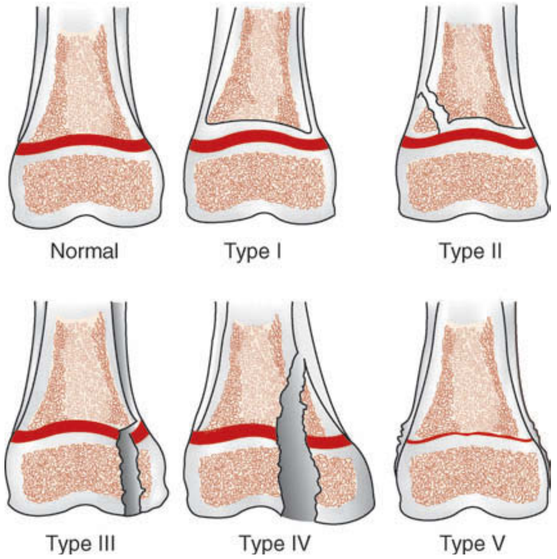


Figure 25.33
Salter-Harris classification. Reproduced from Mandavia DP, *Color Atlas of Emergency Trauma*, Cambridge University Press, Cambridge, 2003.

Table 25.10 Extremity injuries and recommended immobilization

Injury	Immobilization
Shoulder dislocation	Sling and swathe
Rotator cuff tear	Sling and swathe
Acromioclavicular joint sprain	Sling
Clavicle fracture	Sling
Elbow dislocation	Long arm posterior splint
Supracondylar fracture	Long arm posterior splint
Radial head fracture	Sugar tong splint
Olecranon fracture	Long arm posterior splint
Subluxation of radial head	No post-reduction immobilization required
Midshaft ulnar fracture	Long arm posterior splint
Radial and ulnar fracture	Long arm posterior splint
Wrist fracture	Long arm posterior or sugar tong splint
Navicular fracture	Short arm thumb spica splint
Thumb metacarpal fracture	Short arm thumb spica splint
Metacarpal fracture	Ulnar gutter or radial gutter splint
Metacarpophalangeal joint dislocation	Short arm posterior splint
Ulnar collateral ligament tear	Short arm thumb spica splint
Phalangeal tuft fracture	Aluminum splint
Proximal phalanx fracture	Short arm posterior splint
Middle phalanx fracture	Aluminum splint or dynamic splinting
Interphalangeal joint injury	Aluminum splint

Adapted from Hamilton GC (ed). *Emergency Medicine: An Approach to Clinical Problem-Solving,* 2nd ed. W.B. Saunders, Philadelphia, PA, 2003.

Special patients

Pediatric

Children present with a different set of injuries following extremity trauma, because their developing bones are more pliable. Unique fractures such as torus, buckle and greenstick have already been discussed. Of particular concern are fractures involving the growth plate, as they may result in lifelong morbidity. Injuries to the growth plate are classified according to the involvement of metaphysis, epiphyseal plate and epiphysis.

The Salter-Harris classification consists of five different types of growth plate fractures based on the location of the injury (Figure 25.33). In general, the higher the number, the worse the prognosis. *Salter Type I* fractures are through the epiphyseal plate. *Salter Type II* fractures are the most common and involve a fracture of the metaphysis with extension through the epiphyseal plate (Figure 25.34). *Salter Type III* fractures extend from epiphysis into the epiphyseal plate. *Salter Type IV* fractures involve a fracture through the metaphysis, epiphysis and epiphyseal plate. *Salter Type V* fractures are crush injuries to the epiphyseal plate, and are most common in the knee and ankle.

Any joint tenderness in pediatric patients should be treated conservatively, as they are more likely to suffer growth plate fractures than ligament sprains. Even in the setting of negative radiographs, the clinician should

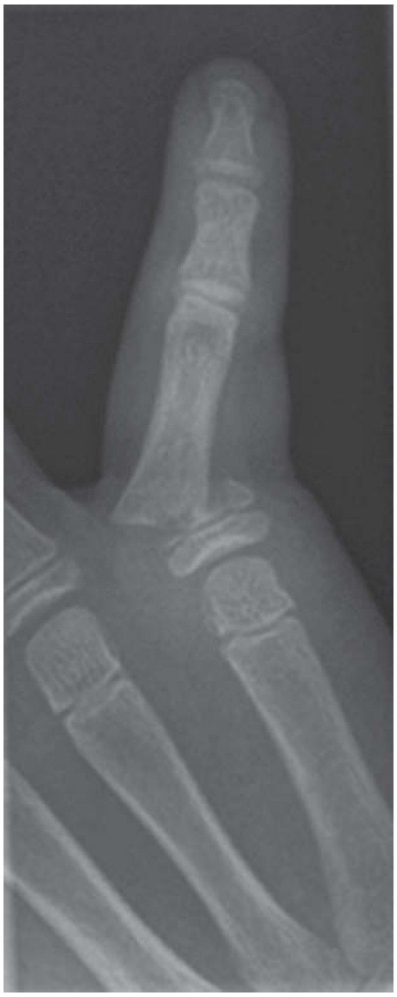


Figure 25.34
Salter-Haris type II fracture. AP radiograph of the fifth digit demonstrating a type II Salter-Harris fracture through the base of the fifth proximal phalanx. Courtesy: Kathryn Stevens, MD.

maintain a low threshold to treat with immobilization and obtain appropriate orthopedic follow-up for a presumed Salter-Harris fracture. In fact, given the presence of multiple ossification centers (six alone in the elbow), the clinician may actually "over-read" a fracture where there is not one present. If the emergency physician is unfamiliar with pediatric radiographs or a radiologist is not available, one option may be to obtain plain films of the unaffected joint for comparison.

Elderly

Elderly patients are likely to have osteoporosis and sustain fractures with even minimal trauma. Falls are a source of considerable morbidity. The threshold for ordering radiographs should be low. Elderly patients may have substantial difficulties wearing splints and using crutches. Social support considerations and temporary care arrangements may become necessary. Beware of occult hip fractures in the elderly patient with negative radiographs but significant pain on weightbearing.

Disposition

Although it is impractical to provide an exhaustive list of injuries requiring consultation for possible admission, in general, the following injuries meet such requirements:

1. Open fractures
2. Open joint injuries
3. Vascular injuries
4. Hip fractures and dislocations
5. Compartment syndrome
6. Dislocations or displaced fractures that cannot be reduced in the ED

Any injury with which the clinician is not familiar or comfortable mandates at least a phone consultation to secure adequate guidance and follow-up. Depending on the nature of the injury, the patient may require immobilization, non-weightbearing status, or weightbearing as tolerated. Follow-up may be with the primary care physician, orthopedic specialist, or hand surgeon.

As discussed earlier, RICE is the mainstay of treatment for extremity injuries and should be continued at home. Although rest is essential, assistive devices such as crutches should be used to ensure that a patient does not become bed-bound. Resumption of activities of daily living as early as appropriate is an important consideration.

Pain reduction to a tolerable level, not pain elimination, is a realistic goal while in the ED. Outpatient narcotics may be needed to maintain adequate analgesia. Patients should be instructed to return for signs of neurovascular compromise, infection, pleuritic chest pain suggesting pulmonary embolism, severe pain not controlled by appropriate analgesia, or a splint that feels too tight.

Pearls, pitfalls and myths

The following constitute important pitfalls related to the management of a patient presenting with extremity injury.

- Failure to warn patients that some hairline or non-displaced fractures may not become apparent on radiographs until 7–10 days later
- Failure to repeat radiographs when the affected area is inadequately visualized
- Missed open fracture because the extremity is not inspected or cleaned adequately to reveal an overlying laceration
- Missed foreign bodies due to inadequate irrigation and exploration
- Missed tendon injury due to inadequate visualization or examination through the entire range of motion
- Failure to document a complete neurovascular examination prior to administering local or regional anesthesia
- Missed injuries due to focusing on obvious trauma without performing a complete physical examination

- Failure to update tetanus immunization status in open fractures, or in extremity injuries in which skin integrity is compromised

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26 Eye pain, redness and visual loss

Janet G. Alteveer, MD

Scope of the problem

Eye complaints are estimated to represent 3% of emergency department (ED) visits annually, with conjunctivitis, corneal abrasions and eye trauma accounting for 75% of ocular disorders. Patients may complain of redness, swelling, pain, foreign body sensation, flashing lights, floating spots, visual field defects, blurred vision and/or decreased vision. The diagnoses may be common and benign, such as allergic conjunctivitis, or uncommon and vision threatening, such as acute angle closure glaucoma (AACG), corneal ulcers, or central retinal artery occlusion (CRAO). Careful attention to the history and physical examination helps delineate the problem and define the treatment.

Anatomic essentials

The bony structure of the orbit is formed by a confluence of the frontal, maxillary and zygomatic bones. The walls of the orbit are referred to by their anatomic location: superior, inferior, medial and lateral. The inferior orbital wall is extremely thin and may be fractured following a direct blow to the globe or orbit; this can lead to blood or orbital contents entering the maxillary sinus through the orbital floor. The eyelids, lacrimal gland, and canalicular system (that drains tears into the nasal cavity) make up the adnexal structures of the eye (Figure 26.1).

The globe (Figure 26.2) is divided into two segments: anterior and posterior. The anterior segment includes the cornea, limbal conjunctiva, iris, anterior chamber and lens. The conjunctiva is a thin, transparent mucus membrane that covers the sclera (bulbar conjunctiva) and the inner surface of the eyelids (palpebral conjunctiva). The sclera is a tough layer of collagen and elastic fiber surrounding the entire globe, except the cornea. The sclera gives the eye its white appearance. The cornea is made up of a dense layer of collagen 500–600 µm thick. It consists of five layers including Bowman's and Descemet's membranes. The cornea is the anterior-most aspect of the eye. It is transparent and allows light to be transmitted and focused through the pupil. The iris is a diaphragm anterior to the lens and is responsible for eye color; it has two layers: the stromal and the pigmented layers. The iris has two sets of muscles: the constrictor and the dilator muscles, innervated by separate nerves. The pupil is the circular aperture in the iris that controls the amount of light entering the eye, based on the tone of the iris dilator and constrictor muscles. The trabecular meshwork, located anterior to the iris in a circumferential pattern around the globe, filters and removes the aqueous humor; malfunction can result in elevated intraocular pressure (IOP). The ciliary body, located posterior to the iris in an inner-tube configuration circumferentially around the globe, has two main functions:

1. Production of aqueous humor, a transparent protein-free liquid contained in the anterior and posterior chambers that provides oxygen and nutrients to the avascular cornea and lens.

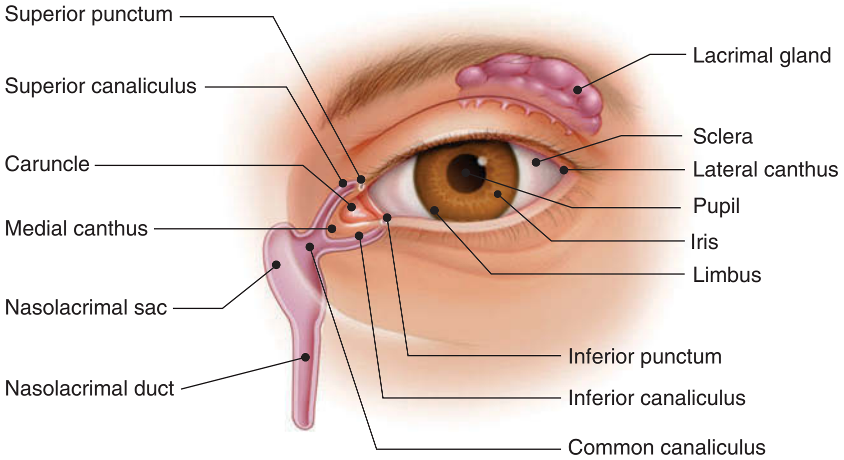

Figure 26.1
View of the eye and adnexal structures. Lacrimal gland is situated superotemporally. The superior and inferior puncta drain into the canalicular system, which eventually empties into the nasal cavity. © Chris Gralapp.

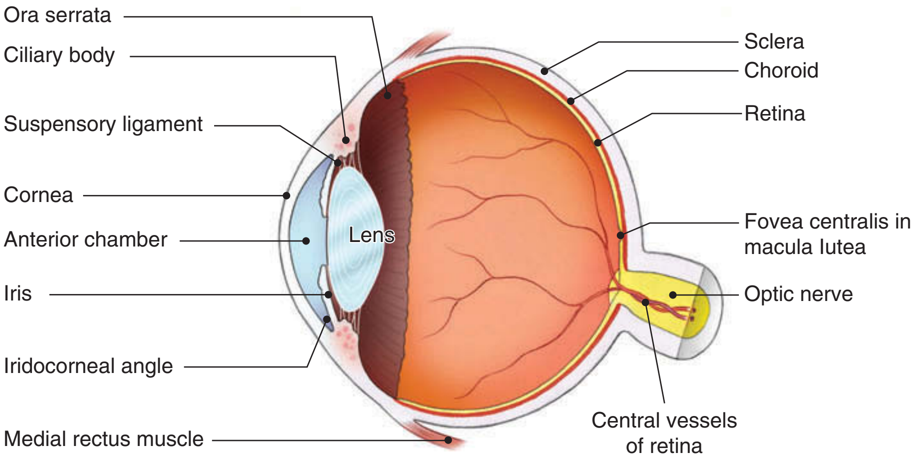

Figure 26.2
The globe in cross-section. The iris diaphragm outlines the margins of the pupil. The anterior surface of the lens abuts the posterior surface of the iris. Zonular suspensory fibers are seen emanating from the ciliary body, adjacent to the iris root. © Chris Gralapp.

2. Control of accommodation by changing the shape of the crystalline lens. The lens sits behind the iris and is responsible for focusing light on the retina. When the ciliary body contracts, the lens thickens, increasing the eye's ability to focus up close. When looking at a distant object, the ciliary body relaxes and the lens becomes thinner, adjusting the eye's focus for distance vision.

The lens thickens and becomes less compressible with age, and is the site of cataract formation. The lens also establishes the anterior boundary of the posterior chamber.

The posterior segment of the globe contains the vitreous humor, choroid, retina and optic nerve. The vitreous humor is a clear hydrogel that fills the vitreous cavity. It is 98–99% water on a collagen framework. The choroid is the vascular structure between the sclera and the retina's pigmented epithelium. It is responsible for cooling the retina, which has a high metabolic rate and the highest blood flow per gram of tissue in the human body. The retina contains 10 layers and is a complex interplay of neuronal elements, supporting tissue, photoactive cells and blood vessels. When the rods and cones absorb light energy, an electrical potential is created. This is then amplified and conducted through the optic nerve to the occipital cortex. The nerve fiber layer of the retina enters the optic disk of the optic nerve, makes a 90-degree turn posteriorly, and exits the globe. The optic disk (optic nerve head) is also referred to as the "blind spot." The optic nerve is a bundle of myelinated nerve fibers that exits the globe through the superior orbital fissure.

The eye is under the influence of both the sympathetic and parasympathetic nervous systems. The sympathetic fibers exit the spinal cord with the T11–T12 outflow. These fibers then travel up the neck in the sympathetic plexus to the nasociliary and long ciliary nerves. These nerves innervate the dilator muscle of the iris. Increased sympathetic tone results in pupillary dilation. Horner's syndrome, an interruption of these sympathetic fibers (by an apical lung tumor or brainstem lesion, for example) results in ipsilateral pupillary constriction (miosis), ptosis and anhydrosis. The parasympathetic fibers travel to the eye via the oculomotor nerve and the ciliary ganglion. These fibers innervate the constrictor muscle of the iris. Increased parasympathetic tone causes pupillary constriction; compression or injury of the oculomotor nerve results in pupillary dilation.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 26.1).

History

Is your eye red?

Injection of the conjunctiva is a finding shared by many infectious, inflammatory, and allergic conditions. Eye redness alone does not distinguish vision-threatening from benign eye conditions.

Does your eye hurt? How would you describe the pain?

A sharp pain accompanied by the sensation of something in the eye is characteristic of corneal foreign bodies, corneal abrasions, infections, or ulcers. Pain made worse by light or accommodation is typical of acute anterior uveitis. Deeper pain often accompanies inflammation of the

Table 26.1 Eye pain, redness and visual loss red flags

History	Concerning diagnosis
Painless visual loss	Central retinal artery occlusion, central retinal vein occlusion, retinal detachment, vitreous hemorrhage
Painful visual loss	Acute angle closure glaucoma, optic neuritis, temporal arteritis
Red eye and visual loss	Acute angle closure glaucoma, corneal abrasion, corneal ulcer, uveitis
Eye pain and photophobia in a contact lens wearer	Corneal abrasion or corneal ulcer
Eye pain and photophobia in an immunosuppressed patient	HSV or herpes zoster
Eye pain, headache and vomiting	Acute angle closure glaucoma
Photophobia	Corneal abrasion, uveitis
Headache and vision loss, age >65 years	Temporal arteritis
Foreign body sensation	Foreign body, corneal abrasion, corneal ulcer, conjunctivitis
Recent eye surgery	Keratitis, endophthalmitis
Examination finding	**Concerning diagnosis**
Ciliary flush (perilimbic erythema)	Acute angle closure glaucoma, uveitis
Afferent pupillary defect	Central retinal artery occlusion, optic neuritis, retinal detachment, temporal arteritis
Red eye, shallow anterior chamber	Acute angle closure glaucoma
Cloudy anterior chamber	Uveitis, hypopyon
Blood in the anterior chamber	Hyphema
Profuse purulent eye discharge	Gonococcal conjunctivitis
Blue tint to sclera	Thinning of sclera in scleritis
Dendrites on fluorescein staining of cornea	HSV, Herpes zoster ophthalmicus
Red eye and lesion on tip of nose	Herpes zoster ophthalmicus
Decreased red reflex	Corneal edema, cataract, hyphema, vitreous hemorrhage
Cranial nerve III palsy	Aneurysm of posterior communicating artery with or without subarachnoid hemorrhage
Swollen eye with decreased or painful extraocular movement	Orbital cellulitis
Red eye and/or discharge, age <1 month	Ophthalmia neonatorum
Swelling, redness of nasolacrimal duct in infant or young child	Acute dacryocystitis

HSV: herpes simplex virus.

sclera or anterior uvea. A deep boring pain made worse by movement of the eye is often present in optic neuritis, whereas severe pain accompanied by a headache on the same side is common in AACG. The presence of pain is significant, as acute conjunctivitis is rarely painful.

Is your eye sensitive to light?

Photophobia (light sensitivity) and increased pain on exposure to bright light suggest anterior uveitis. The pain represents ciliary muscle spasm that may accompany corneal abrasions, blunt trauma to the eye, infections, or inflammatory processes.

How is your vision?

Benign conditions such as bacterial, viral, or allergic conjunctivitis generally do not affect visual acuity. Mild to moderate vision loss may accompany anterior uveitis, scleritis, herpes simplex virus (HSV), or herpes zoster virus (HZV) infections of the eye. Severe deficits in vision may occur with optic neuritis, AACG, CRAO, central retinal vein occlusion (CRVO), central corneal abrasions, or ulcers. A halo around lights is characteristic of acute glaucoma, whereas abnormal color vision is common with optic neuritis.

Have you noticed any discharge?

Purulent discharge is highly suggestive of acute bacterial conjunctivitis. Patients often wake up with their lids and lashes crusted or matted together, and may need to apply warm water to remove the crust and open their eyes. The discharge of gonococcal (GC) conjunctivitis is so profuse that it may "well up" as soon as it is wiped away. By contrast, adenoviral infections produce a copious watery discharge. These patients often describe a clear puddle on their pillow. Chlamydia infection and certain types of allergic conjunctivitis produce a mucoid discharge. Scleritis, anterior uveitis, glaucoma and optic neuritis are not associated with discharge. It is important to differentiate tearing in response to pain or light from discharge.

Any itching?

The presence of itching is a hallmark for an allergic process.

How long has your eye been bothering you?

Patients with a corneal foreign body or abrasion are usually able to tell you the moment their discomfort began. AACG often occurs at night or after emerging from a darkened location – dilation of the pupil in response to decreased light often precipitates the crisis. Bacterial conjunctivitis often occurs overnight. A more indolent onset is typical of inflammatory processes, such as scleritis, uveitis, or optic neuritis.

Is it one eye or both eyes?

Corneal foreign body, abrasion, acute glaucoma, CRAO, CRVO, optic neuritis, periorbital and orbital cellulitis (POC and OC, respectively) generally develop in one eye. Conjunctivitis, either bacterial or viral, typically begins in one eye and spreads rapidly to the other. Inflammatory conditions such as thyroid-related ophthalmopathy, scleritis and allergic conjunctivitis are generally bilateral.

Have you had a recent exposure to someone with a red eye?

Adenoviral infections often present in epidemics, often in schools, dormitories, military barracks and swim clubs. HSV, gonorrhea and chlamydia infections are transmitted via direct contact.

Do you wear contact lenses?

Contact lens wearers are at increased risk of corneal infections and ulcers, such as infectious keratitis or epidemic keratoconjunctivitis (EKC). A “simple” corneal abrasion in a contact lens wearer can progress overnight to a vision-threatening ulcer. Contact lens users are also at risk for rare parasitic infections.

Did you rub your eye?

Rubbing the eye may result in a mechanical corneal abrasion.

Were you hit in the eye?

Traumatic anterior uveitis may develop several days after a blow to the eye. These patients will complain of pain, tearing and photophobia, but may forget to tell you about the injury. Eye trauma accompanied by visual impairment is an ophthalmologic emergency.

What kind of work do you do?

Machinists, drill workers and individuals who do any metal-on-metal work are at risk for metallic ocular foreign bodies. Welders, boaters and skiers, particularly those who do not use proper eyewear, are at risk for ultraviolet (UV) burns to the cornea.

Are you using eye drops?

Allergic conjunctivitis may develop from certain eye preparations, particularly those containing neomycin. Patients on long-term topical steroid therapy are at risk for glaucoma or cataracts.

Have you ever had this before?

Thirty percent of patients with HSV infection will have a recurrent episode. Allergic phenomena and some inflammatory conditions (i.e., scleritis or optic neuritis) can remit and recur.

Do you use eye make-up?

Contamination of mascara and liquid eyeliners may lead to bacterial or viral conjunctivitis. This is especially true with shared products. Patients with such conditions should discard these products.

Have you had recent eye surgery?

Recent eye surgery has been associated with acute keratitis as well as endophthalmitis (a severe vision-threatening infection of the globe).

Do you have any chronic medical conditions?

Immunosuppression is associated with ocular fungal infections. Rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) are associated with scleritis. There is a strong association between multiple sclerosis (MS) and optic neuritis.

Physical examination

Systematic examination of the eye and surrounding structures is critical to narrowing the differential diagnosis.

Vital signs

A complete set of vital signs is essential. Note whether the patient is febrile, tachycardic, or hypertensive. Patients with glaucoma may be dehydrated from vomiting. Fever may be a clue to an infectious process. Hypertension and visual loss may point to a vascular etiology.

Visual acuity

Visual acuity is the vital sign of the eye and *must be measured* on every patient with an eye complaint. It may be obtained by the following methods:

Snellen eye chart

This standard eye chart, consisting of 11 rows of progressively smaller letters, should be used for all patients who can stand and follow simple instructions. Patients who require glasses (other than for reading) should wear them during the examination; this should be noted in the chart. With one eye covered, the patient is asked to identify the largest letters at a distance of 20 feet. This is repeated with the other eye, and then both eyes together. For non-readers, a "tumbling E" chart may be used in which patients indicate with their hands which direction the E faces. A chart with pictures is often used for children and serves the same purpose. The smallest row in which the patient correctly identifies more than half the letters/symbols is noted. The visual acuity is recorded as the number of this row under 20 (e.g., 20/30 OD [right eye], 20/40 OS [left eye], 20/30 OU [both eyes]).

Near chart

For those too sick to stand, a "near card" held 14 inches from the face is used, following the same procedure as with the Snellen chart.

Pinhole testing or ophthalmoscope

If the patient wears corrective lenses but does not have them, pinhole testing or a handheld ophthalmoscope may facilitate visual acuity testing. These methods will also determine if the visual deficit can be corrected mechanically. The patient should be instructed to read the eye chart while looking through the pinhole, or to read a near chart through a handheld ophthalmoscope. The patient can "dial" the ophthalmoscope until the letters appear legible; the visual acuity is then recorded as 20/40 using lens – 10. If either method corrects the visual deficit, the problem is optical (refractive error) and not pathologic.

Gross visual acuity testing

In patients with vision less than 20/400, gross visual acuity should be quantified in terms of the patient's ability to count fingers (CF), detect hand motion (HM) or waving, or perceive light (light perception [LP] or no light perception [NLP]), in that sequence.

Orbit and adnexal structures

Inspect the orbits and the tissue around the eyes for redness, swelling, lesions (e.g., vesicular eruption from HZV infection), and asymmetry. Proptosis (protruding eye) suggests that an orbital infection, inflammation, or tumor is forcing the eye forward (Figure 26.3). This condition is best assessed looking from above the head downward toward the eyes. Keratoconus differs from proptosis in that the cornea is cone-shaped while the globe does not protrude. Examine the eyelashes carefully for crusting, lashes pointing in the wrong direction, or lice.

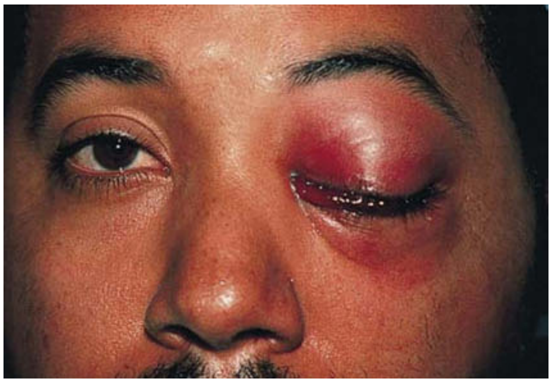

Figure 26.3
Orbital cellulitis presenting with massive swelling, proptosis, erythema and poor ocular motility. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

Ocular motility

It is particularly important to evaluate ocular motility in cases of eye trauma, suspected infection, or double vision. While standing directly in front of the patient, have him hold his head still and follow your finger with his eyes. Study the movement of one eye at a time as you move your finger first up and down, and then from side to side. The extraocular muscles (EOMs) responsible for eye movements are innervated by cranial nerves III, IV and VI (Figure 26.4, Table 26.2).

Entrapment of the inferior rectus muscle within a fracture of the inferior orbital plate may result from a direct blow to the eye. Diplopia may result from orbital infection, tumor, inflammation, compression or ischemia of the individual nerves between the eye and the brainstem, or aneurysms of the circle of Willis. Monocular diplopia is usually the result of problems of the cornea, iris, lens, or retina of that particular eye, and is almost always pathologic.

Pupils

Note the size, shape, and symmetry of the pupils and their reaction to direct light. An asymmetric or teardrop-shaped

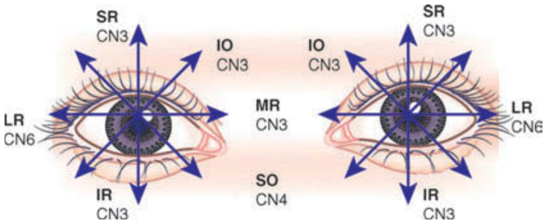

Figure 26.4
Depiction of extraocular muscle innervation by cranial nerves III, IV and VI, and the direction of eye movements that result with contraction of the different extraocular muscles. CN: cranial nerve; SR: superior rectus; MR: medial rectus; LR: lateral rectus; IR: inferior rectus; SO: superior oblique; IO: inferior oblique. Reproduced with permission from Shah SM, Kelly KM (eds), *Principles and Practice of Emergency Neurology*, Cambridge University Press, Cambridge, 2003, p. 199, Figure 18.1.

Table 26.2 Ocular motility

Nerve	Muscle	Lesion
Oculomotor (cranial nerve III)	Superior rectus Inferior rectus Medial rectus Superior oblique	*At rest*: eye deviated down and out *With activity*: unable to look up or adduct
Trigeminal (cranial nerve IV)	Inferior oblique	*At rest*: eye externally rotated (extorted); patients tilt their head to compensate
Abducens (cranial nerve VI)	Lateral rectus	*At rest*: eye slightly adducted *With activity*: unable to abduct

pupil following trauma may indicate injury to the iris or a ruptured globe. Topical medications can affect the size of the pupil. Sympathomimetic (tropicamide) and anticholinergic (homatropine or scopolamine) drops, also known as *mydriatics*, will dilate the pupil. Beta-blockers (timolol) and cholinergic preparations (pilocarpine), known as *miotics*, result in pupillary constriction.

The swinging flashlight test

The swinging flashlight test is used to test for "paradoxical" pupillary dilation in response to light. Abnormal results are referred to as a relative afferent pupillary defect (RAPD) or a Marcus–Gunn pupil involving the affected eye (Figure 26.5). This test is performed in a darkened room by shining a bright light in the patient's eye for 1–2 seconds followed by swinging the light to stimulate the other eye, and repeating the process in each eye. A normal physiologic response is constriction of the pupil with direct light stimulation. Pupillary dilation in response to direct light stimulation indicates an afferent (sensory) visual lesion, most commonly an optic neuropathy. Failure of the pupil to constrict with either direct or consensual light stimulation indicates an efferent pupillary defect.

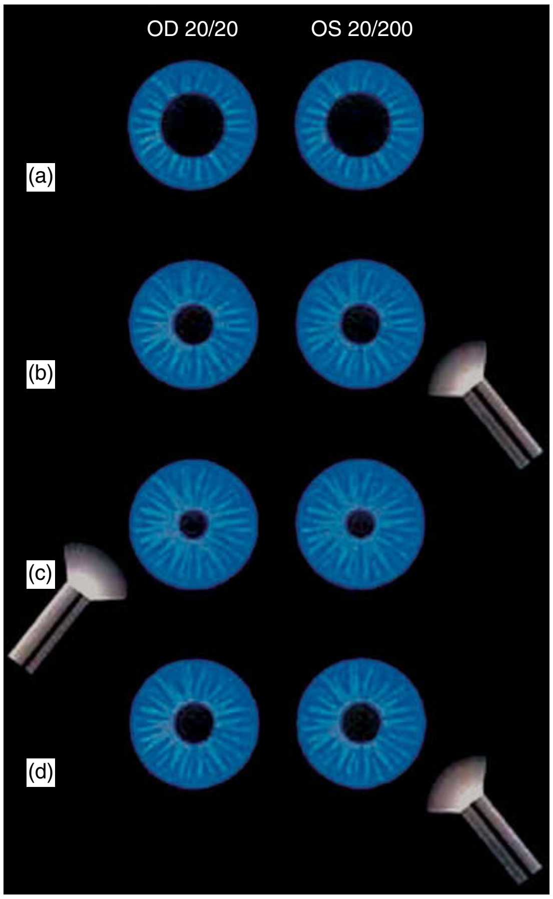

Figure 26.5
Relative afferent pupillary defect (RAPD). Vision in the right eye is 20/20, but vision in the left eye is 20/200 because of optic neuropathy. (a) The pupils in dim light are equal. (b) Light directed into the left eye results in a partial and sluggish contraction in each eye. (c) Light directed into the right eye results in a brisk and normal reaction in each eye. (d) The light quickly redirected into the left eye results in dilation of both pupils. It is possible to detect RAPD even in the presence of a dilated, dysfunctional pupil, as in a traumatic mydriasis or cranial nerve III palsy, by observing the other pupil. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

Anisocoria

Lesions that result in anisocoria (unequal pupils) include:

1. *Adie's tonic pupil*: A dilated pupil that responds poorly to direct light but constricts with accommodation. The lesion is in the cranial nerve III ciliary ganglion, usually the result of a viral infection or local inflammation.
2. *Argyll–Robertson pupil*: A small, irregular pupil that reacts poorly to light. It develops over months to years and is associated with central nervous system (CNS) syphilis.
3. *Horner's syndrome*: A miotic pupil that reacts to light. There is often accompanying ptosis of the lid on the affected side, as well as anhydrosis of the ipsilateral face. It is the result of sympathetic denervation from apical lung tumors, trauma to the spinal cord, brainstem lesions, carotid artery dissection, or syringomyelia.
4. *Oculomotor nerve palsy:* A patient presenting with anisocoria due to a new-onset oculomotor nerve palsy is presumed to have a cerebral aneurysm until proven otherwise, as compression may cause only pupillary findings initially. Uncal herniation causing a compressive 3rd nerve palsy ("blown" pupil) is typically associated with abnormal consciousness.
5. *Essential anisocoria*: A small percentage of the population has unequal pupils at baseline.
6. *Factitious dilated pupil*: Occasionally a patient will self-medicate with anticholinergic drops (scopolamine or homatropine). They may also inadvertently get flea medication, another

anticholinesterase, or permethrin-based agents with mydriatic properties in their eye. In these situations, a patient will present with a fixed and dilated pupil. The administration of two drops of 1% pilocarpine can be diagnostic, as a "normal" pupil will constrict over 45 minutes, whereas a "medicated" pupil will remain dilated.

Visual fields

Visual field testing will detect disorders affecting the retina, optic nerve, optic chiasm and visual cortex. Patients with visual complaints (irrespective of their visual acuity) should always be screened for visual field defects. Confrontational visual fields are measured for one eye at a time, and can detect segmental retinal detachments (often a horizontal defect) or intracranial pathology (usually a vertical defect obeying the midline). Care must be taken to ensure that the non-examined eye of the patient is completely covered. The visual field examination may detect a central scotoma common in optic neuritis, a localized defect due to a retinal detachment, or bitemporal hemianopsia indicative of a lesion affecting the optic chiasm, such as a pituitary tumor (Figure 26.6).

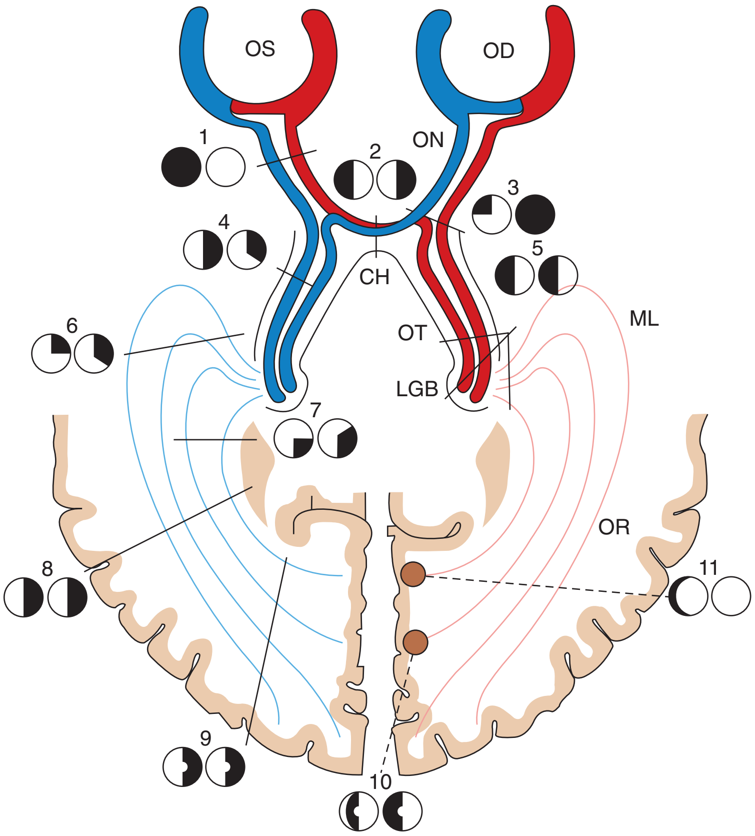

Figure 26.6
Schematic illustration of the **visual pathway** and **visual field defects** produced by lesions in various areas of the pathway. [OS, oculus sinister; OD, oculus dexter; ON, optic nerve; CH, chiasm; OT, optic tract; LGB, lateral geniculate body; ML, Meyer's loop; OR, optic radiations]. (1) Compromise of the left optic nerve results in a central scotoma in the left eye, with a normal right visual field. (2) A lesion of the optic chiasm may cause a bitemporal hemianopia. (3) A lesion at the junction of the right optic nerve and the chiasm results in a central scotoma in the right eye and a superior visual field defect that respects the vertical meridian in the left eye. This effect results from compromise of the inferior nasal crossing fibers from the left eye, which extend into the prechiasmal portion of the right optic nerve (i.e., Wilbrand's knee). The resulting visual field defect is known as a junctional scotoma, which is localized at the junction of the optic nerve and chiasm. (4) Complete interruption of the optic tract produces a homonymous hemianopic field defect. Subtotal lesions produce highly incongruous homonymous hemianopias. (5) Complete interruption of the optic tract, lateral geniculate body, and optic radiations results in a total contralateral homonymous hemianopia. (6) Fibers originating in the ipsilateral inferior temporal retina and the contralateral inferior nasal retina sweep anteriorly and laterally around the temporal horn (i.e., Meyer's loop) before transversing posteriorly. As a result, lesions of the temporal lobe characteristically produce superior, often incongruous homonymous quadrantanopias. (7) Parietal lobe lesions may interrupt visual pathway fibers from the superior retinas pursuing a more direct posterior course. This results in an inferior homonymous quadrantanopia. (8) Complete interruption of the optic radiations results in contralateral total homonymous hemianopia. (9) Posterior occipital lobe lesions result in homonymous hemianopic defects, which may spare the macula. Subtotal occipital lesions produce exquisitely congruous visual field defects because the fibers are more highly segregated in the occipital area. (10) Lesions affecting the posterior portion of the occipital lobe may spare the more anteriorly placed unpaired crossing peripheral nasal retinal fibers, resulting in a preserved temporal crescent in an otherwise congruous homonymous hemianopia. (11) Focal lesions involving the anterior-most portion of the occipital lobe may affect the receptive area for the unpaired crossing fibers from the contralateral nasal retina, resulting in a unilateral peripheral temporal visual field defect. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

Anterior segment

General inspection and slit lamp examination will detect abnormalities of the anterior segment, including the conjunctiva, sclera, cornea, iris, lens and anterior chamber. The conjunctiva and sclera should be examined for discharge, injection, chemosis (swelling), or foreign bodies. When discharge is present, its amount, quality and color should be noted. The pattern of erythema (generalized vs. focal) should also be noted. Acute conjunctivitis usually results in diffuse erythema, whereas anterior uveitis presents with redness largely around the cornea (perilimbic erythema). The white sclera may become thin in scleritis, resulting in a bluish hue to the eye.

Topical anesthetics

Patients with eye pain, infection, inflammation or foreign body often require the application of a topical anesthetic to facilitate examination. A few drops of a preparation such as 0.5% proparacaine should be placed in the inferior palpebral sulcus. Prompt relief of symptoms following application of a topical anesthetic is nearly diagnostic of corneal abrasion. Never dispense a topical anesthetic for outpatient use; extended application has been associated with persistent corneal defects, infections, ulcers, and increased risk of additional trauma.

Eyelid eversion

After instillation of a topical anesthetic, the eyelids should be everted to look for adherent foreign bodies. With the patient looking down, apply a cotton-tipped applicator to the mid-portion of the upper lid. Gently grab the upper lashes and pull the eyelid over the applicator. When the patient looks up, this action is reversed.

The slit lamp examination

The ideal method of examining the cornea, iris and anterior chamber is the slit lamp microscope, which allows magnification of these structures. Normally, the anterior chamber is clear. However, careful inspection under magnification may reveal proteinaceous debris (flare), red blood cells (hyphema), or purulent exudate (hypopyon). Flare resembles a motion projector beam in a dark smoky room. The presence of cells and flare in the anterior chamber is commonly associated with anterior uveitis (also known as iritis or iridocyclitis).

The oblique flashlight test

This test may demonstrate a narrow or closed angle in AACG (Figures 26.7 and 26.8). A penlight is held to the side of the patient's head, with the beam parallel to the iris and shining across the anterior chamber. If the entire iris is illuminated, the "angle" is open. If there is a shadow projected on the nasal part of the iris, the "angle" is narrow or closed. If the cornea of the affected eye is too hazy to perform this test effectively, it should be performed on the other eye. Although AACG usually occurs in only one eye, both eyes will have "narrow" or "closed" angles.

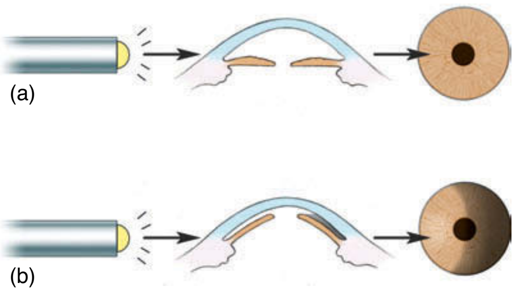

Figure 26.7
Oblique flashlight test: (a) normal and (b) shallow anterior chamber. © Chris Gralapp.

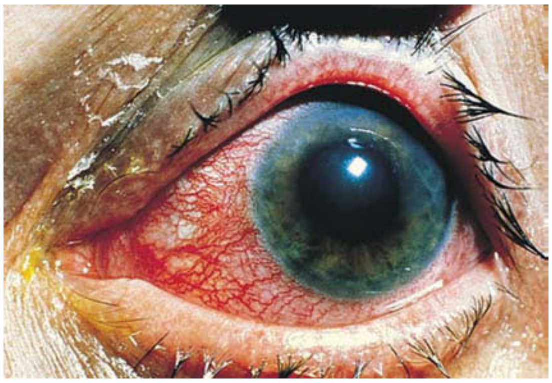

Figure 26.8
Acute angle closure glaucoma. Typical external appearance of acute angle closure caused by pupillary block, with diffuse hyperemia of the conjunctiva, mid-dilated pupil, and steamy cornea. The intraocular pressure is 64 mmHg. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

Fluorescein staining

The cornea should be examined for clarity, surface irregularities and foreign bodies. Fluorescein dye is used to identify epithelial defects of the cornea. After the application of a topical anesthetic, a fluorescein-coated strip is lightly applied to the lower conjunctival sulcus and the patient is asked to blink a few times. The damaged corneal epithelium picks up the dye and fluoresces brightly when the cornea is examined with a cobalt blue light (Figure 26.9). The slit lamp provides superior visualization of any epithelial defects. Fluorescein staining will identify scratches from a foreign body, corneal ulcers (Figure 26.10), and the "grape-like clusters" of herpetic infections (dendrites) (Figure 26.11). Removal of contact lenses prior to the application of fluorescein will prevent permanent staining.

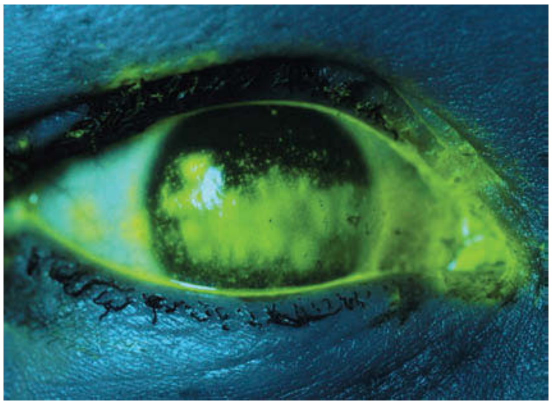

Figure 26.9
Corneal abrasion. A large traumatic corneal abrasion stains brightly with the cobalt blue light after topical fluorescein instillation. Courtesy: Lawrence Stack, MD.

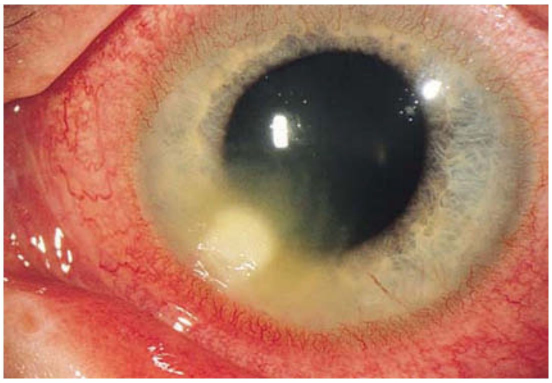

Figure 26.10
Corneal ulcer. Infected corneal ulcers are characterized by corneal infiltrates associated with overlying epithelial defects and an anterior chamber reaction. This ulcer caused by *Staphylococcus aureus* extends toward the center of the cornea and is associated with surrounding corneal edema. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

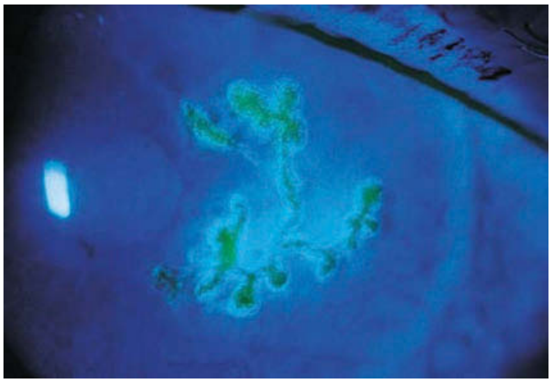

Figure 26.11
Herpetic simplex dendrites. The hallmark of herpes simplex keratitis is the dendrite, a branching, epithelial ulceration with swollen, raised edges and terminal bulbs. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

Posterior segment (funduscopy)

Funduscopy is rarely useful in the evaluation of the red eye; however, it may identify the cause of visual loss or eliminate possible etiologies. The funduscopic examination may be facilitated by dilation of the pupils with topical short-acting mydriatics (e.g., tropicamide) or cycloplegics (e.g., cyclopentolate). Prior to inducing pupillary dilation, patients should be screened for contraindications to dilation of the pupil, such as suspicion of globe rupture, history of angle closure glaucoma, or narrow anterior angles.

Funduscopy may reveal abnormalities of the retina, optic nerve, or vitreous cavity. A diminished red reflex may result from corneal edema, cataract formation, vitreous hemorrhage, or a large retinal detachment. A classic (late) finding of CRAO is a pale retina with a "cherry red spot" indicating preservation of the blood supply to the fovea (Figure 26.12). Venous congestion and hemorrhage may be evident in CRVO, with the retina having a "blood and thunder" appearance (Figure 26.13). Some patients with optic neuritis may present with edema of the optic disk. Funduscopic evidence of a retinal detachment may explain a patient's sudden visual loss (Figure 26.14). Any patient with acute vision loss thought to be due to posterior segment disease should be immediately seen by an ophthalmologist for further evaluation.

Intraocular pressure (IOP)

A portable Tono-Pen (electronic tonometer), Schiotz pressure tonometer, or applanation tonometer (part of the slit lamp microscope) can be used to measure IOP. These techniques require topical anesthesia prior to direct application of the device over the surface of the

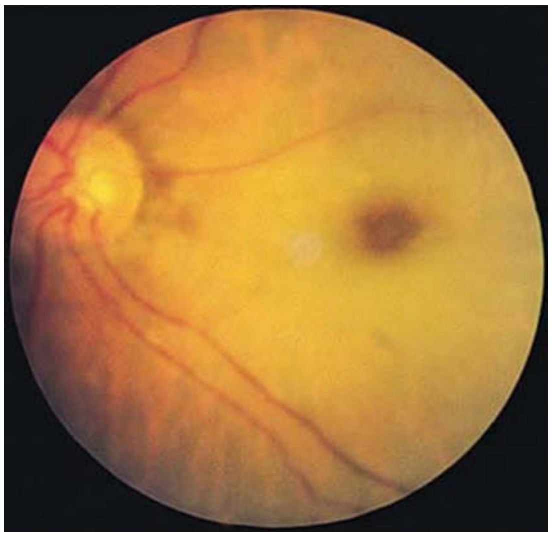

Figure 26.12
Central retinal artery occlusion. An acute central retinal artery obstruction with superficial retinal whitening and a cherry red spot in the fovea. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

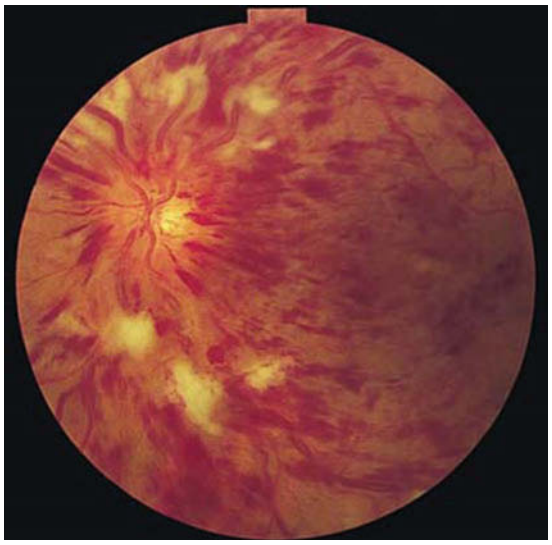

Figure 26.13
Central retinal vein occlusion. Fundus examination shows dilated and tortuous retinal veins, a swollen optic disc and retinal hemorrhages. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

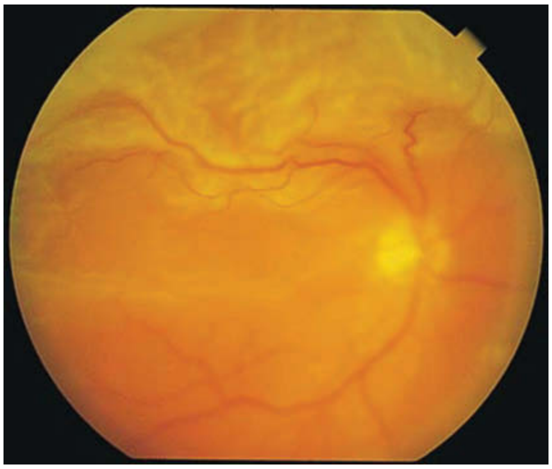

Figure 26.14
Retinal detachment. Corrugated, opaque appearance of the detached retina in a patient with rhegmatogenous retinal detachment. Reproduced with permission from Tasman W, Jaeger EA, *Wills Eye Hospital Atlas of Clinical Ophthalmology*, 2nd ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2001.

cornea. Measurement of IOP is contraindicated in cases of globe rupture. Normal IOPs range between 12 and 21 mmHg. People who have pressures between 21 and 30 mmHg with normal optic disks have ocular hypertension. Although any value over 30 mmHg is abnormal, a measurement of >50 mmHg suggests acute angle closure glaucoma.

General physical examination

The general physical examination is used to assess the overall health of the patient, and may provide important clues to the nature of the eye problem. Joint deformities may indicate a connective tissue disease, whereas signs of malnourishment may indicate chronic or acute immunosuppression. The skin should be examined for lesions indicating systemic infection or autoimmune disorders.

Differential diagnosis

Tables 26.3 and 26.4 describe causes of the red eye and etiologies of visual change or vision loss, respectively.

Diagnostic testing

Laboratory studies

Very few laboratory tests are used in evaluating the red eye (Table 26.3) or acute visual loss (Table 26.4).

The red eye

1. *A Gram stain* may be useful for GC conjunctivitis, but is rarely helpful in other cases of conjunctivitis.
2. A *corneal culture* and *scraping* is useful in the evaluation of a corneal ulcer, but bacterial sensitivities often do not correlate with the clinical response.
3. *Blood cultures* may be useful in defining the infectious agent in orbital cellulitis.
4. Specific blood tests for collagen vascular disorders or autoimmune diseases may be useful in the evaluation of scleritis.

Vision loss

1. *Erythrocyte sedimentation rate* (ESR): This test should only be ordered if temporal (giant cell) arteritis is highly suspected. An ESR >50 in an older individual with headache and/or visual change is highly suggestive of temporal arteritis.
2. *Electrolytes, blood urea nitrogen* (BUN), *creatinine*: A patient with AACG may have electrolyte abnormalities or dehydration from nausea and vomiting.
3. *Glucose, lipid profile*: Diabetes and hyperlipidemia are associated with CRAO.

Radiologic studies

Computed tomography (CT) of the brain, orbits and sinuses may be useful in delineating infectious, inflammatory, and malignant processes. CT will differentiate POC (superficial) from OC (deep). It will also define any coexisting conditions, such as sinusitis, abscess, or tumor.

Magnetic resonance imaging (MRI) is often ordered for optic neuritis, primarily to look for demyelinating lesions

Table 26.3 The red eye

Diagnosis	Symptoms	Signs	Work-up
Acute angle closure glaucoma	Severe unilateral eye pain, decreased vision, halos around lights. May have nausea, vomiting, headache, abdominal pain or constitutional symptoms.	Red eye; hazy ("steamy") cornea; mid-dilated, minimally reactive pupil. Elevated IOP (>50 mmHg).	Evaluate other eye for shallow anterior chamber. Measure IOP using tonometry. Consult an ophthalmologist immediately.
Acute anterior uveitis (iritis, iridocyclitis)	Pain, tearing, photophobia and mildly decreased vision (may have history of trauma several days before)	Marked perilimbal conjunctival injection (ciliary flush); "cells" (WBCs) and "flare" (protein) may be visible in the anterior chamber on slit lamp examination.	None if unilateral, first occurrence. Refer to an ophthalmologist for a further work-up.
Allergic conjunctivitis	Itching, burning, redness and tearing	Inflamed and watery eye, may have chronic changes on the lids and/or conjunctivae.	None
Bacterial conjunctivitis (non-gonococcal)	Redness and mucopurulent discharge from one eye, then the other; foreign body sensation; normal or decreased vision	Diffuse conjunctival injection, purulent discharge, preauricular node formation.	Culture and sensitivity of discharge (for severe or refractory cases).
Blepharitis	Eye irritation, foreign body sensation, crusting and swelling of the lids	Chronic scaling, edema, or erythema of lid margins. May have abnormal apposition of lids.	None
Chalazion	Typically painless, slowly growing erythematous nodule of the eyelid	Lump usually located on the conjunctival portion of eyelid; often an incidental finding.	None
Chlamydial conjunctivitis	Redness and mucoid discharge, foreign body sensation	Looks like viral conjunctivitis; follicular conjunctival changes.	Chlamydia culture. Refer to an ophthalmologist immediately.
Corneal abrasions	Sudden-onset of excruciating pain, tearing, photophobia; decrease in vision, foreign body sensation	Conjunctival injection, blepharospasm, light sensitivity and corneal defect on fluorescein staining.	Evert the lids to search for retained foreign body. Topical anesthetics provide immediate pain relief.
Corneal ulcers	Pain, decreased vision, foreign body sensation, photophobia	Focal white corneal infiltrate. Raised borders and crater with slit lamp exam. Intense staining with fluorescein if there is an epithelial defect.	Corneal scrapings for smear and culture. Refer to an ophthalmologist immediately.
Dacryocystitis	Pain, redness, swelling over the lacrimal sac; may have tearing, discharge and fever; may be recurrent. Associated with nasolacrimal duct obstruction.	Erythematous, tender swelling over nasal aspect of lower eyelid; purulent discharge may be expressed with gentle compression.	Culture and sensitivity of discharge. Refer to ophthalmologist.
Episcleritis	Acute localized redness and mild pain in one or both eyes	Localized engorgement of episcleral blood vessels; may be a small nodule.	Topical phenylephrine 2.5% may reduce the redness and differentiate it from scleritis.
Gonococcal conjunctivitis	Redness, acute onset, profuse purulent discharge	"Angry eye" (bloody conjunctival injection and red swollen lids); "waterfall of pus" (the discharge is so copious, it reaccumulates after wiping).	GC culture and Gram stain. Refer to an ophthalmologist immediately.
Herpes simplex keratitis	Irritation, tearing, decreased vision and photophobia. May have history of previous episodes.	Decreased visual acuity; decreased corneal sensation in 80% (test blink reflex with cotton applicator). Characteristic dendrites on fluorescein staining.	Refer to an ophthalmologist immediately.
Herpes zoster ophthalmicus	Eye pain, redness and decreased vision. May have prodrome of HA, fever and malaise.	Pseudodendrites on fluorescein staining. Vesicular lesions on the tip of the nose (Hutchinson's sign) suggest ocular involvement.	Refer to an ophthalmologist immediately.
Hordeolum	Localized pain and swelling of one eyelid	Localized swelling of eyelid, sometimes with "pointing" inside lid or on lid margin.	None
Orbital cellulitis	Similar to POC; may have headache, blurred vision or diplopia	Similar to POC, but testing EOMs may produce pain. May also have proptosis, restricted ocular motility and decreased vision.	CT scan to evaluate the extent of orbital involvement and to prepare for possible surgical debridement. Refer to an ophthalmologist immediately.

(*continued*)

Table 26.3 The red eye (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Periorbital cellulitis	Swelling, redness and pain around one eye	Unilateral swelling, redness and tenderness around the eye, including the lids; often with fever.	CT scan to rule out spread to the orbit and to evaluate the sinuses
Scleritis	Severe, boring eye pain, redness and decreased vision	Inflammation of scleral, episcleral and conjunctival blood vessels. If severe, bluish hue can be seen.	Evaluate for underlying connective tissue disease. Refer to an ophthalmologist immediately.
Superficial keratitis (UV keratitis, Welder's flash)	Pain, redness, tearing and photophobia; may have history of UV light exposure	Conjunctival injection. Fluorescein staining may show pinpoint corneal epithelial defects.	None
Viral conjunctivitis	Redness, burning and watery discharge; often profuse; normal vision. May have had a recent URI or contact with someone with a red eye.	Diffuse conjunctival injection; red, edematous eyelids; watery discharge; pre-auricular node.	Contact precautions for 2 weeks, consider DFA for adenovirus.

CT: computed tomography; DFA: direct fluorescent antibody; EOMs: extraocular muscles; GC: gonococcus; HA: headache; IOP: intraocular pressure; POC: periorbital cellulitis; URI: upper respiratory infection; UV: ultraviolet; WBC: white blood cell.

Table 26.4 Visual change or vision loss

Diagnosis	Symptoms	Signs	Work-up
Central retinal artery occlusion	Sudden painless vision loss	Severe vision loss. Afferent pupillary defect. Pale edematous retina with cherry red spot (late).	Refer to an ophthalmologist immediately. Obtain ESR in elderly patients to evaluate for giant cell arteritis.
Amaurosis fugax	Transient sudden, painless, monocular vision loss. Vision often returns prior to ED visit.	Hollenhorst plaque (embolus composed of cholesterol) variably present in the retinal circulation. Examination may be normal.	Search for the source of the embolus: ECG, echocardiogram and carotid Doppler. Obtain ESR in elderly patients to evaluate for giant cell arteritis.
Binocular diplopia	Present when both eyes are open; may occur when looking in only one direction. Resolves with closing either eye.	May have abnormalities on EOM examination. May have signs of external eye trauma.	Evaluate for diabetes, thyroid disease and neuromuscular disorders (myasthenia gravis, botulism). CT scan of the orbit and brain (especially in cases of trauma). MRI for suspicion of brainstem lesion. MRA for suspicion of circle of Willis aneurysm.
Central retinal vein occlusion	Abrupt or gradual decrease in vision	Mild to very severe vision loss. Retinal hemorrhages, dilated veins in four quadrants with optic disc edema.	Evaluate for hypertension, DM, ASCVD, hyperlipidemia and glaucoma.
Functional vision loss	Varies from blurry vision to complete vision loss, monocular or binocular	Normal examination: normal pupillary light reflex, normal optokinetic testing.	Psychiatric consult. Consider ophthalmology consult.
Monocular diplopia	Double vision in one eye	May see cataract or dislocated lens.	Evaluate for refractive error involving the patient's contact lenses or bifocal glasses. Inquire about trauma, eye surgery, flashing lights or floating spots (retinal detachment, posterior vitreous detachment).
Optic neuritis	Progressive visual loss over hours to days. Periorbital pain, worse with movement of eye. Alterations in color vision. Age <50 years.	Decreased visual acuity. Normal disc although 1/3 may have disc edema. Afferent pupillary defect.	MRI within 2 weeks for possible MS. Test for Lyme disease, syphilis, toxoplasmosis. Refer to an ophthalmologist immediately.
Retinal detachment	Sudden onset of light flashes or floaters; a "curtain" or "shade" descending over a field of vision. Visual loss or a visual field defect.	Pigmented cells in the vitreous, vitreous detachment, retinal detachment, or retinal break. An afferent pupillary defect may be present.	Refer to an ophthalmologist immediately.

(*continued*)

Table 26.4 Visual change or vision loss (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Temporal arteritis	Sudden painless visual loss. May have unilateral headache, pain with chewing (jaw claudication), proximal muscle and joint aches (polymyalgia rheumatica), weight loss, anorexia, or fever. Rare <65 years of age.	Afferent pupillary defect; devastating visual loss; pale, swollen optic disc. May have tenderness over the temporal artery.	ESR >50 (usually <100). Temporal artery biopsy. Refer to an ophthalmologist immediately.
Vitreous hemorrhage	Sudden painless visual loss. Visual “floaters” or “cobwebs.”	Decreased or absent red reflex, pigmented cells in the vitreous.	Evaluate for DM, trauma, leukemias and thrombocytopenia. Refer to an ophthalmologist immediately. Evaluate for retinal tear or detachment.

ASCVD: arteriosclerotic cardiovascular disease; CT: computed tomography; DM: diabetes mellitus; ECG: electrocardiogram; ED: emergency department; EOM: extraocular muscle; ESR: erythrocyte sedimentation rate; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging; MS: multiple sclerosis.

of multiple sclerosis (MS). As many as 40% of individuals diagnosed with MS initially present with symptoms of retrobulbar or optic neuritis. In this setting, the MRI is not needed emergently. MRI is contraindicated in patients with metallic intraocular or intraorbital foreign bodies given the risk of further injury from movement of these objects.

General treatment principles

Treatment of eye disorders is predicated on the specific diagnosis.

The red eye

Common disorders not requiring specialty care:

Allergic conjunctivitis

Treatment depends on the etiology. For example, in individuals sensitive to cat dander, avoidance of the allergen may be the only treatment necessary. Topical over-the-counter vasoactive drops, oral antihistamines, or both may help in the short term. Mast cell stabilizer ophthalmic drops are effective, but usually take 2 weeks before any effect is appreciated.

Bacterial conjunctivitis

Although generally considered benign and self-limited, the course of bacterial conjunctivitis can be shortened by the use of broad-spectrum topical antibiotics. Inexpensive drops such as polymyxin/trimethoprim are perfectly acceptable. The decision of which agent to use is based on cost, availability, local resistance patterns and practice. Neomycin preparations should be avoided because of the high incidence of allergic conjunctivitis.

Blepharitis

Treatment includes a daily regimen of lid hygiene, including warm compresses, lid massage, and cleaning the lids with diluted (1:10) baby shampoo.

Chalazion

Warm compresses should be applied for 15 minutes four times daily. The use of antimicrobial ointments is controversial; they are not recommended in ophthalmology textbooks, but are commonly used in practice. Steroid injection and elective excision should be left to the ophthalmologist.

Corneal abrasions

The goals of treatment of corneal abrasions are symptom relief, prevention of infection, and monitoring of healing. A short course of topical broad-spectrum antibiotic ophthalmic drops and short-acting topical cycloplegics (e.g., cyclopentolate) are usually prescribed. Topical nonsteroidal antiinflammatory drug (NSAID) preparations may reduce the pain associated with corneal abrasions, but are relatively expensive. Oral narcotic pain medications may be prescribed for patient comfort. Eye patching for corneal abrasions less than 10 mm in diameter is no longer recommended, and is contraindicated in abrasions associated with contact lens use due to an increased risk of *Pseudomonas* infections. Corneal abrasions associated with contact lens use should be treated with broad-spectrum topical antibiotics with good antipseudomonal coverage (i.e., gentamicin, polymyxin/bacitracin, ciprofloxacin or tobramycin). Tetanus status should be investigated and updated as needed. Under no circumstances should patients be prescribed outpatient topical anesthetics.

Episcleritis

Topical or oral NSAIDs are often effective for episcleritis.

Hordeolum (stye)

Warm compresses are helpful, as for chalazion. Topical antibiotics should be prescribed if the inflammation extends beyond the stye. Surgical drainage with an 18-G needle or No. 11 blade may speed resolution but runs the risk of scarring if done incorrectly (i.e., on the external lid surface). A hordeolum that points directly into the lid margin and affects vision should be drained, but only by someone with experience.

Superficial keratitis

Broad-spectrum topical antibiotics, cycloplegics (if associated anterior uveitis is present), and oral pain medication usually control the symptoms and generally prevent complications.

Viral conjunctivitis

There is no specific treatment for viral conjunctivitis. Adults who are capable of good hygiene may return to work if they have a negative direct fluorescent antibody (DFA) test to the viruses responsible for EKC. In the presence of a positive DFA for EKC, the patient should remain off work for 2 weeks to control the spread of this debilitating infection. Children who cannot wash their hands meticulously must be kept home from school.

The following conditions threaten vision and require immediate consultation:

Acute angle closure glaucoma

If untreated, AACG will result in blindness. The initial treatment goal is to reduce IOP. Accepted acute medical therapy includes topical beta-blockers (timolol 0.5%), low-dose parasympathomimetics (pilocarpine 0.2%), topical steroids (prednisolone 1%), topical alpha-2-agonists (apraclonidine 0.5%), and oral or IV acetazolamide. Acetazolamide should be avoided or used with caution in patients with significant pulmonary disease, because of the combined effects of metabolic and respiratory acidosis. IV mannitol is effective in lowering IOP, but is relatively contraindicated in patients with congestive heart failure (CHF) or renal failure because of the significant osmotic load. Immediate consultation with an ophthalmologist is necessary. Reduction of IOP may take hours, requiring repeated administration of medications to achieve a satisfactory pressure. Close observation and management by an ophthalmologist is mandatory.

Anterior uveitis

Topical steroids (e.g., prednisolone) are the mainstay of anterior uveitis therapy. As steroids themselves are associated with complications (e.g., cataract formation, glaucoma and pupillary abnormalities), they should only be prescribed following consultation with an ophthalmologist. Intermediate-acting cycloplegics, such as homatropine or scopolamine, are used to alleviate ciliary spasm, which is responsible for much of the pain associated with anterior uveitis.

Corneal ulcers

Immediate referral to an ophthalmologist is necessary. Never attempt to treat a suspected corneal ulcer without ophthalmologic consultation. The medicolegal ramifications of under- or inappropriately treated disease are profound, as permanent visual loss is common.

Gonococcal conjunctivitis

These patients often require parenteral antibiotics (e.g., Ceftriaxone), ocular saline lavages, and daily ophthalmologic evaluations.

Herpes zoster keratitis

Oral antivirals and topical antibiotics (to prevent secondary bacterial infection) are used to treat the skin lesions of HZV ophthalmicus. Topical antivirals have not been proven effective. Topical steroids are used if anterior uveitis or deep stromal keratitis is present, although only after direct consultation with an ophthalmologist. As 50% of division I trigeminal nerve HZV infections involve the cornea and have potential for scarring, these cases should be referred to an ophthalmologist.

Herpetic simplex keratitis

For disease limited to the epithelium, either topical (trifluridine, vidarabine, idoxuridine) or oral (acyclovir, famciclovir, valacyclovir) antivirals are effective in the majority of cases. When corneal ulcers are present, both an oral and a topical agent are used. Steroid preparations should be used only after consultation with an ophthalmologist. Oral acyclovir at prophylactic doses has been shown to prevent recurrences of HSV eye disease.

Suspected globe rupture/perforation

Shield the eye immediately and discontinue the examination. This condition is a surgical emergency that threatens the entire globe. Care should be rapidly transferred to an ophthalmologist. Patients should not be allowed to eat or drink, as surgical repair is likely and vomiting is likely to increase IOP. Antiemetics may be needed for patients with a perforated or ruptured globe. Seidel's test is positive when fluorescein dye is seen streaming from the site of globe rupture. This test should not be performed in obvious cases of globe perforation or rupture.

Visual change or vision loss

Most of these conditions require immediate or urgent ophthalmologic consultation.

Central retinal artery occlusion

Few interventions have much effect on the generally poor outcome from CRAO. While awaiting the arrival of the ophthalmologist, some low-risk (and potentially beneficial) actions may be taken. Ocular massage (5 seconds on, 5 seconds off) for 15–30 minutes may manually dislodge the clot. Rebreathing carbon dioxide (CO_2) by breathing into a paper bag may result in vasodilation of the central retinal artery. Acetazolamide IV has also been advocated as it may lower IOP. Paracentesis of the anterior chamber is no longer recommended as a therapeutic intervention by emergency providers.

Central retinal vein occlusion

No specific medical therapy is effective for CRVO. Surgery and thrombolytic therapy remain experimental. Some patients may be candidates for laser surgery. An ophthalmologist should be involved in the care of patients with CRVO.

Optic neuritis

IV methylprednisolone 250 mg 4 times daily for 3 days improves the short-term visual outcome and may slow the progression of MS over the subsequent 2 years. Oral prednisone is contraindicated because of adverse visual outcomes. Always consult with an ophthalmologist or neurologist.

Retinal detachment

Suspected retinal detachments should be urgently referred to an ophthalmologist. No specific sight-saving therapy can be instituted in the ED. Identification of the problem, exclusion of other conditions, and urgent ophthalmologic consultation are essential.

Temporal (giant cell) arteritis

The suspicion of temporal arteritis and the presence of visual symptoms warrant the administration of oral or parenteral steroids prior to the establishment of a definitive diagnosis by temporal artery biopsy. This condition warrants immediate ophthalmologic referral.

Special patients

Pediatric

1. *Neonatal conjunctivitis*: Caused by GC or HSV acquired during passage through the birth canal, it can be vision- and life-threatening, requiring a complete septic work-up, admission, IV antibiotics and antivirals. Chlamydia infection, acquired in the same manner, is more benign and can be treated on an outpatient basis.
2. *Periorbital* or *orbital cellulitis*: Since the advent of the *Haemophilus influenzae* type B (HIB) vaccine, POC and OC are much less common. In the past, due to the aggressive spread of *H. influenza*, a complete septic work-up and IV antibiotics were recommended for suspected cases of POC or OC. In the post-HIB vaccine era, POC and OC are more commonly associated with sinusitis or contiguous spread from trauma or skin infections. In patients over the age of 12 months, and in the absence of orbital or intracranial involvement, POC may be treated on an outpatient basis (if the parents are compliant and reliable follow-up can be arranged); OC still requires admission.
3. *Kawasaki disease*: A multisystem disease occurring primarily in children under 8 years of age. Bilateral conjunctival injection that spares the perilimbic area is one component of the disease. The incidence of coronary artery aneurysms, a cause of significant morbidity, can be significantly reduced by the administration of IV gamma globulin and high-dose aspirin.
4. *Acute dacryocystitis* (AD): Neonates, infants and small children with AD require admission for IV antibiotics covering *Staphylococcus* and *Streptococcus*. These patients also require ear, nose and throat (ENT) or ophthalmologic consultation for nasolacrimal duct probing.
5. *Congenital nasolacrimal duct obstruction* and *chronic dacryocystitis*: Chronic duct obstruction usually resolves by 1 year of age and is managed by instructing the parents to "milk" the sac and duct in an effort to improve drainage. Chronic dacryocystitis requires topical antibiotics and subsequent referral to an ophthalmologist.
6. *Suspected shaken baby syndrome (child abuse)*: Ophthalmology consultation should be obtained to look for retinal hemorrhages, which are highly concerning for shaken baby syndrome in the appropriate setting.

Immune compromised

Patients with diabetes, hematologic malignancies, those on immunosuppressive drugs, and generally debilitated individuals are susceptible to mucormycosis, an aggressive fungal infection. This infection begins in the sinuses and spreads contiguously to the orbits. Mucormycosis presents as unilateral eye swelling, accompanied by proptosis and decreased vision. Treatment is surgical debridement and IV antifungal agents. Immediate ophthalmologic referral is essential.

Disposition

Table 26.5 describes ophthalmologic referral and disposition recommendations for important causes of eye pain, redness and visual loss.

Table 26.5 Ophthalmologic referral and disposition

Disease	Emergent ophthalmology consult	Urgent ophthalmology consult	"Next day" ophthalmology consult	Admission
Acute angle closure glaucoma	Yes			Yes
Anterior uveitis			Yes	No
Corneal abrasion		Yes (in contact lens wearer)	Yes	No
Corneal ulcer	Yes			If compliance issues
CRAO	Yes			Yes
CRVO		Yes		Yes
GC conjunctivitis	Yes			Often, depends on compliance
Globe rupture/perforation	Yes			Yes
HSV keratitis	Yes			Not usually
HZV keratitis	Yes			Not usually
Neonatal conjunctivitis	Yes if <1 month of age			Yes, if GC or HSV
Optic neuritis		Yes (<24 hrs)		IV steroids if <72 hours from onset
Orbital cellulitis	Yes			Yes
Periorbital cellulitis			Yes	If <12 months, toxic, or compliance issues
Retinal detachment	Yes			No
Scleritis		Yes	Yes	No
Temporal arteritis (with visual changes)		Yes		Yes (IV steroids)

CRAO: central retinal artery occlusion; CRVO: central retinal vein occlusion; GC: gonococcus; HSV: herpes simplex virus; HZV: herpes zoster virus; IV: intravenous.

Pearls, pitfalls and myths

Pearls

- Obtain a thorough history, as it will often point you in the right direction.
- Remember to ask about chronic conditions, sexual contacts, job-related exposures and trauma.
- Do not forget visual acuity, the vital sign of the eye.
- Age-related vision loss: <50 years, consider optic neuritis; >65 years, consider temporal arteritis.

Pitfalls

- Do not patch corneal abrasions associated with contact lens use.
- Do not confuse a herpetic dendrite with a corneal abrasion.
- Remember to measure IOP in an older patient with sudden onset of unilateral eye pain and redness; it may be due to AACG.
- It is important to evert the eyelids to identify a possible foreign body in patients with corneal abrasions.
- Be wary of iris prolapse in the setting of ocular trauma, which may have the appearance of a corneal foreign body.
- The use of any steroid preparation in the eye requires direct consultation with an ophthalmologist.
- Patients with corneal abrasions or injuries should **never** be prescribed (or go home with) topical anesthetic preparations.

Myths

- *All patients with red eyes and tearing need topical antibiotics.* Viral conjunctivitis requires only attention to hygiene; allergic conjunctivitis may respond to antihistamines, vasoactive drops, or mast cell stabilizers. Only patients with a purulent discharge should be treated with a short course of broad-spectrum topical antibiotics.
- *Patching corneal abrasions is a necessary part of treatment.* This once-common practice is no longer recommended, as several studies have demonstrated neither improvement in pain relief nor healing when abrasions <10 mm are patched. Patching the eye is specifically contraindicated in contact lens wearers.

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27 Fever in adults

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Scope of the problem

According to the National Hospital Ambulatory Medical Care Survey (NHAMCS): 2006 Emergency Department Summary, fever is the third most common reason for visiting the emergency department (ED) in the United States. It accounted for 3.8% of all 119.2 million ED visits. This percentage is likely higher, not only due to the manner in which these data were recorded, but also since fever is ubiquitous to the human experience.

Many individuals feel that fever is harmful or a sign of serious underlying disease. Although most individuals with fever don't feel well, serious infections do not always produce a febrile response. Most healthy adults suffer only minimally from fever, as its cause is typically a self-limited illness responsive to symptomatic therapy. Morbidity and mortality from infectious causes of fever rise sharply with age.

In the ED, consideration of a fever's cause must be taken in the context of patient stability. Fever may not always be the patient's initial concern, and may be discovered on measurement of the initial vital signs. Other patients may present with a history of feeling "feverish" that has resolved. The absence of fever on presentation does not exclude serious illness, as the temperature curve fluctuates over time.

Anatomic essentials

It is important to distinguish whether a high temperature is from a *fever* (a deliberate hypothalamus-controlled reflex elevation of body temperature) or *hyperpyrexia* (an uncontrolled heat accumulation overwhelming compensatory mechanisms). This distinction has important immediate diagnostic and therapeutic implications. Antipyretics are ineffective in treating hyperpyrexia, whereas rapid cooling of a patient with a fever may interfere with the body's efforts to reach the new set point temperature.

When considering body temperature, one must first understand what is "normal," including common circadian variations. A healthy, unclothed adult can maintain euthermia within 1°F (0.5°C) when dry, ambient air is between 55°F and 140°F (13–60°C). Sample populations exhibit ranges of baseline temperatures between 97.6°F and 99.4°F (36.4–37.4°C) +/– 0.7°F (0.4°C). Women experience changes in body temperature during their menstrual cycle. Healthy elderly persons do not have lower core temperatures as is popularly believed, although their body composition makes it more difficult to retain heat (one reason why many older individuals feel cold in the ED).

Temperatures vary throughout the day by as much as 3.6°F (2°C), from a mean nadir oral temperature of 97.6°F (36.4°C) between 0400–0600 to a peak mean of 99.4°F (37.4°C) between 1600–2200. The lower limit of an abnormally elevated oral temperature may therefore be considered 100°F (37.8°C). Taking into account thermometer precision, fever is conservatively defined as a temperature of greater than 100.4 (38°C). It is generally believed that the fever response has a physiologic upper limit between 105.8°F and 107.6°F rectally (41–42°C). Infectious etiologies rarely lead to hyperpyrexia with collapse of compensatory thermoregulatory mechanisms in a normal host.

Systemic fever response

Body temperature is controlled within a narrow range that varies predictably throughout the day. Compensatory mechanisms ensure thermal homeostasis through autonomic nervous control by inducing changes in smooth muscle tone, shunting blood flow to and away from peripheral vascular beds, and provoking heat-seeking or heat-avoidance behaviors. Exogenous substances, such as bacterial cell wall components (lipopolysaccharides), bacterial breakdown products, endotoxins, drugs, immune complexes and activated complement factors, induce polymorphonuclear cells to release endogenous pyrogenic cytokines. These cellular mediators (interleukins [IL]-1 and 6, tumor necrosis factor [TNF], and interferon G) induce the production of prostaglandin E2 (PGE2) by endothelial cells. In a vascular area near the preoptic nucleus of the anterior hypothalamus, PGE2 diffuses to the neurons of the thermoregulatory center. Upon stimulation, efferent discharge increases peripheral heat-generating processes until a new temperature set point is established. The thermoregulatory center is also influenced directly by toxic or hormonal mediators, direct central nervous system (CNS) insult, or medications. Cyclooxygenase (COX) inhibitors that suppress fever by blocking PGE2 synthesis include aspirin, acetaminophen, nonsteroidal antiinflammatory drugs (NSAIDs) and COX-2 selective inhibitors.

In response to fever, chills (and less frequently rigors) subside within minutes of reaching the new febrile set point. During the fever peak, adults may experience a brief, mild delirium that is more prominent in the elderly. Myalgias and arthralgias occur as muscle tone and circulating inflammatory mediators increase. The increased metabolic state causes an increased pulse (10 beats/min/°F) and respiratory rate. Defervescence occurs and results in heat-dissipating processes, such as sweating, facial flushing and the sensation of warmth. Experimental models confirm that fever-inducing agents introduced directly into the bloodstream can generate clinical signs within minutes (Table 27.1).

Table 27.1 Common causes of fever

Infectious	Non-infectious
Viruses Bacteria Fungi Parasites	Allergic reactions CNS injury Inflammatory conditions Medications Neoplasm Hyperthyroidism Thromboembolic disease

CNS: central nervous system.

A febrile response enhances host defense mechanisms by increasing neutrophil migration and T-cell proliferation (known as *cellular immunity*). Tissue levels of antiviral interferon and TNF are also increased (*humoral immunity*). Antibacterial substances such as cytotoxic-free radicals are generated by polymorphonuclear cells (PMNs). Iron, a bacterial growth substrate, is sequestered. Metabolism is shifted from glucose stores toward increased protein and fatty acid breakdown.

This cascade of events, an alteration of the host's immune system to a more protective state, comes at a cost. For each degree Fahrenheit of temperature rise, the basal metabolic rate increases approximately 7%. Increased caloric demands are compounded by increased utilization of less efficient protein and fat fuels. Tissue oxygen demands also rise despite a temperature-induced shift of the oxygen-hemoglobin dissociation curve to the right. Tachycardia occurs from a combination of catecholamine stimulation and relative dehydration, as circulation shifts to the periphery. The CNS is the most vulnerable organ to heat injury. Subtle damage first manifests as mild delirium, especially in the elderly. Unlike in children, seizure activity or convulsions due solely to fever are rarely seen in adults.

Hyperpyrexia

Hyperpyrexia represents a critical imbalance of heat-producing and heat-dissipating processes, and should be considered at temperatures above 105°F (40.5°C). Extreme temperature elevations can occur from excess heat generation, impaired heat loss, a combination of these, or direct CNS insult. Heat rise in hyperpyrexia is independent of pyrogenic cytokine production and does not involve resetting the thermoregulatory set point. Antipyretics are therefore ineffective. Extreme temperature elevations are not seen until compensatory mechanisms have failed. It is important to act quickly to correct the heat imbalance before irreversible neurologic injury, rhabdomyolysis, cardiac dysrhythmias and circulatory collapse result (Table 27.2).

Local fever response

Inflammatory stimuli within the body activate a cascade of cellular and cytokine changes. These assume control of local homeostatic mechanisms at this site, resulting in changes that are characteristic but site-specific. If local control is not achieved rapidly, a systemic febrile response may result. This is more likely in highly vascular areas or those possessing a high concentration of immunologically active cells. Local inflammatory responses have been appreciated for centuries. These include *rubor* (erythema from vasodilatation), *dolor* (activation of pain fibers), *calor* (local temperature increase) and *tumor* (swelling or edema). Infection may also manifest locally by the accumulation or drainage of purulent material.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 27.3).

History

Adult patients presenting with fever represent a potential exposure risk to health care personnel. Universal precautions including gloves, gown, and mask or face shield are strongly encouraged in order to reduce or prevent direct blood-borne pathogen contact. Strict hand washing is mandatory. Indirect contact via stethoscope, thermometer, bed railing, or aerosolized droplet may transmit disease. Additional measures such as patient isolation or

Table 27.2 Causes of hyperpyrexia

Excess heat	Impaired heat loss	CNS dysfunction
Exertional heat stroke Delirium tremens Stimulant abuse Salicylism Thyroid storm Pheochromocytoma Status epilepticus Neuroleptic malignant syndrome Malignant hyperthermia (anesthetic) Muscle tetany Serotonin syndrome	Classic heat stroke Phenothiazines Anticholinergics Spinal cord injury Bundling	CNS trauma Tumor Encephalitis Stroke

CNS: central nervous system.

Table 27.3 Fever in adults red flags

History	Concerning diagnosis
Tactile or documented fever, reported chills or rigors	Febrile illness (including viral etiology), UTI, pneumonia, sepsis
New medication prescribed or taken (including herbal and OTC)	Drug–drug interaction or adverse drug reaction
Recent surgery	Postoperative infection, DVT, cellulitis, phlebitis, pneumonia (esp. aspiration), UTI
Headache, stiff neck, confusion, weakness	Meningitis, encephalitis, brain abscess or CNS infection
Environmental conditions or extreme activity	Heat stroke, heat exhaustion
Recent foreign travel (despite chemoprophylaxis or vaccination)	Malaria, dengue fever, other travel-associated febrile illnesses
Extremely high fever, especially during or immediately following general anesthesia (esp. if family history)	Malignant hyperthermia
Recent chemotherapy or other treatment for malignancy	Cancer-related fever, tumor fever, neutropenic fever, tumor lysis syndrome, opportunistic infection
Injection drug history, prosthetic or damaged heart valve	Endocarditis, bacteremia, septicemia, epidural or other abscess, osteomyelitis
Examination finding	**Concerning diagnosis**
Rales, rhonchi, asymmetry of lung auscultation	Pneumonia, bronchitis
Flank tenderness (costovertebral angle)	Pyelonephritis, kidney infection, cystitis
Scrotal erythema, swelling, tenderness, lesions	Fournier's disease (necrotizing fasciitis of the scrotum or perineum)
Rash (Table 27.7)	Meningococcemia, purpura fulminans, viral exanthems or enanthems, thrush (oral lesions)
Kernig's or Brudzinski's sign, nuchal rigidity	Meningitis, encephalitis
Cardiac murmur, Janeway lesions, splinter hemorrhages, Roth spots, track marks, "shooter's" abscess(es)	Endocarditis
Back or vertebral (spinal) tenderness	Spinal epidural abscess, osteomyelitis
Abdominal pain	Appendicitis, cholecystitis, diverticulitis, ischemic colitis or mesenteric ischemia, hepatitis, pancreatitis, abscess
Negative examination	Immunocompromised patient may have any of the above findings (or this situation may identify an immunocompromised host)

CNS: central nervous system; DVT: deep venous thrombosis; OTC: over-the-counter; UTI: urinary tract infection.

negative pressure rooms are appropriate for highly contagious diseases such as active tuberculosis, severe acute respiratory syndrome (SARS), and hemorrhagic fevers (such as Ebola or Lassa). Depending on disease incidence and prevalence, many EDs begin isolating patients with fever, cough, or suspicious rash in the waiting room.

The amount of history obtained prior to the initiation of treatment must be tailored to the severity of illness and the potential for life-threatening processes (Tables 27.4 and 27.5).

How long have you been sick? What was your temperature, and how did you take it? Have you appreciated a daily fever pattern?

General characteristics of the fever pattern, magnitude and duration may be of some clinical value. Continuous fevers may be seen in Gram-negative lobar pneumonias, rickettsial disease, typhoid fever and CNS disorders.

Table 27.4 Framework for history of present illness

Fever descriptors
Duration, magnitude, pattern

Contagion risks
- Known sick contacts
- Exposure to nosocomial infections (time spent in nursing home or hospital)
- Travel
- Occupation (restaurant, farm, industry, day care)
- Dietary (raw seafood, home canning)
- Social habits (sex, alcohol, tobacco, IV drug use)

Host factors
- Relevant medical conditions (diabetes, sickle cell, HIV, systemic lupus erythematosus)
- Immunization status
- Medical hardware (indwelling catheter, pacemaker, shunts)
- Medications (including antipyretics)

HIV: human immunodeficiency virus; IV: intravenous.

Table 27.5 Immediate life-threats associated with fever

Associated signs	Concerns
Airway compromise	Epiglottitis, pharyngeal or retropharyngeal abscess, tracheitis
Respiratory distress	Pneumonia, empyema, ARDS
Circulatory collapse	SIRS, sepsis, septic shock
Altered mental status	Meningitis, encephalitis, brain abscess
Peritonitis	Perforated bowel, cholangitis, abscess, SBP, appendicitis

ARDS: adult respiratory distress syndrome; SIRS: systemic inflammatory response syndrome; SBP: spontaneous bacterial peritonitis.

Intermittent relapsing fevers are characteristic (but not diagnostic) of endocarditis, osteomyelitis, and deep tissue abscesses. The maximum temperature reading is an insensitive sign to discriminate a viral from bacterial source. Patients with high fevers tend to appear more ill and have a greater overall incidence of serious bacterial illness (SBI). Purulent complications of common viral upper respiratory tract illness, such as sinusitis, otitis media, or pneumonia, often manifest following a 3–10 day prodrome. Fevers present for greater than 2 weeks should prompt a more comprehensive work-up, focused on identifying inflammatory, immunologic, endocrine, toxicologic, or iatrogenic causes. Although important to patients, response to antipyretics provides little clinical information.

What medications, including antibiotics, do you take? Have you recently stopped any medication?

Most patients are able to describe the medications they take on a regular basis, especially those prescribed by their physician. Patients may not mention medications they take intermittently, and may not offer inhalers, eye drops, oral contraceptives, non-prescription medications, or herbal supplements. Many patients don't consider insulin, aspirin, or acetaminophen as medications. Since many prescription analgesics and over-the-counter cold preparations contain antipyretics, anticholinergics and stimulants, these must be scrutinized. Unless specifically asked, patients may not volunteer that they have taken one or more doses of someone else's (or their own) remaining antibiotics for a fever. This might be an appropriate opportunity to encourage patients to carry an updated list of their medications and allergies at all times.

Have you been exposed to individuals at home, school, or work with similar symptoms? Do you have any special risks for infection?

It is critical to determine whether any special circumstances or contagion risks exist in adults presenting with fever. These include sick household contacts; extended exposure at a hospital, nursing home or day care facility; recent foreign travel; dietary and occupational risks; new or symptomatic prior sexual partners; and intravenous (IV) drug habits. Although most patients can recall a family member, colleague, or friend being ill, patterns of similar symptoms in several close contacts may be helpful. This is true of individuals who camp, travel, or eat together. High-risk dietary habits include eating raw or undercooked meats or fish, home canning and "direct from source" food use (milk, honey, chickens).

Do you have any chronic medical conditions that may make you susceptible to infection? Do you take steroids?

A multitude of comorbidities predispose patients to infection through their immune system's inability to access the affected area, as in the case of peripheral vascular disease. Defenses are attenuated in individuals with diabetes, or deliberately suppressed in organ transplant recipients. Patients without a spleen (or functionally asplenic) are at increased risk of overwhelming infection and sepsis. Chronic steroid use diminishes already vulnerable host defenses.

Do you have any indwelling or implanted medical devices?

Implanted medical devices have great potential for hematogenous contamination in the setting of transient bacteremia. Non-native heart valves and indwelling vascular and urinary catheters are at greatest risk. Implantable cardioverter-defibrillators (ICDs), pacemakers, breast and penile implants, artificial joints, nerve stimulators and medication pumps are particularly vulnerable immediately following placement. Fortunately, their seeding potential diminishes after several months, although it does not disappear.

Are your immunizations current?

Immunization status is important and should include questions regarding childhood vaccination series, subsequent titers, hepatitis B series, Pneumovax, tetanus boosters, and influenza prophylaxis. Recent immunizations prior to foreign travel should be investigated. Vaccine research continues that may alter our current approach to certain diseases, and vaccines exist that protect us from biological agents.

Associated symptoms

Obtaining a detailed history often directs the physical examination to findings that help identify the source of the fever (Table 27.6). Fever is a systemic sign that should force clinicians to consider causes from head to toe, including the skin. Physicians must also consider external sources, like drugs or drug–drug interactions. It is therefore prudent to organize an approach to the history by physiologic systems as opposed to anatomical location. Each organ system will have characteristic but non–pathogen-specific signs and symptoms.

Table 27.6 Associated localizing symptoms by organ system

Respiratory	Dyspnea, cough, phlegm, sputum, pleuritic pain
Gastrointestinal	Nausea, vomiting, abdominal pain, cramping, diarrhea, anal pain, itching, blood, jaundice
Skin/soft tissue	Pain, rash, redness, streaks, warmth, induration, drainage, mass, edema
Musculoskeletal	Pain on movement or palpation, swelling, inability to bear weight, decreased ROM
Genitourinary	Dysuria, frequency, urgency, discharge, dyspareunia, flank pain, pelvic pain
Head and neck (including CNS)	Nasal drainage, tooth, throat or ear pain, difficulty swallowing, confusion, neck stiffness, vomiting, headache, back pain

CNS: central nervous system; ROM: range of motion.

Past medical

An extensive list of comorbidities exists that impede normal defense mechanisms and place patients at risk from their fever or associated serious illness. These include chronic cardiopulmonary disease, cancer, chronic renal failure, human immunodeficiency virus (HIV), diabetes mellitus (DM), hemoglobinopathies, and transplanted (or removed) organs. Recent surgery, childbirth, or exacerbation of chronic conditions may lower host defenses. Prior infections recently treated with antibiotics place patients at increased risk for developing drug-resistant organisms, including methicillin-resistant *Staphylococcus aureus* (MRSA), drug-resistant *Streptococcus pneumoniae* (DRSP), vancomycin-resistant enterococci (VRE) and fluoroquinolone-resistant enterics. The loss of protective non-pathogenic flora may result in vaginitis, colitis, or esophagitis.

Physical examination

A thorough history allows clinicians to concentrate on target areas of the physical examination. Local inflammatory-mediated changes provide clues to the potential source of fever. If localized findings are not identified, treatment is based largely on host factors. Young, previously healthy adults with physical examination findings consistent with benign viral illnesses (nasal discharge, congestion, cough, diffuse myalgias and arthralgias, or non-bloody loose stools) can be safely discharged with symptomatic therapy and close follow-up. In contrast, the elderly or immune-compromised hosts with few historical or physical examination findings require consideration for further work-up, cultures, empiric antibiotic treatment, and possible admission or observation.

General appearance

The term "toxic-appearing" applies to those who look ill. Behavioral clues such as responsiveness to voice, posture, hygiene and energy level provide insight into illness severity. The inability to sit up in a stretcher or ambulate to the bathroom without assistance is a gross marker of functional impairment.

Temperature

Factors influencing temperature measurement include environmental conditions, testing technique, anatomic site and site-specific confounders. The distinction of core versus shell temperature is most important in extreme states of hyper- and hypothermia. Invasive methods to determine temperature include pulmonary artery catheters and esophageal or urinary bladder probes, although these are usually not necessary. Temperatures are typically obtained from the rectum, mouth, tympanic membrane, axillae, or forehead, listed in decreasing reliability.

Tympanic thermometers have gained popularity because they are noninvasive and calibrate quickly, and do not rely on direct contact with the tympanic membrane. Errors occur through improper positioning, anatomic abnormalities, cerumen obstruction, or local inflammatory processes such as otitis media or externa. Many authors oppose tympanic thermometers as a screen for fever in the ED, especially in the elderly.

Rectal, oral and tympanic temperatures correlate poorly with one another. On average, rectal temperatures are 0.8°F (0.4°C) higher than oral and 1.6°F (0.8°C) higher than tympanic readings. Rectal temperatures are maximal at a depth of 2.5 inches (6.4 cm). Fecal impaction and shock states may falsely reduce rectal measurements, whereas elevated fecal bacterial counts and proctitis may erroneously increase readings. Sublingual or oral temperature measurements require cooperative patients able to breathe with their mouths closed. Mastication, smoking, recently ingested liquids and foods, and respiratory distress affect these readings.

Because temperatures can vary during time spent in the ED, it may be prudent to repeat temperature measurements. A high degree of clinical suspicion in the setting of a normal temperature should prompt at least one or more rechecks during a patient's stay, and consideration of measurement at a different site. Patients at the extremes of age or with significant comorbidity may have a serious infection despite the absence of fever.

Vital signs

Review of the remaining vital signs (Chapter 1) may provide clues to the source of fever. For example, an increased respiratory rate with low oxygen saturation suggests pneumonia or other respiratory causes of fever.

Central nervous system

Despite its protective barriers, devastating CNS infections can occur as a result of direct (e.g., otitis media, sinusitis) or hematogenous spread of pathogens. Fever may impair cognitive function, particularly in the elderly, resulting in agitation or decreased responsiveness. This makes discerning a primary CNS infection from a systemic problem difficult.

An effort should be made to detect signs of meningeal irritation or increased intracranial pressure (ICP). *Meningismus* (pain due to irritation of the meninges surrounding the brain and spinal cord) is often a late, nonspecific finding; it is unreliable below 18 months of age. Maneuvers that demonstrate meningismus include difficulty with or resistance to neck or hip flexion. Increased ICP, if progressing slowly, may manifest subtly as mental status or personality changes, a bulging fontanelle, papilledema, or focal neurologic abnormalities. The absence of signs of increased ICP or abnormal neurologic findings does not exclude a serious CNS infection. Fever and an abnormal sensorium or neurologic examination should raise immediate concern for meningoencephalitis, with prompt consideration of empiric antimicrobial or antiviral therapy. In the setting of a clinically suspected CNS infection, broad-spectrum antibiotics that cross the blood–brain barrier should be administered prior to lumbar puncture (LP). Despite controversy, IV dexamethasone is recommended prior to antibiotics in cases of acute bacterial meningitis.

Upper respiratory tract

The upper respiratory tract is colonized by diverse flora. A delicate balance exists at this strategic opening to the respiratory and digestive systems. Droplet transmission of potential pathogens upon inhalation initially seeds the nasal mucosa. As a result, the upper respiratory tract is the most commonly infected site in humans.

A detailed examination should include the eyes and surrounding structures, the nares, and the oropharynx, including teeth, gums, tonsils and mucosal surfaces. Tender anterior cervical adenopathy may provide clues to a current or recent facial or oral infection. The paranasal sinuses should be palpated or percussed gently. The ear canal, tympanic membrane, mastoid bone and posterior cervical lymph nodes should also be evaluated. Skin abscesses can manifest anywhere on the body and may be hidden behind an ear, beneath the hair, or in the perianal or rectal area. Neck stiffness may be a manifestation of severe pharyngitis or soft tissue neck abscess, or a component of diffuse myalgias. Palpation of the salivary, parotid and thyroid glands, and auscultation of the upper airway complete this portion of the examination.

Lower respiratory tract

Acute bronchitis and pneumonia are common infections resulting from translocation of upper respiratory flora. These may occur from the aspiration of gastric contents. Smoking renders the lower respiratory tract extremely vulnerable to pathogens. An overall global assessment of a patient's respiratory status includes his or her work of breathing (WOB), respiratory rate, pulse oximetry and breath sounds. A normal pulse oximetry accompanied by tachypnea, abnormal breath sounds, or increased WOB provides little reassurance. Asymmetric breath sounds or rhonchi suggest air space disease, air sac collapse, or an effusion. A comprehensive lung examination includes auscultation anteriorly, posteriorly, and in the axillae in multiple locations within these areas, with intentional cough, phonation and forced exhalation to elicit sounds not otherwise heard. Sputum, if produced, may be collected in a sterile container for Gram stain and culture, although this is rarely helpful in the ED.

Cardiovascular

A pleural or pericardial rub may point to inflammatory processes. Any new murmur, especially in the setting of IV drug use, should prompt immediate concern for endocarditis, as fever may be the only symptom. Evidence of impaired cardiac function such as congestive heart failure (CHF) may indicate myocarditis. Rheumatic heart disease (RHD) or known cardiac valve abnormalities should also be considered.

Gastrointestinal

Analogous to the skin, the gut represents an important protective barrier against pathogens. Any weakening from systemic or local bowel disease can allow translocation of intraluminal disease-causing agents. The first concern is whether or not peritonitis is present. Reflexively or persistently increased abdominal wall muscle tone, worsening pain with movement of the abdomen, and the loss of bowel sounds in the appropriate setting should prompt surgical consultation. Evaluation of the abdomen by quadrant helps narrow the list of abdominal causes of fever, although anatomic overlap and referred pain must be considered. Special consideration should be made to examine liver size, texture and tenderness; gallbladder prominence and tenderness with and without inspiration; pain isolated to the right or left lower quadrants; or unilateral flank tenderness. A rectal examination is needed to identify a perirectal abscess or prostatitis. If a patient is (or might be) neutropenic, a rectal examination should be deferred, other than inspection. The perineum should be visually inspected for soft tissue abnormalities.

Genitourinary

Urinary tract infections (UTIs) proceed in an ascending manner from the introitus. Initially, local irritation or burning at the urethral meatus can progress to classic signs of cystitis (dysuria, hesitancy, urgency and frequency). Flank pain may represent irritation of the kidney's fibrous capsule due to pyelonephritis. Upper tract signs are not reliable to distinguish cystitis from pyelonephritis. A thorough inspection of the foreskin, glans, penis and scrotal structures in males, as well as inguinal lymph nodes, is important to identify sources of fever. In females, inspection of the external genitalia, vulva and labia, as well as a speculum and bimanual examination, are important given the high prevalence of pathogens transmitted by sexual intercourse.

Soft tissues and musculoskeletal

Diffuse transient myalgias and arthralgias are common with many pathogens. However, isolated pain localized to the soft tissue or one joint should be investigated thoroughly. Special concern includes the perineum, feet, and areas of continuous external pressure. All debilitated patients need to be rolled on both sides using at least two providers to examine these areas, especially the sacrum. When examining cutaneous wounds, look for drainage, spreading erythematous margins, crepitus and functional impairment. Recent shaving, body piercing and tattoos can serve as a nidus of infection or inflammation. Skin abscesses, carbuncles, or furuncles from "skin popping" are common in the forearms of IV drug users. Abscesses may occur anywhere, including the axillae, breast, groin, buttock and labia.

Skin

Characteristic skin lesions often accompany febrile adults. It is important to use proper dermatologic terminology so that rash categorization algorithms are utilized appropriately. A wide variety of infectious and non-infectious conditions manifest as skin lesions (Table 27.7). Petechiae are worrisome vasculitic lesions in the setting of a fever, and may be seen in early meningococcemia.

Table 27.7 Infections presenting with fever and rash

Maculopapular
Central
Rubeola
Rubella
Roseola
Erythema infectiosum
Lyme disease
Drug reactions
Peripheral
Erythema multiforme
Secondary syphilis
Petechiae
Meningococcemia
Rocky Mountain spotted fever (RMSF)
Viral hemorrhagic fevers
Thrombotic thrombocytopenic purpura (TTP)
Diffuse erythema
Scarlet fever
Toxic shock syndrome (TSS)
Scalded skin syndrome
Ehrlichiosis
Vesicles/pustules
Varicella
Herpes zoster
Gonococcemia
Impetigo
Nodules
Erythema nodosum
Fungus

Differential diagnosis

The differential diagnosis of the febrile adult is protean and includes a large number of both infectious and non-infectious causes. It is important to focus on:

1. What illness or injury may have occurred causing host vulnerability and allowing the infection to develop;
2. What site-specific pathogens are usually responsible for disease and exist in the local community;
3. What special exposure risks may have occurred from professional or personal habits.

The most difficult diagnoses to establish are in patients with systemic illness without an obvious cause or portal of entry. This should prompt a broad search for suspected causes. From an emergency physician's (EP's) perspective, it is generally best to organize this search by organ system (Table 27.8). Local Departments of Health require physicians to report infectious diseases that are of public health concern.

Infections can be divided into viral, bacterial, fungal and parasitic. Viral infections are the most common, and include a variety of upper respiratory, gastrointestinal, blood-borne, CNS, skin and genital pathogens. A variety of hemorrhagic and mosquito-borne viral illnesses occur, such as West Nile Virus, rabies and H1N1.

Bacterial illness is typically divided into Gram-positive skin and respiratory organisms and Gram-negative gastric and urinary pathogens. The disruption or translocation of ordinary flora is often the cause of disease. Encapsulated organisms pose a special threat to patients without a spleen (e.g., sickle cell disease, surgical removal). Prompt initiation of antibiotics greatly reduces mortality but is not a benign practice overall, as it has resulted in the evolution of drug-resistant organisms. The practice of treating unknown or non-infectious illnesses with antibacterial agents in healthy adults is not supported in the literature.

Most EDs have universal precaution, isolation and environmental control policies to prevent the spread of drug-resistant organisms. Fungi can cause devastating disease in immunosuppressed hosts, but rarely have significant impact on healthy individuals. In the United States, parasitic infections are generally limited to adverse social conditions or special occupational and recreational exposures. These typically require special vectors for transmission. As with all infectious diseases, a careful exposure history including recent travel is important.

Non-infectious causes of fever

Medication/drug fever

Often a diagnosis of exclusion, drug fever is not always identified during a single ED visit. Fevers may develop from the administration of irritating substances that cause phlebitis, sterile abscesses, or aseptic meningitis. Other drugs may have intrinsic properties that interfere directly in the thermoregulatory process. A classic febrile response to the treatment of syphilis is the *Jarisch–Herxheimer* reaction. Febrile reactions to inhalational anesthetics and agents that induce red cell hemolysis can occur in individuals with certain genetic predispositions. Patients with drug fever do not always appear well, and may have relative bradycardia, eosinophilia, or cutaneous signs. Common drugs associated with drug fever include cardiovascular agents α-methyl dopa, procainamide and quinidine; antineoplastic agents; antibiotics; antiepileptics; and cimetidine. Drug combinations that increase levels of synaptic serotonin induce autonomic hyperactivity, including fever. Exposure to neuroleptics may induce a progressive state of fever and rigidity, although this is rare.

Table 27.8 Differential diagnosis of infectious causes of fever

Diagnosis	Symptoms and signs	Etiologic agents	Work-up considerations
Systemic			
AIDS	Altered mental status, dyspnea, diarrhea, vesicular rash	HIV	Western blot, ELISA, CBC, CD4, viral load, CXR, ABG, acid-fast sputum, brain CT with contrast precedes LP, fecal leukocytes
Malaria	Malaise, myalgias, headache, chest pain, diarrhea, anemia, hepatosplenomegaly	*Plasmodia* species	Travel history, CBC, thick and thin blood smears
Rabies	Malaise, paresthesias, agitation, coma	Rabies virus	PCR saliva
Sepsis	Altered mental status, ↓BP, respiratory distress, tachycardia	Gram-positive, Gram-negative, rarely anaerobes, fungus	CBC, blood and urine cultures, CXR, lactate
Tetanus	Muscle rigidity, lockjaw, sardonic smile, opisthotonus	*Clostridium tetani*	Electrolytes, control of airway and muscle spasms
Toxic shock syndrome	↓BP, erythroderma, desquamation, sore throat, diarrhea, myalgias	*Staph. aureus* (toxin-mediated) Groups A, B, C, G *Strep. pyogenes*	Remove any FB, CBC, blood cultures
Head and neck			
Brain abscess	Altered mental status, headache	Streptococci, bacteroides, Enterobacteriaceae, *Staph. aureus*, *Toxoplasma gondii* (if HIV+)	Brain CT (contrast may be needed), MRI
Epidural abscess	Fever, back pain, signs of spinal cord involvement	*Staph. aureus*, *Pseudomonas*, TB	MRI, spine CT, myelogram, LP
Meningitis/encephalitis	Meningismus, headache, altered mental status, nausea, vomiting, seizure	Depends on patient's age, chronicity, and immunocompetence: viral, *Strep. pneumoniae* (especially post-neurosurgical or CSF leak), *N. meningitidis*, *Listeria monocytogenes* (unlikely if young and immunocompetent), Gram-negative bacilli (*H. influenza* rare), *Herpes simplex*, chemical/drug	Brain CT (in select patients); LP with CSF for cell count and differential, Gram stain, culture, protein, glucose, LDH, viral cultures, India ink, cryptococcal antigen, and special studies
Periorbital/orbital cellulitis	Facial swelling, painful eye movement	*Strep. pneumoniae*, *H. influenzae, M. catarrhalis*, *Staph. aureus*, anaerobes, Group A Streptococcus, occasional Gram-negative bacilli post-trauma	Facial CT if concerned with orbital cellulitis or abscess
Respiratory			
Lung abscess/empyema	Toxic, dyspnea, chest pain	*Strep. pneumoniae*, Group A *Streptococcus*, *Staph. aureus*, *H. influenzae*	CXR, thoracentesis, drainage (chest tube thoracostomy, IR or surgery)
Pharyngeal abscess	Stridor, sore throat	*Strep. viridans*, Group A *Streptococcus,* Mixed oral flora	Soft tissue neck X-ray, neck CT, I&D
Pneumonia	Cough, sputum, dyspnea, rhonchi, chest pain	Factors include community- or hospital-acquired, tobacco, age, and comorbidities: viruses, mycoplasma, *Strep. pneumoniae, H. influenzae, M. catarrhalis,* TB, *Legionella* and *Pseudomonas* species	CXR
Sinusitis/otitis media/ pharyngitis/dental	Localized pain	*Strep. pneumoniae, H. influenzae, M. catarrhalis*, Group A *Streptococcus*	Limited sinus CT, throat cultures
URI/bronchitis	Cough	Viruses	None
Cardiac			
Endocarditis	Murmur, microemboli, myalgias, weakness, Osler's nodes, Roth's spots, Janeway lesions, petechiae	Depends on native or prosthetic valve, IV drug abuse*: Strep. viridans, S. bovis, S. epidermidis, Staph. aureus*, other streptococci, enterococci, and Enterobacteriaceae species	CBC and blood cultures, TEE, ID consultation recommended

(*continued*)

Table 27.8 Differential diagnosis of infectious causes of fever (*cont.*)

Diagnosis	Symptoms and signs	Etiologic agents	Work-up considerations
Gastrointestinal			
Colitis	Abdominal pain, diarrhea	Enterobacteriaceae, anaerobes	*C. difficile* toxin
Diverticulitis/abscess	Abdominal/pelvic pain, peritonitis	Enterobacteriaceae, enterococci, bacteroides species, anaerobes	CT scan, surgical consultation (possibly IR)
Gastroenteritis	N/V/D	Viral, enteric bacteria, including *Shigella, Salmonella, E. coli* O157:H7, *Campylobacter, C. difficile, E. histolytica*, parasites	Fecal leukocytes, stool tests, *C. difficile* toxin, possibly stool culture (although rarely helpful in most circumstances)
Hepatitis	N/V/D, jaundice	Hepatitis A, B, C, D	Hepatitis screen, LFTs
Spontaneous bacterial peritonitis	Abdominal pain, distention, ascites	Enterobacteriaceae*, Strep. pneumoniae*, enterococci, anaerobes	Paracentesis, cell count, Gram stain/culture
Genitourinary			
Epididymitis/orchitis	Testicular pain	Mumps, treponema, other viruses	Urine dip, scrotal US to rule out abscess or testicular torsion
Herpes simplex	Burning pain, itching	HSV 1, 2	Tzanck, DFA
Pelvic inflammatory disease/tubo-ovarian abscess	Purulent vaginal discharge, cervical motion tenderness, abdominal pain, shuffling gait	*N. gonorrhoeae, C. trachomatis,* bacteroides, Enterobacteriaceae, Streptococci, *Trichomonas vaginalis*	UA, DNA probe, swabs (cervical, rectal, throat), CBC (ESR, CRP more common at many centers), pelvic US, surgery or IR (if abscess)
Perirectal abscess	Pain, purulent drainage	Enterobacteriaceae, occasional *P. aeruginosa*, bacteroides species, enterococci	I&D
Prostatitis	Dysuria, abdominal pain	Etiology depends on age, chronicity, and sexual practices: *N. gonorrhoeae, C. trachomatis,* Enterobacteriaceae (coliforms), other (including unknown)	Urine dip
Pyelonephritis/abscess	Dysuria, flank pain	Enterobacteriaceae (*E. coli*), enterococci, occasional *P. aeruginosa*	Urine dip, UA, urine culture, renal CT
1° Syphilis, chancroid, lymphogranuloma venereum (LGV)	Chancre (painful versus nonpainful), lymphadenopathy	*Treponema pallidum, Hemophilus ducreyi, Chlamydia trachomatis*	RPR, VDRL, FTA-ABS, DNA probe
Urethritis	Urethral discharge (may be age- and activity-related, scant, mucoid, or purulent)	Chlamydia, Trichomonas	Urethral swab for GC, urine tests in some centers
Skin/soft tissue			
Cellulitis/fasciitis	Pain, swelling, redness, possible drainage, occasional crepitus	Group A *Streptococcus*, other *Streptococcus* species, *Staph. aureus*, anaerobes, *Clostridium* species (fasciitis is often polymicrobial)	CBC, I&D, fasciotomy
Folliculitis/skin abscess	Pain, swelling, redness, possible drainage	*Staph. aureus*, *S. epidermidis,* candida, anaerobes, *P. aeruginosa* (hot tub)	I&D, US if concern for FB
Osteomyelitis	Pain	*Staph. aureus, P. aeruginosa,* may be polymicrobial (especially if chronic); *Salmonella* common in sickle cell anemia; *S. epidermidis* possible postoperative	I&D
Septic arthritis	Pain with range of motion	*Staph. aureus, N. gonorrhoeae*, Streptococci, anaerobes	Aspiration via arthrocentesis, cell count, Gram stain, culture of synovial fluid

ABG: arterial blood gas; AIDS: acquired immune deficiency syndrome; CBC: complete blood count; CP: chest pain; CSF: cerebrospinal fluid; CXR: chest X-ray; CRP: C-reactive protein; CT: computed tomography; DFA: direct fluorescent antibody; ELISA; enzyme-linked immunosorbent assay; ESR: erythrocyte sedimentation rate; FB: foreign body; FTA-ABS: flourescent treponemal antibody absorbed; GC: gonococcus; HIV: human immunodeficiency virus; HSV: herpes simplex virus; I&D: incision and drainage; ID: infectious disease; IR: interventional radiology; LDH: lactate dehydrogenase; LFTs: liver function tests; LP: lumbar puncture; MRI: magnetic resonance imaging; N/V/D: nausea/vomiting/diarrhea; PCR: polymerase chain reaction; RPR: rapid plasma reagin; TB: tuberculosis; TEE: transesophageal echocardiography; UA: urinalysis; URI: upper respiratory infection; US: ultrasound; VDRL: Venereal Disease Research Laboratory.

Thromboembolic disease

Between one-quarter and one-third of patients with documented pulmonary embolism (PE) will have a low-grade fever (<101°F or 38.5°C). Many conditions that predispose patients to PE, such as malignancy or deep venous thrombosis (DVT), may be responsible for this. There is no difference in frequency of fever in complications such as pulmonary infarction. The presence of thrombophlebitis may also produce temperature elevation.

Tumor fever

A variety of solid organ and hematogenous neoplasms cause persistent fever. Fever is common among leukemias and lymphomas due to the proliferation of neoplastic cells capable of endogenous pyrogen release. Liver, CNS, and renal cell cancers also produce temperature elevations. Because both the cancer and its treatment cause immune compromise, it is important to rule out infection in individuals with cancer and fever. Neutropenic patients who develop fever should be thoroughly investigated for a source of infection. Broad-spectrum empiric antibiotics should be administered promptly after appropriate cultures are obtained, even in the absence of a source.

Inflammatory/immunological disease

Diseases that induce a chronic inflammatory state may manifest with fever. These include systemic lupus erythematosus (SLE), juvenile rheumatoid arthritis (JRA) and polyarteritis nodosa (PAN). Although localizing signs may be present, the systemic nature of these conditions cause diffuse symptoms. A relative immunodeficient state exacerbated by immunosuppressive therapy increases the risk of infection, particularly from Gram-negative bacteria and fungi. A history of inflammatory bowel disease should raise concern for an intra-abdominal abscess. Pancreatitis often leads to a cascade of enzymatic autodigestion and profound inflammatory-mediated third-space fluid losses, resulting in shock. Many vasculitides present with unexplained low-grade fever.

Environmental/occupational

Heat stroke is divided into classic and exertional forms. *Classic heat stroke* typically affects deconditioned or elderly individuals; those with chronic morbidity, or a history of drug or alcohol use; and individuals on medication that may exacerbate fluid losses or prevent heat dissipation. *Exertional heat stroke* occurs more commonly in individuals involved in endurance athletics. Despite the fact that exertional heat stroke patients retain the ability to sweat, they have overwhelmed their reparative mechanisms and require aggressive management. Metal and polymer fume fevers are examples of environmental exposures that can masquerade as flu-like illnesses with fever; welders are at particular risk. Aerosolized metals induce symptoms that may be delayed by several hours, often lasting 1–2 days until the exposure is discontinued.

Miscellaneous

Patients with a variety of conditions responsible for a hypermetabolic state may present to the ED with fever. Hyperthyroidism, pheochromocytoma, carcinoid syndrome and anaphylaxis increase basal metabolic rate as well as catecholamine secretion. Hyperlipidemia has also been shown to result in fever. Although uncommon, factitious fever, Munchausen syndrome, and Munchausen syndrome by proxy are additional etiologies.

Diagnostic testing

Laboratory studies

Prior to ordering ancillary studies, EPs should consider the broad array of treatable diseases being ruled in or out. Objective data should be viewed as confirmatory of the history and physical examination. In the majority of febrile patients, the source of illness is determined clinically, prior to objective testing. Examples of conditions occurring in healthy hosts that do not require confirmatory diagnostic testing include rhinitis, bronchitis, gastritis and uncomplicated enteritis.

When laboratory studies are indicated, every effort should be made to obtain adequate sample quantities of appropriate specimens. This includes specimens collected correctly, using acceptable media. Failure to collect specimens prior to the initiation of antimicrobial therapy may interfere with the future clinical picture. Specimens include blood, urine, sputum, stool, cerebrospinal fluid (CSF), throat, penile, cervical and wound swabs, and pleural, peritoneal, or joint aspirates.

Selected blood tests

Complete blood count

The complete blood count (CBC) is neither sensitive nor specific for distinguishing bacterial from nonbacterial illness. Cutoff values of white blood cell (WBC) counts greater than 15,000/mm^3 suggest a higher likelihood of serious illness. Leukocytosis initially occurs through demargination of existing mature WBC stores, then by increased bone marrow production and release of mature and immature forms (left shift). Demargination and leukocytosis as high as 30,000 WBC/mm^3 occur under many conditions, including physiologic stress. Emotional upset, glucocorticoid use, myeloproliferative disorders and pregnancy may result in leukocytosis. Automated WBC typing accompanies the standard CBC report. Manual differentials that identify immature forms are generally only performed above laboratory-established total cell count cutoffs, unless specifically requested. A low WBC level may represent transient viral bone marrow suppression or the host's inability to mount a significant immune response. WBC counts greater than 40,000/mm^3 can occur in severe infections, but should prompt consideration for a myelodysplastic disorder or leukemia. The WBC is important to exclude neutropenia in patients undergoing chemotherapy.

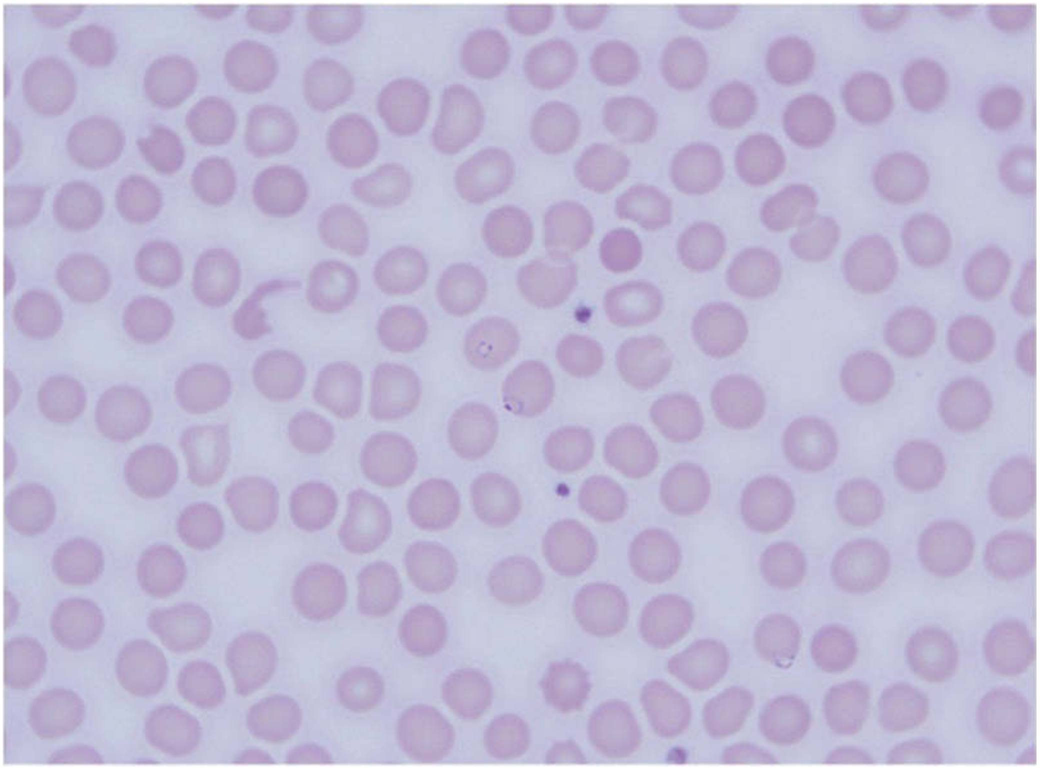

Figure 27.1
A Giemsa-stained peripheral blood smear revealing numerous ring forms of Plasmodium falciparum.
Courtesy: S.V. Mahadevan, MD and Niaz Banaei, MD

Acute phase reactants

C-reactive protein (CRP), serum amyloid A, pro-calcitonin, haptoglobin and fibrinogen are examples of acute phase reactants. Pyrogenic cytokines stimulate production of this group of proteins during the acute inflammatory process. Literature demonstrates that these tests add little to the clinical impression except in special circumstances. These serum markers may be useful to follow the clinical progression of complex infections such as epidural abscesses, septic arthritis and chronic inflammatory conditions (rheumatoid arthritis or temporal arteritis). The erythrocyte sedimentation rate (ESR) is an indirect marker of the level of acute phase proteins in the blood, but suffers the same limitations as the direct markers. None of these tests is highly specific. Decisions regarding treatment or disposition are rarely based solely on these tests.

Blood cultures

Blood cultures should be drawn prior to antibiotic therapy on patients with a likelihood of bacteremia. Two sets of blood cultures, two bottles each (one anaerobic and one aerobic), with 15 mL of blood in each set should be drawn from two distinct puncture sites. One site should include any indwelling vascular catheter. The positive rate of blood cultures in the ED is approximately 10%, with many of these growing contaminants. Therefore, care should be taken to order blood cultures selectively. Blood cultures drawn from afebrile patients are of limited value. Blood cultures should be obtained on patients being admitted for systemic infection, patients with impaired defenses placed on empiric antibiotics, and patients who may have uncommon or atypical bacterial growth. Blood cultures for patients with pneumonia are a core measure, and they should be obtained for patients with suspected systemic inflammatory response syndrome (SIRS), sepsis, or septic shock.

Thin and thick smear

If the patient's fever might be due to malaria, given exposure or travel to an endemic area, both thin and thick smears of blood should be obtained. Microscopic examination of these smears is the easiest and most reliable test for malaria. The thick blood smear is most useful for detecting parasites, because it examines a larger sample of blood. The thin smear helps identify the malaria species responsible for infection (Figure 27.1). A single normal smear (parasites absent) does not rule out malaria; smears should be repeated at 12- to 24-hour intervals for 48–72 hours in patients with suspected malaria. Asymptomatic parasitemia may be common in children from endemic areas with partial immunity.

Other selected specimen tests

Stool

Bacterial enteritis is less common in the United States than other parts of the world. Stool specimens for culture are generally not necessary in the setting of a well-appearing patient with non-bloody diarrhea. Fever, abdominal cramping, mucous or pus, immune compromise and recent foreign travel make a bacterial cause more likely. Potential exposure to certain pathogens, as might occur during a recent camping trip, may prompt further testing. Fecal leukocyte detection (enteroinvasive disease) represents the best screening test for a potential bacterial cause of diarrheal illness. Routine stool cultures can identify common bacterial pathogens including *Campylobacter*, *Salmonella* and *Shigella*. Special specimen processing is necessary to serotype *Escherichia coli* O157:H7. Stool ova and parasites can be ordered if *Giardia* or other parasites are suspected. *Clostridium difficile* toxin detection should be checked for patients with possible toxic colonic bacterial overgrowth due to recent antibiotic use.

Cerebrospinal fluid (CSF)

Lumbar puncture is necessary in all patients with suspected CNS infection, especially those with fever, altered sensorium, headache, neurologic changes, meningismus, or localized spinal tenderness. At least 10 mL of CSF should be collected, with care taken not to contaminate the specimen with skin flora or blood. Repeat puncture attempts increase the likelihood of a traumatic (bloody) tap, which may diminish the interpretation accuracy (Table 27.9).

Table 27.9 Lumbar puncture order set

Tube 1	Cell count and differential
Tube 2	Protein, glucose
Tube 3	Gram stain, bacterial culture, viral culture (herpes)
Tube 4	India ink, cryptococcal antigen, bacterial antigen (CIE, ELISA); repeat cell count and differential

CIE: countercurrent immunoelectrophoresis; ELISA: enzyme-linked immunosorbent assay.

Joint aspiration

Localized pain, erythema and swelling of a joint causing painful or limited range of motion in the setting of systemic fever should prompt consideration of a septic joint until proven otherwise. Aspiration and analysis of synovial fluid is crucial to the diagnosis, as this is the only manner in which an infectious etiology can be distinguished from a sterile process, such as aseptic synovitis or crystal arthropathy. Bedside ultrasound may be used to guide sample collection. Joint aspirates should be tested for cell type and count, bacteria on Gram stain, culture, and crystals under polarizing microscopy. Leukocyte counts greater than 50,000/mm^3 are likely the result of infection, prompting immediate antibiotics and orthopedic consultation. Bacterial infections can occur with lower leukocyte counts, however. Cultures have a high rate of detecting *Staphylococcus* and *Streptococcus*, but are less sensitive for gonorrhea.

Radiologic studies

Refer also to Table 27.8 or specific chapters describing primary complaints that cause fever in adults.

Chest X-ray

The chest X-ray is the gold standard for the identification of pneumonia. Findings may not appear on radiograph until symptoms have been present for several days. Two views of the chest (posteroanterior [PA] and lateral) are generally of better quality than a portable anteroposterior [AP] view, and a retrocardiac process may be seen more readily on the lateral view. However, the AP view is generally sufficient in most patients, and may be obtained more efficiently, with added patient safety, monitoring and decreased radiation exposure. Community-acquired pneumonia (CAP) may be lobar or a diffuse interstitial process (often referred to by patients as "walking pneumonia"). Focal infiltrates represent consolidation and fluid collection within air spaces, resulting in the loss of air-solid interfaces. Chest X-ray may not help distinguish infection (bacterial, viral, or fungal) from other causes of consolidation, such as atelectasis (air sac collapse). Pulmonary complications, such as empyema (abscess), lobar collapse, mediastinal adenopathy or pleural effusion, may be seen. Endobronchial processes, such as bronchitis or reactive airway disease, rarely demonstrate clinically important radiographic findings, although hyperinflation or peribronchial fluid may be identified. Female patients who are or might be pregnant should have appropriate shielding with lead to reduce radiation exposure to the fetus or reproductive organs.

Additional plain films

Soft tissue neck X-rays may help with the diagnosis of epiglottitis. The classic thumbprint replaces the normally thin epiglottic shadow. The prevertebral soft tissue space thickness may be increased due to retropharyngeal abscess; air in these soft tissues may represent infection by a gas-forming organism. Plain films of suspected infected extremities are usually unnecessary unless a suspicion of foreign body, necrotizing fasciitis, or long-standing deep infection exists. Osteomyelitis is the slow destruction of bony architecture that may be apparent after more than 1 week of fever and symptoms.

Ultrasound

Ultrasound (US) can accurately and rapidly identify fluid-filled structures such as deep tissue abscesses (perinephric, subhepatic, tubo-ovarian) without risk of radiation exposure. US is the modality of choice for identifying biliary tract disease. Gallbladder wall thickening greater than 3 mm, pericholecystic fluid or ductal dilatation in a patient with pain, right upper quadrant tenderness and fever (with or without gallstones) should prompt surgical consultation for acute cholecystitis or cholangitis. Increased availability of focused bedside sonography in the ED and emergency physicians' increased skills using this imaging modality has resulted in its greater use. Current applications of US for emergency medicine practice include the identification and/or drainage of abscesses, ascites, joint aspiration, foreign body and deep venous thrombosis, all which may cause fever in adults. Many clinicians now use US to assist with landmark identification for lumbar puncture.

Computed tomography

Computed tomography (CT) scanning provides definitive anatomical information regarding deep infections, especially in those areas inaccessible by US or obscured by bone or air-filled structures, such as the brain, paranasal sinuses, or deep abdomen. The appropriate contrast material and route of administration (PO, IV, rectal) should be determined based on the most likely etiology of fever, according to institutional protocols. All patients must be evaluated for potential contraindications to contrast or radiation, such as prior contrast allergy, pregnancy, or renal insufficiency. Patients taking metformin should be instructed to hold their dose for 48 hours, drink plenty of fluids, and monitor their sugars carefully. Most CT scanners have weight restrictions, often based on the patient's abdominal girth and the ability of the gantry to move a patient into the proper position.

Additional radiologic tests

Magnetic resonance imaging (MRI) is rarely necessary for the initial work-up of an adult with fever. One exception to this would be if spinal epidural abscess is suspected. Hepatobiliary iminodiacetic acid (HIDA) scanning or other radionuclide imaging of the gallbladder may be indicated if acute cholecystitis is suspected but not identified using US or CT scan. Radionucleotide-tagged bone scintillography (to rule out osteomyelitis) may be useful to isolate a recurring fever of unknown etiology.

General treatment principles

The duration of patient interview is directly related to the severity of illness, which is most often related to the host's ability to fight disease rather than the particular pathogen. Febrile adults presenting with abnormal vital signs, altered sensorium, airway compromise, respiratory or circulatory distress require rapid simultaneous diagnostic testing and resuscitative therapy. Recognition of the severity of illness and immediate transfer to a resuscitation area in the ED are essential. Managing the ABCs (airway, breathing, circulation) takes precedence over treating the fever itself. Immediate interventions include assuring airway patency, providing supplemental oxygen, supporting ventilatory efforts, and obtaining adequate vascular access for fluid resuscitation.

Further history gathering may be limited by the severity of illness, resulting in the need for alternative sources of pertinent history. These include reports from emergency medical services, accompanying family members, medical records, or the primary care or specialty physician. Families of elderly patients often understand that infection is a frequent cause of disability or death. Care must be taken to honor existing advance directives prior to initiating invasive diagnostic and stabilizing measures.

Antipyretics

The administration of antipyretics for fever to provide comfort has become standard practice. It is important to inquire about the time and dose of the most recently administered antipyretic. Acetaminophen is the most common antipyretic used in the ED, and can be used concurrently with an NSAID such as ibuprofen. NSAIDs may result in greater gastrointestinal irritation and reduced renal blood flow, which may limit their use in some patients. Although extremely dangerous in the overdose setting, acetaminophen has a wide margin of safety in doses up to current recommendations (1 g every 4 hours, maximum 4 g/day). Hepatic metabolism may limit its use in liver failure. Ibuprofen is inexpensive and may be given in any combination not exceeding 2,400 mg/24 hours. Aspirin for febrile illness should be avoided secondary to its association with Reye syndrome in children. Selective COX-2 inhibitors have no role in the treatment of fever in the ED.

Intravenous fluids

IV fluid therapy should be initiated in patients with fever who appear ill or dehydrated. Peripheral vasodilation results in a relative decrease in central circulating volume. Fluid losses from vomiting and diarrhea are exacerbated by increased insensible losses from the skin and respiratory system. IV fluids provide a modest cooling effect, replace volume, and thus reduce tachycardia and thirst, improving overall comfort.

Cooling measures

Patients presenting with temperatures confirmed to be greater than 105°F (40.5°C) are more likely experiencing hyperpyrexia. In addition to standard resuscitative efforts, attempts should also be made to immediately lower body temperature. Excessive fluids should be avoided unless there is a clear history of fluid losses or oliguric renal failure. Rapid cooling can be achieved using cool sponge baths augmented by evaporative cooling from fans. Ice packs to the groin and axillae are also extremely effective. The use of alcohol applied to the skin as a cooling agent is discouraged. Invasive techniques instituting cool cavity lavage are last resorts, as these can be associated with serious morbidity.

Antimicrobial therapy

The initiation of antimicrobial therapy should occur after careful consideration and rapid collection of appropriate laboratory specimens. Delaying antibiotic therapy in order to obtain specimens for suspected sepsis or meningitis (e.g., CSF) is not appropriate. Timely administration of reasonably selected antibiotics based on the most likely pathogens while in the ED improves outcome and decreases hospital length of stay in serious bacterial illnesses, even if the selection does not prove perfect.

When the likelihood of a bacterial cause of fever is sufficiently great, or when the host is vulnerable to systemic illness, empiric antibiotics may be given prior to the identification of a specific source or organism. In cases of potentially life-threatening infections, broad-spectrum antibiotic combinations that cover Gram-negative, Gram-positive, and anaerobic organisms should be given until a specific organism is identified. In cases of localized infections, the spectrum of coverage should be narrowed to cover the organism(s) most likely responsible for infection, which helps reduce antibiotic resistance.

The exact antibiotic choice for infections is beyond the scope of this chapter. Most clinicians consult specialized handbooks, computer programs, or hospital antibiograms, which are frequently updated to include (local) pathogen resistance patterns. A general principle to consider when selecting an antibiotic is to confirm the absence of drug allergies. Patients typically do not distinguish adverse reactions (such as nausea) from true allergic reactions. The likely pathogens causing the infection should then be considered. Coverage may need to be broadened based on host defense deficiencies, special exposures, or indwelling devices or ports of entry. Drug–drug interactions need to be considered (e.g., avoid ciprofloxacin or trimethoprim-sulfamethoxazole if taking warfarin). The route of administration depends on the drug selected. Of note, more expensive IV medications do not necessarily offer special benefit over highly bioavailable oral preparations, except in the most severely ill patients, those with poor gut function (from passive congestion or hypoperfusion), or those unable to tolerate oral medication. The dosage of many antibiotics may need adjustment in elderly patients or those with liver and kidney dysfunction. All patients should be warned about medication side effects and potential complications of therapy, such as candidiasis, colitis, or allergic reaction. Some antibiotics render oral contraceptives less effective. Patients should be informed to complete the recommended course of antibiotics regardless of symptom improvement, unless they suspect a true or life-threatening allergy. Patients should discard any remaining pills after completing their prescribed course.

Special patients

Elderly

Elderly patients, especially those residing at skilled nursing facilities, represent groups with an increased risk of serious infection. Greater exposures to pathogens with antibiotic resistance and decreased immunological defenses make them more vulnerable to adverse outcomes. As many as one-third of elderly patients with systemic infection are unable to mount a fever. Often the reason for transfer from a nursing facility is nonspecific, such as weakness, confusion, unexplained falls, or persistent tachycardia. The presence of delirium superimposed on preexisting cognitive defects reduces the ability to rely on history. Greater reliance is therefore placed on a thorough physical examination and laboratories. The most common sources of serious infection include the urinary tract (approximately 50%), respiratory tract and skin. Non-infectious sources are also more common in elderly patients. Admission should be considered for any febrile elderly patient except for those not desiring inpatient management.

Immune compromised

Conditions that diminish a host's ability to fight infection frequently encountered in the ED include socially disadvantaged and substance-dependent patients. Crowded living conditions in shelters predispose patients to communicable diseases through contact (scabies) or droplet infection (tuberculosis). Poor nutrition and exposure to hostile weather conditions compound these risks. Alcoholics are especially vulnerable to pneumonia due to their increased incidence of vomiting and aspiration. Ascites in an individual with alcoholic liver disease should force EPs to consider spontaneous bacterial peritonitis as a cause of fever. IV drug users are at risk for local skin abscesses, cellulitis, endocarditis, osteomyelitis, and blood-borne viruses such as HIV and hepatitis. Diabetics are at risk for chronic fungal and foot infections secondary to impaired microcirculation, decreased local oxygen tension, and diminished wound healing. Sickle cell and asplenic patients are at particular risk for infection by encapsulated organisms, such as *Pneumococcus*. Any sickle cell patient presenting in crisis should have infectious precipitants ruled out.

HIV-related infections

HIV-related infections correlate with a patient's CD4 count. Primary HIV infection manifests as a flu-like illness typically following a 2–4 week incubation. An acute HIV infection should be considered in a patient with acute febrile illness and adenopathy, pharyngitis, or risk factors. Opportunistic infections or AIDS-defining illnesses do not present until the CD4 count falls below 400 cells/mm^3 and viral loads begin to rise. Once the CD4 count falls below 100 cells/mm^3, a variety of otherwise rare pathogens should be considered: *Pneumocystis (carinii) jiroveci* pneumonia (PCP), tuberculosis, cytomegalovirus (CMV), *Mycobacterium avium* complex, herpes simplex, esophageal candidiasis, toxoplasmosis and *Cryptococcus*. The incidence of neoplasms, such as Kaposi's sarcoma and lymphoma, is also high.

Fevers may occur from antibiotics used in prophylaxis or treatment (e.g., sulfonamides or dapsone). Antiretroviral therapy is associated with drug fever, myositis, pancreatitis and hepatitis. The work-up for patients with advanced HIV should include blood, urine, and possibly sputum and stool tests. A chest X-ray is indicated in essentially all HIV-positive individuals who present with cough and fever. A lactate dehydrogenase (LDH) level is often elevated in patients with PCP despite a normal chest X-ray. CT of the brain prior to LP in patients with AIDS is a prudent precaution, as mass lesions such as toxoplasmosis may be present.

Organ transplant

Likely causes of fever in organ transplant patients correlate with the time since transplantation. Fevers within 1 month are likely to be related to surgical wounds and occasionally to transmitted donor infections. Few fevers are secondary to opportunistic infections this early. At one to six months post-transplant, there is an increased incidence of viral infections (e.g., Epstein–Barr virus, CMV, hepatitis B and C, HIV) and opportunistic infections. The incidence of rejection and fever caused by antilymphocytic antibody treatment is also increased. Not until months or years after transplantation are the causes of fever similar to those in the population at large. Organ recipients have an increased risk of malignancy and opportunistic infection due to their immune-modulating therapy. Antibiotic therapy is complex and should involve close consultation with the transplant team.

Returned foreign traveler

The returned foreign traveler represents a particular challenge in the ED. In addition to the typical infections prevalent in the United States, infectious possibilities common in the region of travel must be considered. It is important to identify all foreign destinations and special exposures, such as travel to farms or jungles. Travelers should be encouraged to query their doctors for immunization recommendations and antibiotic prophylaxis prior to foreign travel.

The most common travel affliction is traveler's diarrhea. Other diseases presenting with fever to consider include cholera, typhoid, dengue and yellow fever, malaria, schistosomiasis and trypanosomiasis. The Centers for Disease Control and Prevention (CDC) maintains a comprehensive updated travel resource for tourists and physicians (http://www.cdc.gov/travel). Patients may require quarantine until the diagnosis can be made, although this is rare.

Neutropenia

Patients undergoing chemo- or radiation therapy often present to the ED with fever. Dramatic drops in neutrophil counts occur 1–2 weeks following cytotoxic therapy. Despite the near obliteration of cells that produce endogenous pyrogens (agranulocytosis), the ability to mount a fever is remarkably preserved. Neutropenia is defined as a blood neutrophil count of <500 cells/mm^3. The incidence of bacteremia climbs sharply as neutrophil counts fall below 100 cells/mm^3. Febrile patients who may be neutropenic should be isolated, with blood samples for CBC and culture quickly drawn prior to the administration of broad-spectrum antibiotics (ceftazidime or imipenem). Vancomycin may be added for patients in whom indwelling catheters are the suspected source of infection. Only the most well-appearing, reliable and compliant neutropenic patients should be considered for discharge, and only following discussion with that patient's oncologist or a specialty consultant on call.

Institutionalized

Patients who have been exposed to nosocomial pathogens are at an increased risk for colonization with methicillin-resistant *Staphylococcus aureus* (MRSA) or vancomycin-resistant *Enterococcus* (VRE). Although no more virulent than their susceptible counterparts, infections with these organisms are difficult to eradicate and can produce symptomatic infection. Newer strains of fluoroquinolone-resistant enteric bacteria and vancomycin-resistant *Staphylococcus* pose serious public health risk. It is important that health care providers exercise strict contact precautions. Limiting the inappropriate use of broad-spectrum antibiotics is likely to slow the spread of drug resistance.

Spinal cord injury

The incidence of fever in spinal cord-injured patients is quite high. Decreased mobility and loss of reflexes distal to the lesion predisposes individuals to UTIs, pneumonia, and infected decubiti. Nearly all patients who do not void spontaneously become colonized with bacteria from prolonged urine dwell times and self-catheterization. Loss of function above T6 disrupts thermoregulatory neural circuits, impairing shivering and sweating responses. Patients may suffer from autonomic hyperreflexia, with intermittent excessive sweating contributing to thermal instability. Despite being insensate in infected areas, spinal cord-injured patients often describe spreading infection as malaise.

Postsurgical/postpartum

Patients who have recently undergone surgical procedures or childbirth are at risk for the development of fever. The mnemonic 5 Ws (wind, water, wound, womb, wonder drug) is a helpful reminder for the most common sources of fever. The earliest and most common cause of postoperative fever is atelectasis, often occurring within 1–2 days. Bacterial counts at seeded sites may rise to levels that cause symptoms by postoperative day 3. Cesarean section increases the risk of endometritis compared with vaginal delivery. It is important to look closely at all surgical wounds, including episiotomies, for signs of infection.

Disposition

Fever itself is not considered a reason for hospital admission. Healthy patients with acute viral illnesses who respond to antipyretics, antiemetics, IV hydration and other medication adjuncts do not require hospitalization. Healthy patients with localized bacterial infections who can tolerate oral therapy can also be safely discharged. Appropriate discharge instructions must be provided and close follow-up with outpatient clinicians arranged in the event localized complications develop or deterioration limits a patient's ability to care for himself or herself. Specific criteria explaining reasons to return to the ED are crucial. Patients with significant cardiopulmonary comorbidity, alcoholics, the elderly or homeless, and those with compromised immune systems (including diabetes) may require admission for infectious processes that could be managed as an outpatient in healthy individuals.

The following indications for admission may serve as a helpful guide; however, each case depends on patient preference, social support and outpatient provider availability. Admission should be considered for any patient with:

1. A non-viral systemic infection (although certain virulent viral infections or viral infections that cause deterioration of the cardiopulmonary system warrant admission)
2. Serious deep local or regional infections requiring IV antibiotics
3. Infections that require surgical intervention beyond simple incision and drainage
4. Any infection resulting in alterations of behavior or consciousness

Individuals with limited physiologic reserve and those incapacitated by fever are likely to require admission, even if the source is unknown.

It is the EP's responsibility to ensure that time-specific return criteria are clear and understood. Most infections treated with antibiotics should improve within 72 hours. Therefore, most adult patients with fever should be rechecked in 48–72 hours. Those who are not able to follow through on a reasonable outpatient plan should be considered for admission.

It is important to be aware of national guidelines for the treatment of many serious infections. Severity rating scales, treatment suggestions and admission criteria are often provided based on clinical research, consensus guidelines and expert opinion. Infectious disease consultation is rarely necessary in the ED acutely, but is an option for a difficult case or a challenging disposition. Many hospitals have implemented policies requiring infectious disease specialist approval before prescribing certain antibiotics in an attempt to minimize pathogen resistance.

Pearls, pitfalls and myths

- Fever is a nonspecific symptom with a broad differential including both benign and serious illness. The diagnostic work-up and treatment depend on host factors and specific causes.
- General resuscitative principles take priority over the treatment of fever or the identification of a specific causative agent.
- Fever must be quickly distinguished from hyperpyrexia. Improper treatment of either may lead to serious morbidity and mortality.
- Universal precautions are protective for patients and health care personnel. Care must be taken to safely dispose of all potentially infective materials in the patient's room.
- The etiology of a fever can often be made from the history and physical examination alone. Special historical questions regarding personal, sexual, occupation, travel, pets and dietary habits must be asked.
- Infrared tympanic thermometers are inadequate to assess for fever. A low threshold should exist to recheck the temperature in the appropriate setting by other means, and repeat this as necessary.
- The diagnostic framework for determining the cause of a systemic physiologic abnormality such as fever is best done using an organ system rather than anatomic region approach.
- Blood cultures should be obtained prior to antibiotic treatment if the clinical situation permits. However, antibiotic treatment should never be delayed if sample collection proves difficult.
- Antibiotics are less effective at preventing infection than treating them. Given our current drug-resistance crisis, every effort should be made to limit antibiotic use to those patients with suspected serious or documented bacterial illness, and to approach antibiotic selection with likely or specific pathogens in mind.

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28 Fever in children

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Scope of the problem

Pediatric fever is one of the more common presenting complaints to the emergency department (ED). The objective of ED evaluation of febrile children is to identify and treat the small subset of children who harbor life-threatening bacterial infections. A febrile infant is at risk for a variety of serious bacterial infections (SBI), including bacteremia, meningitis, osteomyelitis, suppurative arthritis, skin and soft tissue infection, urinary tract infection, gastroenteritis and pneumonia. A concurrent goal is to avoid the indiscriminate use of antibiotics in febrile children. The etiology of a child's fever in the majority of cases is an acute viral infection. Unfortunately, the clinical appearance of a child with *occult bacteremia* (presence of pathogenic bacteria in the blood of a well-appearing febrile child without an identifiable focus of infection) may resemble that of a child with fever due to viral illness. As a result, the broad spectrum of management options for febrile children continues to be the subject of much research and controversy.

Anatomic essentials

Fever results from body temperature elevation above normal circadian variation due to an increase in the hypothalamic thermoregulatory set point. A febrile response is thought to result from enhanced metabolic activity and is mediated by the release of pyrogens. These pyrogens (e.g., tumor necrosis factor, interleukin-1 and interferon) are released from host leukocytes, which in turn reset the temperature regulatory center in the hypothalamus. In neonates, fever response pathways are not well-developed; consequently, fever is not a uniformly sensitive marker for acute infections. As a result, hypothermic or normothermic children with altered behavior (e.g., poor feeding and weak cry), especially neonates, warrant careful evaluation for acute infections.

A child's immune system matures with age. A neonate relies primarily on passive transfer of protective maternal immunoglobulins (IgG) to ward off infections. A child's immune system becomes more adept at responding to bacterial and viral pathogens over time. Widespread immunization of children has significantly reduced the morbidity and mortality caused by varicella, *Haemophilus influenzae* type B, and the poliovirus infections, to name a few. Young infants are unable to mount sustained immunologic responses to certain vaccines (e.g., pertussis vaccine). This delay in immune response renders young infants susceptible to these infections.

Elevated body temperatures are often caused by infections, but also result from excessive physiologic stress (e.g., hyperthyroid state), central nervous system (CNS) lesions, inflammation, malignancy, or exposure to chemicals (e.g., drugs). Fever should be distinguished from hyperthermia. *Hyperthermia* is defined as an elevation in body temperature that is not associated with an elevation of the thermoregulatory set point. Hyperthermia is found in conditions characterized by inadequate heat dissipation from the body (e.g., environmental exposure, neuroleptic malignant syndrome, anticholinergic or sympathomimetic toxidromes).

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 28.1).

History

How high was the temperature and how was it measured?

A temperature of ≥38.0°C (100.4°F) is considered the threshold for defining a fever. A rectal temperature is the most accurate method of assessing the core body temperature in the ED. A fever at home should be treated the same as an elevated temperature in the ED. A positive correlation between absolute height of fever and the risk for bacteremia has been established. Although an elevated temperature itself is not believed harmful, it does impose metabolic demands on the body and predisposes certain children to complications. A small subset of children between the ages of 6 months and 5 years will develop febrile seizures as their body temperature rises. Children with epilepsy are at increased risk for seizures as a result of febrile illnesses.

How ill do the parents perceive their child to be?

The primary factor dictating the extent of a febrile child's ED evaluation is the overall appearance in an otherwise healthy child. A severely ill-appearing child should undergo an extensive evaluation and receive empiric antibiotic therapy. A happy, playful child in no visible distress is an ideal candidate for a less aggressive evaluation. Carefully listening to an experienced parent's assessment of how ill their child appears is very important. Parents may be more discerning with regard to their child's

Table 28.1 Fever in children red flags

History	Concerning diagnosis
Tactile or documented fever, reported chills or rigors	Febrile illness, including viral etiology, UTI, pneumonia, sepsis, SBI
Tachypnea with normal lung auscultation	SBI, pneumonia, DKA
Abdominal pain	Appendicitis, pneumonia, DKA, pharyngitis
Headache, stiff neck, confusion, weakness	Meningitis, encephalitis, brain abscess or CNS infection
Refusal to walk, antalgic gait	Osteomyelitis, septic joint (hip, knee)
Warmth, pain with ROM, decreased joint mobility	Septic arthritis
Lethargy, irritability or altered sensorium	SBI, CNS infection (meningitis, encephalitis), intussusception
Drooling, limited neck movement, difficulty swallowing, voice change	Epiglottitis, retropharyngeal abscess
Recent surgery	Postoperative infection, cellulitis, phlebitis, pneumonia, UTI
Recent chemotherapy or other treatment for malignancy	Cancer-related fever, tumor fever, neutropenic fever, tumor lysis syndrome, opportunistic infection
Examination finding	**Concerning diagnosis**
Rales, rhonchi, asymmetry of lung auscultation	Pneumonia, bronchiolitis, foreign body aspiration in very young children to toddlers
Flank tenderness (costovertebral angle)	Pyelonephritis
Petechial or purpuric rash	Meningococcemia, purpura fulminans, RMSF, endocarditis
Kernig's or Brudzinski's sign, nuchal rigidity	Meningitis, encephalitis
Cardiac murmur	Endocarditis
Back or vertebral (spinal) tenderness	Spinal epidural abscess, osteomyelitis
Abdominal tenderness	Appendicitis
Strawberry tongue, injected conjunctiva	Kawasaki disease
Persistent tachycardia after hydration and defervescence	Occult bacteremia or SBI
Negative examination	Occult bacteremia or SBI

CNS: central nervous system; DKA: diabetic ketoacidosis; RMSF: Rocky Mountain spotted fever; ROM: range of motion; SBI: serious bacterial infection; UTI: urinary tract infection.

illness than health care workers practicing in a frenetic ED environment. Difficulties with feeding or anorexia are important clinical indicators of SBI and warrant careful evaluation.

Administration of antipyretics to children presenting to the ED with a fever often results in a rapid improvement in their appearance and behavior. Febrile children tend to be more irritable and ill-appearing, often leading to more extensive ED evaluations. Visual and behavioral clues to illness severity are difficult to ascertain in younger patients. Studies utilizing experienced clinicians have demonstrated that in infants less than 2 months of age, clinical appearance alone is an insensitive indicator of illness severity. This is the guiding principle for the more conservative approach to the evaluation of fever in younger patients.

How long has the child been sick or had a fever?

In order to correctly establish a febrile child's course of illness, careful attention must be paid to the chronology of symptoms. For example, a child with a febrile illness for several days who appears to be improving is less likely to harbor an SBI than a listless and ill-appearing child with fever for several hours.

What are the child's associated symptoms?

Questions regarding the presence of associated symptoms can help identify the source of a child's fever. For example, a preceding history of dysuria and increased frequency of urination are important clues suggesting the diagnosis of pyelonephritis. Other symptom constellations may suggest a viral etiology, such as rhinorrhea, sneezing and cough for viral upper respiratory infection, or vomiting accompanied by diarrhea for acute viral gastroenteritis. Infant teething should not be considered as a source of fever.

The child's hydration status should be determined by inquiring about urine output and oral intake. The current number of wet diapers and the amount of oral fluids should be considered relative to when the child was previously well.

Was there exposure to ill contacts?

Sick household contacts and daycare classmates can be important reservoirs of infection. Inquiries into the specific type of infections (e.g., tuberculosis) that family members are suffering from often provide valuable clues to the etiology of febrile illnesses.

What is the child's medical history?

Prior history of pneumonia or urinary tract infection suggests recurrence as the possible source of fever. Risk factors for SBI in neonates include issues related to birth history, such as maternal fever, prolonged rupture of membranes, premature birth and low birth weight. Comorbid conditions (e.g., congenital heart disease) or immunodeficient states (e.g., organ transplant recipients, cancer patients receiving chemotherapy, sickle cell patients with functional asplenia) are risk factors for SBI at any age. Prior illnesses or comorbid conditions often place children at higher risk for complications from SBI. For example, a child with cyanotic congenital heart disease is at greater risk from an acute pulmonary infection than an otherwise healthy child with a similar illness. Inquiring about a child's immunization status is very important, as a child with an incomplete immunization record (particularly pneumococcus and *Haemophilus influenzae* type B) is at risk for a wider variety of illnesses and may benefit from more conservative care.

Has there been any travel history? Has there been any exposure to animals?

Travel outside the United States may expose children to a variety of infectious diseases (e.g., malaria, typhoid). Noting the geographic areas a patient has traveled or lived within the United States may provide useful information (e.g., Rocky Mountain spotted fever associated with the South Atlantic). Inquiring about exposure to animals, as well as tick and insect bites, may provide important clues to the etiology of infectious illnesses (e.g., cat-scratch disease).

What medications has the child received recently?

It is important to determine the last time the child received antibiotics, what type was given, and for what condition. In the case of recent and recurrent infection, the antibiotic choice may need to be modified to one that has a broader spectrum of coverage to help ensure susceptibility. If the child has become progressively ill despite current antibiotic therapy, the possibility of a partially-treated meningitis must be entertained.

The amount, type, frequency and time of the last dose of antipyretic given prior to ED presentation provide insight into the medication's impact on the fever. This information also presents an opportunity for parental education in cases of inadequate dosing or prolonged dosing intervals that may contribute to the persistence of fever in their child.

Physical examination

General appearance

Observation of a child's behavior while acquiring the history of present illness provides the initial impression of illness severity. Attempts to define clinical features as indicators of SBI have been made (e.g., Yale Observation Scale, which includes an objective assessment of a child's alertness, playfulness, interaction with the environment, color, state of hydration, quality of cry and ability to be consoled). Unfortunately, neither physical examination nor these clinical tools have been shown to be either sensitive or specific enough to identify SBI in children less than 3 months of age. An assessment of a patient's mental status, activity, temperament and interaction with their environment are the initial steps in evaluating febrile children.

The assessment of a child's responsiveness should include observation of the child's interaction with the practitioner, their spontaneous visual or physical exploration of the environment, reaching for and playing with age-appropriate toys, consolability with parents, and an observation of feeding behavior in infants. Toxic children may cry excessively, be irritable, or be lethargic and difficult to arouse. They may demonstrate a lack of interest in their environment or in feeding, and cannot be consoled by their parents. They may appear mottled or pale with tachypnea and grunting respirations, and may be listless with decreased tone and response to external stimuli.

Vital signs

Begin with a review of triage vital signs (Table 28.2). The temperature should be obtained rectally in all children less than 90 days of age, in those with tachypnea, and in all children too young to cooperate adequately with an oral temperature. The height of the fever should be noted, as it has prognostic value. Children with higher fevers have an increased incidence of SBI, although the majority of these patients have viral infections. Elevated heart and respiratory rates may be the direct result of a fever. Greenes demonstrated a linear relationship between heart rate and temperature; for every 1°C increase in temperature, the heart rate can be expected to increase approximately 10 beats per minute. Tachycardia out of proportion to the degree of fever occurs with dehydration, sepsis and cardiac conditions. Persistent tachycardia or tachypnea despite defervescence may be an indication of SBI. Blood pressure is an insensitive indicator of illness severity, as children often maintain normal blood pressures until advanced stages of illness. Pulse oximetry values less than 95% on room air suggest compromised respiratory function and warrant further evaluation.

Head

The anterior fontanelle should be assessed for bulging that occurs in the presence of elevated intracranial pressure. A bulging fontanelle in a toxic-appearing infant suggests meningitis. The eyes are evaluated for conjunctival injection and discharge, and the nasopharynx and oropharynx for erythema, enanthems, or exudates. Injection of the conjunctiva may be seen with viral illness, conjunctivitis, or Kawasaki disease (Figure 28.1). A careful evaluation of the oropharynx may yield important clues to the etiology of an infant's high fever and anorexia (e.g., mucosal

Table 28.2 Vital signs in children by age

Age	HR (beats/min)	SBP (mmHg)	RR (breaths/min)	Weight (kg)
Preterm	120–180 (140)	40–60 (50)	55–65	2
Term newborn	90–170 (125)	52–92 (72)	40–60	3
1 month	110–180 (120)	60–104 (82)	30–50	4
6 months	110–180 (130)	65–125 (94)	25–40	7
1 year	80–160 (125)	70–118 (94)	20–40	10
2 years	80–130 (110)	73–117 (95)	20–30	12
4 years	80–120 (105)	65–117 (91)	20–30	16
6 years	75–115 (100)	76–116 (96)	18–24	20
8 years	70–110 (90)	79–119 (99)	18–22	25
10 years	70–110 (80)	82–122 (102)	16–20	30
12 years	60–110 (75)	84–128 (106)	16–20	40
14 years	60–105 (75)	84–136 (110)	16–20	50

HR: heart rate; RR: respiratory rate; SBP: systolic blood pressure.

Adapted from Barkin RM (ed). *Pediatric Emergency Medicine: Concepts and Clinical Practice*, 2nd ed. Mosby, St. Louis, MO, 1997.

vesicles suggestive of Coxsackie virus infection, or a strawberry tongue associated with Kawasaki disease [Figure 28.2] or late-stage scarlet fever). Exudative tonsillitis in a child less than 2 years of age is most often viral in origin. The definitive diagnosis of acute otitis media is more challenging in the presence of crying or fever, which may both cause the tympanic membrane to appear hyperemic. Acute otitis media is typically associated with a middle ear effusion and a hyperemic tympanic membrane with altered landmarks. Decreased movement of the tympanic membrane with insufflation is widely cited as the most accurate method to assess for a middle ear effusion.

Neck

The neck is examined for localized masses (i.e., lymphadenopathy and abscesses) and for passive range of motion. In the correct clinical context, nuchal rigidity (resistance to flexion and extension of the neck) suggests meningeal inflammation (i.e., meningitis). This sign is of limited value in young children and infants, as they often fail to develop nuchal rigidity despite having meningitis. In older children, nuchal rigidity may be identified by eliciting pain or spasm with knee extension with the knee and hip flexed at 90 degrees (positive Kernig’s sign), and hip flexion that occurs following passive neck flexion (positive Brudzinski’s sign), although these signs are not reliable.

Lungs

Pulmonary examination begins with an assessment of work of breathing (i.e., respiratory rate, presence of retractions, nasal flaring, accessory muscle use). Tachypnea is a valuable clue to serious pulmonary infection. Young children may harbor significant pulmonary infections yet have minimal auscultatory findings. As a result, egophony, crackles, or wheezes may not be appreciated in pneumonia. The finding of diffuse symmetric crackles often signifies acute bronchiolitis. Beta-agonist therapy (e.g., albuterol) will diminish auscultatory crackles in a subset of children with acute bronchiolitis. Stridor most

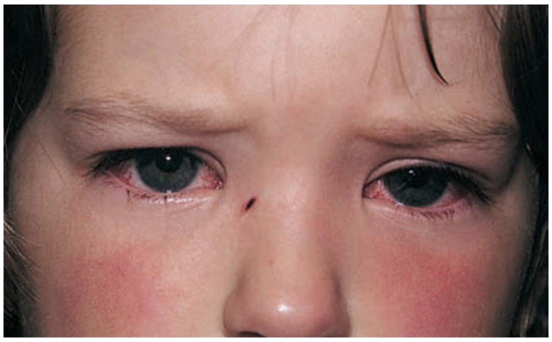

Figure 28.1
Injected conjunctiva of Kawasaki disease. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

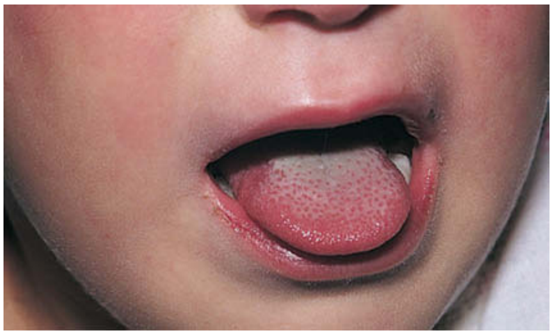

Figure 28.2
Strawberry tongue of Kawasaki disease. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

commonly occurs with upper airway inflammation, as seen with laryngotracheobronchitis (croup), but can also be seen with epiglottitis or retropharyngeal space abscesses.

Heart

Cardiac evaluation involves assessing heart rate, heart sounds, murmurs, and listening for additional findings (e.g., pericardial friction rub). Many children will have accentuation of innocent heart murmurs (e.g., pulmonary flow murmur and Still's murmur) during febrile illnesses. Any grade III or higher murmur, diastolic murmur, or friction rub should be deemed pathologic, warranting further evaluation. Tachycardia may persist despite defervescence and rehydration. SBI should be considered in children with fever and murmurs, especially new murmurs.

Abdomen

The examination of the abdomen is essential to the evaluation of febrile children with concurrent gastrointestinal complaints. Hypoactive bowel sounds indicate decreased intestinal motility, and the absence of bowel sounds is a cause for concern. Any focal tenderness, rebound, or guarding suggests of a potential surgical abdomen (e.g., appendicitis). Hepatosplenomegaly is associated with a variety of diseases.

A majority of cases of acute appendicitis are misdiagnosed in infants and young children. The accurate diagnosis of appendicitis in young children is challenging because these patients have limited communication skills, are difficult to examine, and typically have benign gastrointestinal illnesses (e.g., viral gastroenteritis) as the etiology of their fever. Providers should repeat their examination of the abdomen to ensure it is benign in any febrile child with gastrointestinal complaints. In addition, fever and protracted gastrointestinal symptoms (e.g., anorexia and diarrhea) may be the presentation of a perforated appendix with abscess formation.

Extremities

The extremities should be examined for color, capillary refill and pulse strength. Cool extremities with poor capillary refill and weak pulses are suggestive of diminished peripheral perfusion, sepsis syndrome, or shock.

All joints should be examined for the presence of erythema, edema, warmth, or tenderness. Range of motion also should be assessed. Septic arthritis is a noted source of infection and fever in infants.

Skin

The entire body of a febrile child should be inspected for the presence of a rash. The dermatologic manifestations of infectious diseases are protean. Nonetheless, familiarity with specific exanthems can provide helpful clues in evaluating febrile patients. The most notable example would be the classic cutaneous manifestations of *Neisseria meningitides* infections (Figure 28.3), including the presence of petechiae and purpura. It is important to distinguish diffuse petechiae from those lesions that occur above the nipple line associated with vigorous coughing or crying, as well as those on the upper extremities following tourniquet placement. Other classic descriptions of rashes include "dewdrop on a rose petal" for the lesions of varicella, and the "slapped-cheek" rash of erythema infectiosum (Figure 28.4).

Lymphatic system

Benign lymph nodes commonly palpated in healthy children are typically ≤1 cm in size, painless, mobile, and devoid of any warmth or induration. Any enlarged, warm, indurated or fixed nodes warrant further evaluation.

Anogenital

The circumcision status of a male child with a fever should be noted, as the presence of foreskin correlates with an increased risk for urinary tract infection. The scrotum and testes should be examined to exclude epididymo-orchitis or testicular torsion as a source of fever. The anorectal region should be evaluated to exclude signs of infection

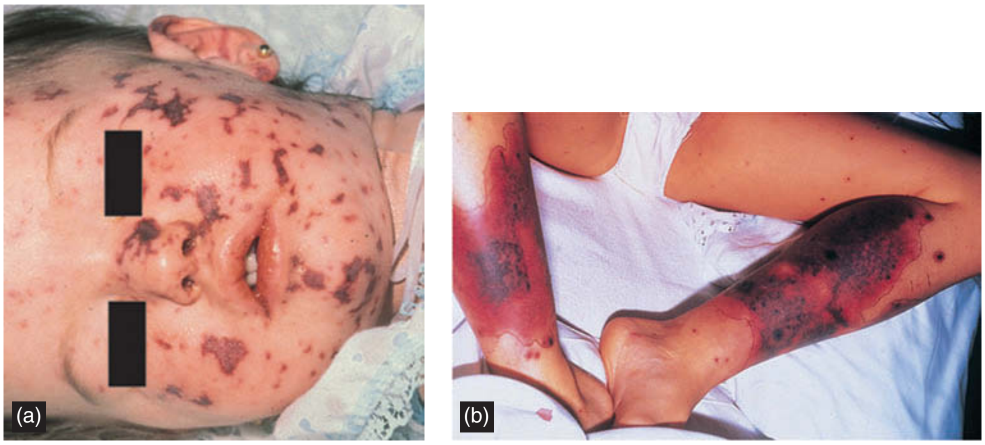

Figure 28.3
Meningococcemia. (a) This child manifests the purpuric and petechial rash characteristic of acute meningococcemia; (b) purpura may progress to form areas of frank cutaneous necrosis, especially in patients with disseminated intravascular coagulation. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

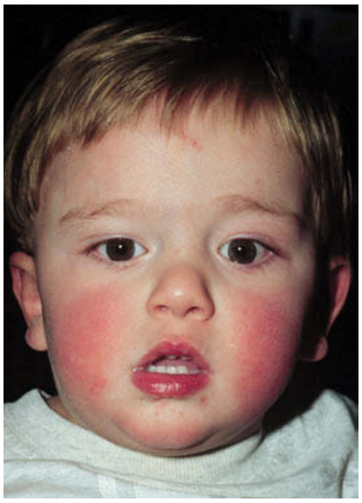

Figure 28.4
Slapped-cheek appearance of erythema infectiosum. Courtesy: Lawrence Stack, MD.

(e.g., perirectal abscess). Diaper dermatitis is a common finding in infants. This is differentiated from more serious exanthems by a lack of associated systemic findings, minimal tenderness and characteristic distribution.

Neurologic

An age-appropriate neurologic examination may provide other clues to the source of the fever. A diminished level of consciousness, lethargy and irritability are worrisome for SBI, as well as other serious conditions such as intussusception (Table 28.3). A reluctance to ambulate or an antalgic gait in an older child with fever may suggest septic arthritis or osteomyelitis as the source.

Table 28.3 Causes of fever and altered sensorium

Bacterial sepsis, other than meningitis
Febrile seizure
Hypoglycemia, secondary to poor oral intake plus vomiting and diarrhea
Hyponatremic dehydration
Intussusception
Meningitis or encephalitis
Shigella gastroenteritis
Toxic ingestion
Unsuspected head trauma, including shaken-baby syndrome with central nervous system bleeding

Adapted from Marx JA (ed). *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 7th ed. Mosby, St. Louis, MO, 2010.

Differential diagnosis

Table 28.4 describes the symptoms, signs and diagnostic tests associated with common diagnoses occurring in febrile children.

Table 28.4 Differential diagnosis of fever in children

Diagnosis	Symptoms	Signs	Diagnostic testing
Acute infectious laryngotracheobronchitis (croup)	Barking cough, hoarse voice	Stridor, harsh barking cough, occasional coarse crackles, rhonchi	Clinical diagnosis. Plain radiographs of neck reveal characteristic findings (dilated hypopharynx on lateral view and steeple sign on PA view).
Acute otitis media	Ear pain, crying, hearing loss (typically preceded by an upper respiratory infection)	Bulging tympanic membrane, abnormal tympanic landmarks, middle ear effusion	Clinical diagnosis. Ear insufflation used as a screening tool for detecting middle ear effusion.
Acute suppurative adenitis	Painful mass, swelling, redness	Tenderness, erythema, warmth, firm or fluctuant mass	Clinical diagnosis. Further diagnostic testing may be indicated (e.g., *Mycobacterium tuberculosis* testing).
Appendicitis	Abdominal pain, anorexia, vomiting, diarrhea	Abdominal tenderness, rebound, guarding	Clinical diagnosis. Abdominal ultrasound or CT may assist in equivocal cases.
Bronchiolitis	Cough, rhinorrhea, wheezing, increased respiratory rate	Tachypnea, crackles, wheezing	Clinical diagnosis. Chest radiograph will demonstrate characteristic symmetric bilateral perihilar infiltrates. RSV nasal swabs may confirm etiology.
Cellulitis	Pain, warmth and redness of the skin	Tenderness, erythema, warmth and edema of skin; lymphangitic streaking in advancing infection	Clinical diagnosis. When associated with abscess formation, wound cultures can be performed.
Encephalitis	Altered behavior, headache, seizures	Altered mental status, focal neurologic deficits and papilledema	LP. The initial analysis is same as for meningitis; additional tests often indicated (e.g., herpes virus testing).

(*continued*)

Table 28.4 Differential diagnosis of fever in children (*cont.*)

Diagnosis	Symptoms	Signs	Diagnostic testing
Epiglottitis	Sore throat, voice change, drooling	Drooling, stridor, dysphonia, tripod posture, toxic appearance	Clinical diagnosis. Plain lateral radiograph often shows characteristic "thumbprint" sign. Definitive diagnosis made by direct laryngoscopy (by a skilled endoscopist in a controlled setting).
Intussusception	Colicky abdominal pain, episodic inconsolability, bilious vomiting follows, 10% have "currant jelly" stool (late finding)	Episodes of lethargy or irritability alternating with normal behavior, soft abdomen between episodes, eventually tender or distended abdomen, 85% mass in right lower quadrant (RLQ) or upper abdomen, heme-positive stools; may be altered	Abdominal X-ray may show dilated loops and no air distal to the obstruction. Ultrasound is operator-dependent, but can have high diagnostic accuracy. Barium or fluoroscopic pneumatic (air) enema diagnostic and therapeutic.
Meningitis	Headache, stiff neck, nausea or vomiting, photophobia, altered behavior	Photophobia, nuchal rigidity, altered mental status	LP. Initial analysis includes cell count, Gram stain, glucose, protein and bacterial culture.
Occult bacteremia	Fever without a source of symptoms	Range from well-appearing to lethargic and ill-appearing	CBC, blood cultures, UA, urine cultures.
Orbital or periorbital cellulitis	Redness, pain, swelling around eye	Pain, erythema, edema of periorbital region. Systemic symptoms (e.g., fever) and limited extraocular eye movement define orbital cellulitis.	Clinical diagnosis. Facial CT will assist in differentiating periorbital from orbital cellulitis in equivocal cases.
Osteomyelitis	Pain, redness over area, warmth, swelling	Point tenderness, decreased mobility, swelling, warmth and erythema	Clinical diagnosis. CBC, ESR, blood cultures, X-rays may be positive if more than 10–20 days of symptoms. CT may show soft tissue changes and swelling at 3 days. MRI is very sensitive after 24–36 hours.
Pharyngitis, tonsillitis or peritonsillar abscess	Sore throat, anorexia	Erythema, exudates, ulcerations, vesicles, cervical adenopathy. Displaced uvula and bulge in peritonsillar region are signs of a peritonsillar abscess.	Clinical diagnosis. Organism identification via throat (or wound) cultures or assay.
Pneumonia	Cough, chest pain, shortness of breath, rapid breathing	Hypoxia, tachypnea, respiratory distress and adventitious breath sounds	Chest X-ray typically reveals an infiltrate.
Pyelonephritis	Nonspecific presentation in young children. Flank pain, abdominal pain and nausea typical in older children.	Flank tenderness. Physical findings outside of fever may be minimal.	Clinical diagnosis. UA is suggestive of and urine culture confirms the diagnosis.
Retropharyngeal abscess	Severe throat pain, stiff neck, loss of appetite	Torticollis, drooling, stridor, bulge in retropharynx	Plain lateral radiograph of neck; CT is more accurate.
Septic arthritis	Painful, warm, swollen joint; if involving the lower extremity, limp	Exquisite tenderness to range of motion, decreased mobility, erythema, warmth and effusion may be present.	CBC, ESR, glucose and blood cultures; arthrocentesis. Joint fluid is sent in heparinized tube for Gram stain, cell count, glucose and culture.
Upper respiratory tract infection	Rhinorrhea, sneezing, sore throat, cough, low-grade headache	Rhinorrhea, pharyngeal erythema, rhonchi	Clinical diagnosis

CBC: complete blood count; CT: computed tomography; ESR: erythrocyte sedimentation rate; LP: lumbar puncture; MRI: magnetic resonance imaging; PA: posteroanterior; RSV: respiratory syncytial virus; UA: urinalysis.

Diagnostic testing

Diagnostic testing of febrile children varies greatly between providers. Febrile child management algorithms incorporate patient age, comorbidities, general appearance, vital signs, and results of ancillary testing (e.g., white blood cell [WBC] count). Some clinicians rely on prior clinical experience, whereas others utilize evidence-based algorithms to dictate their diagnostic evaluations. Laboratory studies may be ordered depending on the clinical scenario. A brief summary of significant abnormalities is provided, as well as potential pitfalls in the interpretation of each test.

Laboratory studies

Complete blood count

Leukocytosis, a left shift and bandemia are suggestive, but neither sensitive nor specific, for the presence of an SBI. Leukopenia (WBC count <5,000) may be a sign of SBI or early sepsis, as well as secondary to viral suppression. Thrombocytopenia may result from advanced disseminated intravascular coagulation associated with sepsis.

Urinalysis

Health care providers should generally obtain urine by a catheterized specimen in children who are not toilet-trained, to decrease the likelihood of contamination from a bag-collected specimen. An elevated specific gravity is indicative of dehydration. The presence of more than 5 WBCs/high-power field (hpf) as well as a positive nitrite test suggest a bacterial infection. It is important to note that the urine may be normal in some children with urinary tract infections, particularly if obtained late in the day. If clinical suspicion is high, a urine culture should be sent, even if the urinalysis (UA) is negative. A Gram stain positive for the presence of bacteria confirms the diagnosis of a urinary tract infection.

Urine culture

A urine culture should be ordered on nearly all febrile infants when the urine specimen is obtained by catheter, and in selected infants when the urine was collected by clean catch. Although results are typically not available for 24–48 hours, the urine culture is helpful for identifying the infecting organism and its sensitivity to commonly prescribed antibiotics.

Blood culture

The delay in obtaining the results of cultures renders them unhelpful in the initial ED management of the febrile child. Nevertheless, blood cultures are invaluable in the evaluation of the febrile child without a source.

Cerebrospinal fluid

Following lumbar puncture (LP), the cerebrospinal fluid (CSF) is routinely sent for glucose, protein, cell count (including differential), Gram stain and culture. In cases of bacterial meningitis, the CSF may reveal an elevated WBC count (>1,000 WBCs/hpf), a decreased glucose concentration (compared with the peripheral blood values), and an elevated protein. An elevated WBC count in an atraumatic LP needs to be evaluated for a leukocyte predominance, suggestive of a bacterial infection, as compared with a monocytic proliferation, which is more likely due to a viral infection of the meninges. Traumatic taps with elevated red blood cell (RBC) counts make the interpretation of these values more problematic. A negative Gram stain does not conclusively rule out the possibility of bacterial meningitis; if other clinical data suggest a bacterial infection, the patient should be presumed to have bacterial meningitis and treated as such. A CSF culture requires 24–72 hours to be conclusive.

Erythrocyte sedimentation rate/C-reactive protein

The erythrocyte sedimentation rate (ESR) and the C-reactive protein (CRP) level are two nonspecific markers of inflammation in the body that tend to be elevated in the presence of SBI. These tests are typically sent when the diagnoses of osteomyelitis, septic arthritis and Kawasaki disease are being entertained.

Stool studies

The stool of a febrile child with a protracted course of diarrhea or bloody diarrhea can be sent for a number of studies: fecal leukocytes/hpf, bacterial culture, ova and parasites, and *Clostridium difficile* toxin (in patients who have received an extended course of antibiotics preceding their diarrhea). A stool smear with a WBC count more than 5/hpf suggests a bacterial etiology of the diarrhea. Bacterial culture results may later define the offending pathogen.

Radiologic studies

Chest radiographs are indicated in any febrile child who presents with signs of respiratory distress. These include the presence of tachypnea, dyspnea, hypoxemia (pulse oximetry on room air below 95%), or abnormal breath sounds. Because young children may harbor significant pulmonary infections with minimal auscultatory findings, a low threshold for imaging is indicated.

Management algorithms

Baraff, et al. (1993) published the most widely publicized study regarding the management of febrile children. They performed a meta-analysis of studies evaluating febrile children, and developed recommendations for the management of children 0–36 months of age with fever without a source. Fever without a source is defined as an acute febrile illness in which the etiology of the fever is not apparent after a careful history and physical examination.

A summary of their recommendations:

Age less than 28 days

A CBC, catheterized urine specimen for UA, urine culture, blood culture and CSF for cell count, glucose, protein, Gram stain and culture should be obtained on all febrile patients less than 28 days of age.

Age 28 days to 3 months

Febrile infants in this age group may be divided into low and high risk for invasive bacterial disease. The low-risk criteria include being previously healthy, having no focal bacterial infection on physical examination, and a negative laboratory screening. A negative laboratory screening examination includes a WBC count between 5,000 and 15,000/mm^3, a neutrophil band count below 1,500/mm^3, UA with less than 10 WBCs/hpf and no bacteria on urine Gram stain, and CSF with less than 8 WBCs/hpf and no bacteria seen on Gram stain. Infants meeting these criteria had only a 1.4% chance of having an SBI.

Studies evaluating the risk of SBI in children 60 days of age or less have demonstrated that identifying respiratory syncytial virus (RSV) infection by radioimmunoassay or influenza by rapid bedside testing is associated with an overall decrease in the risk of SBI. When incorporated into evaluation algorithms, this may further reduce laboratory testing in this age group.

Age 3 months to 36 months

The major concern in these patients is the presence of occult bacteremia. Although there is no single laboratory test that can immediately exclude this possibility, the height of the WBC count is of some value as a screening test. Performing blood cultures is recommended if the WBC count is more than 15,000/mm^3. For febrile patients without a source and a temperature of ≥39°C, a catheterized urine specimen should be obtained in all male infants up to 6 months of age if circumcised, otherwise until 1 year of age, and in all female patients 24 months or less. A urine culture should also be sent. A stool culture should be sent in the presence of diarrhea with gross blood. Children in this age group with fever <39°C without a source need no laboratory evaluation, unless clinically indicated. However, close outpatient follow-up is warranted. It is also important to note that complete vaccination to *Haemophilus influenzae* type B and streptococcus is not accomplished until a child's 6-month immunization series. In addition, although the streptococcus vaccine is heptavalent, there are many other serotypes that could become pathogenic; continued surveillance is warranted despite high rates of vaccination.

Age greater than 36 months

By 36 months of age, a child's immune system has developed to the degree that SBI is much less common. The incidence of occult bacteremia increases with higher temperatures and elevated WBC counts. Laboratory studies in febrile children above 36 months of age are dictated by the clinical presentation.

The widespread utilization of the *Haemophilus influenzae* type B (HIB) vaccine and heptavalent conjugate pneumococcal vaccine (PCV-7) has further challenged traditional diagnostic and therapeutic algorithms. Our approach to febrile children will undoubtedly transform as the microbiology of occult bacteremia changes in the era of universal vaccination.

General treatment principles

Management of fever

Early and appropriate antipyretic therapy should be instituted in all febrile children to facilitate behavioral observation and relieve the child's discomfort. Acetaminophen should be given at a dose of 15 mg/kg either per os (PO) or per rectum (PR) every 4–6 hours. Ibuprofen is also effective and can be given at 10 mg/kg PO every 6–8 hours in combination with acetaminophen.

Aspirin should be avoided in febrile children with viral syndromes (e.g., influenza and varicella), as it has been associated with the development of Reye syndrome.

Empiric antibiotic therapy

Immediate empiric antibiotic therapy and hospitalization are indicated in any ill-appearing or significantly immunocompromised febrile child. Antibiotics are typically administered immediately following diagnostic evaluation. If a delay in performing diagnostic testing occurs, antibiotics should not be withheld from ill-appearing patients. Initial empiric antibiotic coverage should be targeted at the most likely pathogens. For example, a child with clinical evidence of pneumonia should have antibiotic coverage against typical (e.g., *Streptococcus pneumoniae*) and atypical (e.g., *Mycoplasma pneumoniae*) community-acquired pulmonary pathogens.

In many febrile children, ED evaluation does not reveal a definitive source of infection. These patients are still at risk for occult bacteremia. Initial empiric antibiotic coverage against bacteremia in children is often a third-generation cephalosporin (e.g., ceftriaxone or cefotaxime). Ceftriaxone should generally be avoided in the first month of life because of the theoretical risk of encephalopathy caused by displaced bound bilirubin. The pathogen in a majority of cases of occult bacteremia is *Streptococcus pneumoniae*. Additional antibiotic coverage (ampicillin) against *Listeria monocytogenes* is recommended in febrile children in the first month of life. This regimen will not provide adequate anaerobic coverage, nor will it provide adequate anti-pseudomonal coverage. Additional antibiotic coverage needs to be administered if these organisms are likely pathogens.

Additional empiric antibiotic coverage may be indicated (e.g., vancomycin) against cephalosporin-resistant strains of *S. pneumoniae* in any child with a life-threatening infection. Finally, any neonate with evidence of a neonatal herpes virus infection should be hospitalized and receive empiric intravenous acyclovir therapy immediately.

All febrile infants less than 28 days of age, irrespective of appearance, should have a complete sepsis evaluation and be hospitalized following the administration of parenteral antibiotic therapy, such as ampicillin plus either cefotaxime or gentamicin. Infants between the ages of 28 days and 3 months meeting low-risk criteria* may or may not be treated with a parenteral dose of ceftriaxone (50 mg/kg), sent home and re-evaluated in 24 hours, if logistically feasible. If a complete sepsis evaluation (including lumbar puncture for CSF) is not performed, empiric antibiotics should not be administered, as they may partially treat meningitis and confound the interpretation of any subsequent CSF studies. All high-risk infants in this age group warrant empiric antibiotic therapy and careful consideration for hospitalization.

Special patients

Immune compromised

The management of immunodeficient pediatric patients with fever varies greatly depending on the specific immunodeficiency. In general, a lower threshold for diagnostic testing, empiric antibiotic therapy, and inpatient care is essential in this patient population. Febrile immune-compromised patients (e.g., chemotherapy patients and post-organ transplant) often require initial broad-spectrum antibiotic coverage (e.g., imipenem) while awaiting results of diagnostic testing. Communication with the patient's subspecialty physicians is a crucial part of their ED care. These physicians are excellent sources of additional clinical information that will optimize patient management. In the event these high-risk patients are sent home, careful documentation and detailed aftercare instructions are crucial.

Patients with indwelling devices

Indwelling devices (e.g., central venous lines) are common sources of infection. Careful inspection of the device's entry site into the skin may reveal signs of infection (i.e., erythema, fluctuance, induration, or tenderness). Patients with indwelling devices represent a management challenge. They are at substantially higher risk of seeding their prosthetic devices or having them serve as a source of infection. A low threshold for obtaining blood cultures in these patients is warranted. Strong consideration for empiric antibiotic therapy and hospitalization should be given to febrile children with indwelling devices and no definitive source of infection.

Disposition

All toxic-appearing febrile children should be hospitalized, irrespective of age. Serial monitoring of vital signs and clinical appearance will influence the selection of an appropriate level of inpatient care (e.g., intensive care unit). As a general rule, any febrile child below 28 days of life, regardless of clinical appearance, should be hospitalized and treated with empiric antibiotic therapy pending culture results from a complete septic work-up.

The decision of whether to hospitalize patients older than 1 month with a fever is based on a variety of factors. These include presence or absence of systemic symptoms (e.g., respiratory symptoms), comorbidities, course of illness, access to health care and parental reliability. If a decision is made to discharge such a patient home, a thorough discussion and documentation of aftercare instructions and return precautions are paramount. It is also essential to ensure that the patient has follow-up to assess whether or not the child is improving. Children who remain febrile or become less interactive with their environment should be reassessed immediately.

Pearls, pitfalls and myths

- Failure to realize that neonates with SBI often have subtle and nonspecific presentations (e.g., poor feeding), or no symptoms at all (other than fever).
- The most accurate method for assessing core body temperature in the ED is with a rectal thermometer.
- Always inquire about antipyretic administration prior to ED presentation. Antipyretics may temporarily mask a fever, leading to the omission of an otherwise indicated diagnostic evaluation.
- An infant's fever should not be attributed to teething, bundling, or a parent's incorrect measurement until SBI has been eliminated as the etiology.
- Normal auscultation of the chest does not exclude the presence of pneumonia in young, febrile children.
- The urinary tract is a common site of bacterial infection in infants. Failure to obtain a catheterized urine sample may lead to misleading laboratory results.
- Failure to recognize that vaccinated children are not completely immunized against streptococcus and *Haemophilus influenzae* type B until after the 6-month immunization series.
- Failure to administer antibiotic coverage (e.g., vancomycin) against cephalosporin-resistant strains of *S. pneumoniae* in patients with life-threatening infections and organisms likely to be resistant.
- Failure to provide prompt parental antibiotic therapy (e.g., intramuscular route) in ill-appearing febrile children when there is a delay in obtaining intravenous access or completing diagnostic testing.

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*Low-risk criteria include being previously healthy, having no focal bacterial infection on physical examination, a WBC count between 5,000 and 15,000/mm^3, a neutrophil band count below 1,500/mm^3, UA with less than 10 WBCs/hpf and no bacteria on urine Gram stain, and CSF with less than 8 WBCs/hpf and no bacteria on Gram stain.

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29 Gastrointestinal bleeding

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Scope of the problem

Bleeding may occur anywhere along the gastrointestinal (GI) tract, and ranges in severity from microscopic and asymptomatic to massive and life-threatening. The hemodynamic stability of a patient with GI bleeding (GIB) is often difficult to predict. Always assume the worst and respect the potential threat posed by GIB.

GIB may occur at any age, but is most common between the fifth and eighth decades. It has the highest mortality in patients over 60 years of age. Gastrointestinal bleeding may be divided into upper (UGIB) and lower (LGIB), with UGIB more likely to be life-threatening. UGIB occurs in 50–150 per 100,000 adults each year. There are 250,000 hospital admissions each year for UGIB in the United States alone, with costs of approximately $1 billion.

Anatomic essentials

The ligament of Treitz crosses the small intestine at the junction of the duodenum and jejunum. Bleeding above the ligament of Treitz is considered UGIB, whereas bleeding below the ligament is considered LGIB. *Hematemesis*, the vomiting of blood, is a sign of UGIB. *Coffee-ground emesis* is usually seen with UGIB and indicates that blood has been in the stomach long enough to have been partially digested. *Melena*, which is black, tarry, foul-smelling stool, results from the digestion of blood as it transits the small intestine. Melena is most often a sign of UGIB but is seen in some cases of LGIB. Maroon or dark red stool is referred to as *hematochezia* and is most often associated with LGIB. Bright red blood per rectum (BRBPR) is typically from distal LGIB bleeding (descending colon, rectosigmoid, or rectum), but also occurs in some cases of brisk bleeding arising more proximally in the GI tract. Blood is a cathartic, and severe UGIB may markedly decrease intestinal transit time.

Meckel's diverticulum, a congenital malformation in which gastric tissue is found in the GI tract outside of the stomach (most often in the distal ileum), may result in GIB secondary to ulceration.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 29.1).

History

Where are you bleeding?

Patients usually complain of blood either in their vomitus or their stool. Blood per rectum may be bright red, maroon-colored, or melenic. Blood may appear on toilet paper, or may be mixed with stool. Although not definitive, the location of bleeding (i.e., vomitus vs. stool) may help to localize the source of the bleeding (upper vs. lower). Be sensitive to patients who may have visual impairment, difficulty with colors, or are elderly and not able to give you information about their stool or vomitus.

Does your vomitus look brown or like coffee grounds?

Patients may not recognize that these are symptoms of UGIB.

Have you had dark black, tarry, or sticky stool (melena)?

Melena occurs in about two-thirds of patients with UGIB and up to one-third of patients with LGIB. Melena may result from as little as 60 mL of UGIB.

How much bleeding have you had?

Quantifying the bleeding is often difficult for patients and prehospital personnel. Ask if the blood in vomitus was merely streaking or frank blood, including the presence of any clots, as this suggests heavier bleeding. Regarding bleeding per rectum, patients may only see a small amount of blood on the toilet paper, or may be passing clots. It only takes a small amount of blood to change the color of toilet bowl water (about 5 mL). When possible, try to have the patient quantify their blood loss as a teaspoon or less, between a teaspoon and a cup, or more than a cup of blood.

When did the bleeding start?

Bleeding over days or weeks may appear mild, but can result in large blood loss and hemodynamic instability. Heavy bleeding just prior to presentation sometimes resolves spontaneously, but should prompt concern for ensuing hemodynamic instability.

Is the bleeding painful or painless?

Abdominal pain is transmitted via visceral or somatic nerves. If inflamed, the stomach and intestines can cause

Table 29.1 Gastrointestinal bleeding red flags

History	Concerning diagnosis
Previous GI bleed	Recurrence/progression of prior disease
Brisk, active, voluminous hematemesis	Massively hemorrhaging ulcer or varix
Known portal hypertension, cirrhosis, or hepatitis	Upper GI variceal/portal hypertensive bleeding (and likely coagulopathy)
Alcohol abuse	Portal HTN and varices
Previous thoracic or abdominal surgery (especially if aortic)	Aortoenteric fistula
Taking anticoagulants	Coagulopathy
Syncope	Massive hemorrhage GI hemorrhagic induction of vagal tone Acute coronary syndrome or dysrhythmia
Chest pain	Acute coronary syndrome Boerhaave syndrome or Mallory-Weiss tear Gastric/duodenal ulcer or perforation
Renal disease	Uremic platelet inhibition (DDAVP)
Toxic ingestions	Heavy metals (iron, lead) Caustic liquids (bases > acids, volatile alcohols, solvents)
Fever	Infectious, autoimmune, malignancy
NSAIDs, corticosteroids	Bleeding gastric ulcers
Weight loss, fatigue, pain	Autoimmune, malignancy
Severe pain (out of proportion to examination), endocarditis/embolic disease, severe atherosclerosis	Mesenteric ischemia
Significant constipation	Diverticular disease, hemorrhoids
Examination finding	**Concerning diagnosis**
Hemodynamic instability	Hemorrhagic shock
Altered mental status	Hypotension/hypoxia/intoxication Severe anemia Infection Ammonia/urea-related encephalopathy Caustic ingestion (suicide attempt)
Thoracic/abdominal scars (pulsatile abdominal masses)	Aortoenteric fistula Vascular malformation
Hematemesis with hematochezia (BRBPR)	Brisk UGIB
Stigmata of liver disease (caput medusa, spider telangiectasias, icterus, splenomegaly, ascites, asterixis)	Variceal UGIB Coagulopathy Spontaneous bacterial peritonitis
Fever	Infectious Autoimmune Malignancy
Other bleeding, easy bruising, petechiae	Coagulopathy or hemorrhage
Abdominal tenderness	Ulceration or perforation, SBP or other infection, malignancy, autoimmune
Pretracheal subcutaneous emphysema	Boerhaave syndrome or esophageal perforation with ascending pneumomediastinum

BRBPR: bright red blood per rectum; DDAVP: desmopressin acetate; GI: gastrointestinal; HTN: hypertension; NSAIDs: nonsteroidal antiinflammatory drugs; SBP: spontaneous bacterial peritonitis; UGIB: upper gastrointestinal bleeding.

visceral pain. The oropharynx and anal verge have somatic pain fibers, and bleeding from these areas may result in somatic pain. In cases of UGIB, pain may represent an ulcer or gastritis. In LGIB, pain may be associated with inflammatory bowel disease or infectious diarrhea. Somatic pain may arise from anal fissures or external hemorrhoids. Painless bleeding is usually from intestinal sources or internal hemorrhoids (without inflammation).

Did you have vomiting or retching prior to hematemesis?

These symptoms suggest a Mallory–Weiss tear (partial) or Boerhaave's syndrome (full-thickness rupture) of the esophagus. Mallory-Weiss tears are frequently benign and resolve spontaneously, whereas Boerhaave's syndrome may result in a life-threatening mediastinal infection and septic shock. Unfortunately, alcohol intoxication is involved in many cases of esophageal injury, and a history of vomiting may not be reported or apparent.

What other symptoms do you have?

- *Are you dizzy or lightheaded?* Patients with large blood loss may have symptoms of hypovolemia, orthostasis, or shock.
- *Are you having chest pain or shortness of breath?* Blood loss and shock may precipitate cardiac ischemia and metabolic acidosis, resulting in tachypnea. Also consider non-ischemic causes of pain, such as Boerhaave's syndrome. It should be noted that most life-threatening causes of bleeding are not associated with pain.
- *Have you had a fever?* Fever may accompany infectious gastroenteritis, inflammatory bowel disease and sepsis-associated esophageal perforation.

Have you ever had gastrointestinal bleeding before?

Bleeding from a previous site recurs in 60% of cases.

What other medical problems do you have?

Previous ulcers or gastritis can recur. Patients with *Helicobacter pylori* infection have a higher incidence of gastritis and UGIB. Liver disease with portal hypertension can lead to bleeding gastroesophageal varices. Patients with bleeding disorders, such as hemophilia and thrombocytopenia, can require specific treatment with blood products and factors. Colonic polyps, diverticulae, vascular malformations and carcinomas can cause LGIB.

What prior surgeries have you had?

Any patient who has had an aortic aneurysm repair is at risk for an aortoenteric fistula. The graft can erode through the aorta into the bowel and can lead to catastrophic blood loss.

Have you ever had an endoscopic procedure?

Documented ulcers, varices, or diverticulae can give information about current bleeding. Previous banding or sclerosing of esophageal varices raises the risk of repeat bleeding due to portal hypertension or hepatic coagulopathy.

What medications do you take?

Aspirin, nonsteroidal antiinflammatory drugs (NSAIDs) and corticosteroids increase the likelihood of bleeding gastric ulcers. Patients on anticoagulants may have significant GIB. Iron ingestion may cause significant GIB, whereas both bismuth and iron may produce black-colored stool that simulates melena. Beets and red wine may cause dark stool or mimic bloody stools.

Do you drink alcohol?

Alcohol ingestion increases the likelihood of gastritis. It also contributes to coagulopathy from malnutrition, liver disease and bone marrow suppression. Alcoholic cirrhosis can progress to portal hypertension with associated esophageal and gastric varices and hemorrhoids, all which can present with significant GIB.

Physical examination

The primary goal of the physical examination is to assess the severity of the patient's illness and, if possible, quantify the amount of blood loss. This can be difficult as patients with GIB may appear acutely ill with shock or may be asymptomatic. The secondary goal of the examination is to establish the location of GIB. Although physical examination may provide clues to the site of bleeding, it is usually not possible to determine the location with certainty until endoscopy or other imaging studies have been performed. The rectal examination is very important in GIB.

General appearance

Cool, clammy, pale skin, a decreased level of consciousness, and/or respiratory distress is concerning, as these imply that the patient is acutely ill, in shock, and needs immediate resuscitation.

Vital signs

Signs of blood loss can be identified through the vital signs. Tachycardia is generally the first indicator of blood loss and tends to correlate with mild (stage 2) hemorrhagic shock. Hypotension is generally associated with more significant hemorrhage, indicating moderate or severe (stage 3 or 4) hemorrhagic shock, and should prompt immediate aggressive resuscitation. Vagal stimulation from the GIB itself or medications that the patient is taking (e.g., beta-blockers) may mask the tachycardic response of stage 2 shock and further promote hypotension. Patients with cardiac conduction disorders or pacemakers may be unable to respond to bleeding with pulse changes. Vital signs can be falsely reassuring in patients having great reserve, such as children. Such patients may not demonstrate a drop in blood pressure until they are dangerously hypovolemic. Postural measurements of vital signs can also be misleading; postural changes may be unrelated to blood loss from GIB (e.g., in elderly patients and diabetics with autonomic instability). An increase in the respiratory rate can be subtle, especially if there is no shortness of breath or signs of respiratory distress, but is also an important sign of underlying anemia and acidosis.

Head, eyes, ears, nose and throat

Look for signs of liver disease such as scleral icterus. Pale conjunctivae suggest anemia and blood loss. Inspect the oropharynx for signs of bleeding from the nose or throat. Posterior epistaxis and oral lacerations can mimic GIB because blood is swallowed, resulting in hematemesis, a positive nasogastric (NG) aspiration, and/or a positive hemoccult test. Postoperative tonsillectomy bleeding can occur 5–7 days after the procedure as the eschar falls off; this can compromise the airway and requires ear, nose and throat (ENT) evaluation, even if the bleeding has stopped.

Abdomen

Observe for distension. Auscultate for either increased or decreased bowel sounds, although these findings are nonspecific. Absent bowel sounds and diffuse tenderness or peritoneal signs may represent a surgical emergency. Tenderness in the epigastric region may be secondary to a gastric ulcer or gastritis. An enlarged or tender liver may be a clue to liver disease, but in end-stage liver disease, the liver is typically small and nontender. In patients with advanced cirrhosis and resultant portal hypertension, the spleen is enlarged. Another sign of portal hypertension may include prominent blue vessels around the umbilicus, known as *caput medusa* (or medusa's head). Ascites suggests advanced liver disease with possible coagulopathy or portal hypertension. Palpation of an aortic aneurysm or a midline abdominal scar should raise the concern for an aortoenteric fistula.

Rectal

The rectal examination is essential. Inspection may show anal fissures or hemorrhoids. Digital examination may detect masses or tenderness. The stool should be checked for gross or occult blood. As little as 5 mL of blood in the GI tract will give a positive hemoccult test. *Anoscopy* may be helpful if the GIB is believed to be from a lower GI source. This allows the anal verge to be visualized to identify a bleeding internal hemorrhoid. Caution is advised, however, before attributing all bleeding to a site seen on anoscopy, as bleeding may also occur from a more proximal site in the GI tract.

Skin

The skin should be examined for purpura or petechiae, which suggest an underlying coagulopathy. Stigmata of liver failure include spider angiomata, palmar erythema and jaundice.

Differential diagnosis

Tables 29.2 and 29.3 describe causes of upper and lower GI bleeding, respectively.

Diagnostic testing

Occult blood

The presence of hemoglobin in the stool may be detected using a hemoccult card and specialized developer. Hemoccult testing can detect blood not seen by the naked eye. After developer has been applied to the back of the filter paper (at the stool test site), a blue color change indicates the presence of hemoglobin and probable blood. Tests can be positive up to 14 days after a single episode of bleeding. False-positive results can occur from consumption of red meat, blood-containing food, iodide, cantaloupe, uncooked broccoli, turnip, radish, or horseradish 3 days prior to the test. False-negative results can occur if the blood has not yet transited the intestinal tract, if magnesium-containing antacids have been used, if ascorbic acid has been consumed, or if the test is performed incorrectly.

Laboratory studies

Complete blood count

Hemoglobin of <10 g/dL suggests significant anemia that may be acute or chronic, and may (or may not) be associated with hemodynamic instability. Vital signs and hemoglobin measurements should be compared with previous values, if known, and repeated early in the course of the ED visit. A patient with a hemoglobin of <8 g/dL most often requires blood transfusion, since their reserve is insufficient in the setting of ongoing hemorrhage.

Beware of the confounding variables when assessing hemoglobin levels. With fluid resuscitation, the red blood cell mass becomes diluted and the hemoglobin decreases, not necessarily related to blood loss. Conversely, blood counts may be normal in ongoing hemorrhage if the bleeding began recently and the circulating blood concentration has yet to equilibrate from fluid resuscitation. Keep this in mind when using a single initial blood count to determine therapy or disposition. Serial hemoglobin measurements are much more useful and important.

The white blood count (WBC) may be elevated in infectious diarrhea or inflammatory bowel processes.

Low platelet counts (thrombocytopenia) increase the likelihood of bleeding, result in more severe hemorrhage, and should be corrected if platelets are less than 50,000/mL with ongoing bleeding. Thrombocytopenia often accompanies other coagulopathies such as those seen with chronic liver or renal disease, and may prompt additional resuscitation with fresh-frozen plasma (FFP), vitamin K, or Desmopressin acetate (DDAVP).

Blood urea nitrogen and creatinine

Elevated blood urea nitrogen (BUN) may suggest UGIB in the appropriate clinical setting. The serum urea level rises as protein in the blood is digested and absorbed from the GI tract. Be cautious when evaluating the BUN, as it may also be elevated with dehydration or renal failure. A BUN/creatinine ratio >20 suggests

Table 29.2 Differential diagnosis of upper gastrointestinal bleeding

Diagnosis	Symptoms	Signs	Work-up
Aortoenteric fistula	Range from mild bleeding to severe blood loss. Painless. Occurs in patients with history of AAA repair or AAA.	May be in shock from severe bleeding or have no active bleeding and a normal examination.	Urgent vascular surgery evaluation
Gastritis and esophagitis	Usually hematemesis or coffee-ground emesis. May have epigastric discomfort, melena, hematochezia, or no symptoms.	May have positive NG return for blood or coffee-ground material.	Early endoscopy within 24 hrs
Mallory–Weiss tear	Hematemesis after vomiting. Usually stops spontaneously.	NG suction may be positive. Usually stable.	Early endoscopy within 24 hrs
Ulcer – duodenal	Melena or hematochezia. May have hematemesis, coffee-ground emesis, abdominal discomfort, or no symptoms.	NG positive for blood or coffee-ground material, but often negative if bleeding is distal to pyloric sphincter.	Early endoscopy within 24 hrs
Ulcer – gastric	Hematemesis or coffee-ground emesis. May have epigastric discomfort, melena or hematochezia, or no symptoms.	NG aspirate may be positive for blood or coffee-ground material.	Early endoscopy within 24 hrs
Varices – esophageal or gastric	Hematemesis may be severe or mild. Usually have melena or hematochezia, but this may be delayed due to transit time.	NG aspirate usually positive and may have clots. May not clear with lavage. May be hemodynamically unstable.	Early endoscopy within 24 hrs. If continued bleeding, more emergent endoscopy necessary. May require Sengstaken–Blakemore, Minnesota or Linton tubes to stop severe bleeding and stabilize.

AAA: abdominal aortic aneurysm; NG: nasogastric.

Table 29.3 Differential diagnosis of lower gastrointestinal bleeding

Diagnosis	Symptoms	Signs	Work-up
Anal fissure	Painful bowel movement with blood on toilet paper.	Seen on external examination and very tender to palpation.	None necessary
Angiodysplasia	Painless red bleeding from rectum. May have melena.	BRBPR or melena. May be hemodynamically unstable.	Early colonoscopy (rapid bowel preparation needed). May need tagged red cell study or angiography to identify site of bleeding. If unstable, consult surgery.
Carcinoma	Weight loss or weight gain. Change in caliber of stool. Often asymptomatic.	May have a palpable abdominal or rectal mass. May be cachectic.	Colonoscopy, biopsy, surgical evaluation
Diverticulosis	Painless bleeding from rectum.	BRBPR. May be hemodynamically unstable.	Early colonoscopy (rapid bowel preparation needed). May need tagged red cell study or angiography to identify site of bleeding. If unstable, consult surgery.
Hemorrhoids	*External* – painful bleeding with blood on stool and toilet paper *Internal* – painless bleeding	Seen on external examination or anoscopy	Outpatient surgery evaluation
Infectious diarrhea	Painful diarrhea with fever and blood or pus in stool.	Heme-positive stool, diarrhea	Stool cultures, Gram stain, fecal WBC. Antibiotics indicated.
Inflammatory bowel disease	Abdominal pain and rectal bleeding. Weight loss. Fever.	BRBPR, melena, or heme-positive stool. Abdominal tenderness.	CBC, colonoscopy. May need admission for initial diagnosis or if symptoms are marked. May require surgical consult.
Ischemic colitis	Severe abdominal pain and rectal bleeding.	Diffuse abdominal pain or peritonitis. May have pain out-of-proportion to examination.	Urgent surgical evaluation. May need angiographic intervention. CT for air in intestinal wall (pneumatosis intestinalis).
Meckel's diverticulum	Painless melena or hematochezia. May have abdominal pain.	May have chronic anemia or acute blood loss	Often requires angiography or tagged red cell scanning. Surgical excision is diagnostic and therapeutic.
Upper GI bleed	Most common cause of massive lower GI bleeding is upper GI bleeding. May have hematemesis or abdominal pain.	May present in shock	Immediate GI consultation. CBC, coagulation studies, type and crossmatch, and immediate transfusion depending on clinical condition.

BRBPR: bright red blood per rectum; CBC: complete blood count; CT: computed tomography; GI: gastrointestinal; WBC: white blood cell.

dehydration, or a "pre-renal" cause. Whatever the etiology, elevated BUN interferes with platelet function and aggregation, and DDAVP may be necessary to help offset the detrimental effects of elevated BUN on platelet aggregation.

Type and crossmatch

In patients who require emergent transfusion upon arrival, transfusion should be initiated with universal-donor uncrossmatched blood (O negative or positive). However, it is preferable to transfuse type-specific blood whenever the patient's condition allows. If the patient is stable, a type and screen may be sent, followed by crossmatch should the patient deteriorate.

Prothrombin time

Patients who have liver disease, vitamin K deficiency, or who take warfarin (Coumadin) may have a coagulopathy that requires correction to stop the bleeding.

Electrocardiogram

Cardiac ischemia may result from hemorrhagic shock. Any patient over 50 years of age or with a history of heart disease, significant anemia, hypotension, chest pain, shortness of breath, or other evidence of shock should have an electrocardiogram (ECG). The ECG may reveal evidence of ischemia or infarction in the setting of GIB. If such ECG changes are seen, transfusion and further work-up for acute coronary syndrome should follow.

Nasogastric tube

Routine placement of a nasogastric (NG) tube for UGIB is controversial. NG tubes may assist in determining the location and degree of bleeding; however, they are uncomfortable and may yield misleading negative aspirates in cases of UGIB. Some apparent cases of LGIB are actually brisk UGIB that may be detected by NG evaluation. In the case of severe LGIB, it is reasonable to place an NG tube, as a positive blood return provides evidence of an upper source.

An NG tube may show active bleeding or coffee-ground material. In 10–15% of UGIB patients, bright red blood or clots are found. Gentle *gastric lavage* with saline or sterile water may be performed to see if bleeding has stopped; lavage is continued until the blood clears. If bleeding continues, the NG tube is left in place. If no blood or coffee grounds are found in the NG effluent, the tube may be removed. False-negative NG aspirations are common and may occur with intermittent bleeding or duodenal bleeding, if spasm of the pylorus prevents the reflux of blood into the stomach. If bile is present and blood is absent in the NG aspirate, active bleeding above the ligament of Treitz is unlikely. False positives may occur in the case of traumatic tube placement with bleeding from the nasopharynx. The aspirate should be tested for occult blood using either Gastroccult or a urine test strip for blood, as both of these tests are pH-independent.

In the case of suspected esophageal varices, NG tubes must be placed carefully, if at all; this is an area of ongoing debate. Do not force the tube if resistance is met. Although it is unclear whether NG tube placement aggravates hemorrhage from varices or Mallory–Weiss tears, numerous case reports have cited this complication. Although patients who have had gastric bypass surgery or fundoplication generally should not have an NG tube placed, this should always be discussed with the appropriate service.

Anoscopy

Anoscopy can be performed at the bedside to evaluate for the presence of internal hemorrhoids. It is indicated in patients with mild rectal bleeding without an obvious source. Results of anoscopy must be interpreted with caution.

Radiologic studies

Plain radiographs

Plain films of the abdomen are usually not indicated in cases of GIB. Concern for ruptured viscus associated with vomiting or in suspected cases of gastric or duodenal ulcers should trigger a plain upright chest radiograph. This may reveal free air under the diaphragm (e.g., perforated ulcer) or air in the mediastinum (e.g., ruptured esophagus). A chest radiograph may also be indicated if there is concern for cardiac ischemia. A plain abdominal film may show iron tablets in a case of suspected iron ingestion.

Upper gastrointestinal studies

Barium contrast studies are of limited value in the emergency management of GIB, and are generally contraindicated because the use of barium can limit the utility of subsequent endoscopy or angiography. Barium can also cause problems in the case of esophageal rupture by entering into the mediastinum.

General treatment principles

The initial treatment approach is the same for upper and lower GI bleeding. Recognition of acutely ill patients is paramount. The ABCs (airway, breathing, circulation) are the first priority. UGIB patients can develop airway difficulty as a result of severe hematemesis from a bleeding varix or ulcer. Severe blood loss may also result in a decreased level of consciousness. In such critically ill patients, securing the airway with endotracheal intubation is usually necessary. In patients with severe blood loss, supplemental oxygen, a cardiac monitor, and two large-bore intravenous (IV) catheters (18 G or larger) should be placed immediately. If hypotension, tachycardia, or obvious ongoing blood loss is detected, resuscitation should be initiated with a crystalloid bolus, followed by early transfusion with type O blood (unless type-specific blood is available). The initial crystalloid bolus should be 2 L for adults or 20 mL/kg for children.

Upper gastrointestinal bleeding

Antacids

Antacids are not indicated for the treatment of UGIB. Antacids have not been shown to decrease the incidence of bleeding, and can complicate urgent or emergent esophagogastroduodenoscopy (EGD) by coating the esophageal or gastric mucosa.

Antibiotics

Antibiotic therapy reduces complications of UGIB in patients with advanced cirrhosis. A significant number of these patients (about 20%) have concurrent spontaneous bacterial peritonitis (SBP), and targeted antibiotic therapy can reduce infectious complications, decrease bleeding complications and decrease the need for endoscopic hemostasis.

Histamine blockers and proton pump inhibitors

Histamine (H2) blockers and proton pump inhibitors (PPIs) decrease the acid secretion that contributes to gastric or duodenal ulcer formation. These medications are routinely given to patients with UGIB, not to stop the bleeding but to initiate ulcer or gastritis treatment. This may reduce future bleeding. In patients with severe, acute UGIB, a continuous infusion of a PPI appears to decrease the need for blood transfusion and may decrease mortality.

Somatostatin and octreotide

These are vasoactive peptides that cause selective constriction of the splanchnic vascular bed and decrease gastric acid secretion. They decrease blood flow to the esophagus, stomach and duodenum, and may decrease blood loss from UGIB. Somatostatin is naturally occurring, whereas octreotide is a synthetic equivalent. Although studies of octreotide have shown only a modest benefit, its low-risk profile is advantageous for patients with life-threatening GIB, especially if liver disease and/or portal hypertension is suspected.

Vasopressin

Vasopressin is a vasoconstrictor that affects the entire circulatory system, including the splanchnic bed. It is extremely potent and may be used in an exsanguinating patient when endoscopy is unavailable or not possible. The use of vasopressin requires caution, as end-organ damage may occur from excessive vasoconstriction and subsequent hypoperfusion.

Esophagogastroduodenoscopy

EGD is both diagnostic and in many cases therapeutic. In severe bleeding, the airway must be secured before emergency EGD is performed. Endoscopy provides visual evaluation of the esophagus, gastric mucosa and proximal duodenum. If performed within 12–24 hours of hemorrhage, EGD identifies lesions in the great majority of UGIB patients. When the site of bleeding has been identified, therapeutic intervention may be indicated. Esophageal varices can be sclerosed, injected, or banded. Bleeding gastric or duodenal ulcers can be injected and sclerosed. If perforation is detected, surgery may be necessary.

EGD with sclerotherapy has not been found to reduce mortality or rebleeding compared with vasoactive substances or other treatments in the initial treatment of severe UGIB from esophageal varices. It is, however, the diagnostic modality of choice, and repeat sclerotherapy or banding is the long-term treatment of choice for esophageal varices, in addition to treating underlying causes. It is difficult to perform endoscopy on critical patients who are bleeding heavily, as large amounts of blood may limit visualization with the endoscope. In these cases, tamponade with a gastric balloon device may be necessary.

Gastro-esophageal tamponade

Direct pressure (tamponade) of bleeding esophageal varices may be performed when vasoactive medications are not effective, and endoscopy is either ineffective or unavailable. Tamponade may temporarily control severe hemorrhage in up to 80% of patients with bleeding esophageal varices. It can be used for 12–24 hours. Tamponade may be accomplished with a specialized gastric tube that incorporates two expanding balloons. One balloon is first expanded in the stomach. A second balloon is then expanded in the esophagus. There is a suction eye at the tip. The Sengstaken–Blakemore tube is a multi-lumen tube used for tamponade (Figure 29.1). Some tubes have a modification incorporating suction eyes in the esophagus to decrease the risk of aspiration. A Linton tube has a single stomach balloon that is larger and more effective with gastro-esophageal varices.

Esophageal tamponade carries significant complications, including esophageal rupture, airway compression from the esophageal tube, and aspiration. Most cases of UGIB can be controlled with endoscopy or medications, but tamponade remains an effective modality for severe variceal bleeding.

Interventional radiology

Patients with variceal bleeding may be treated with an emergent transjugular intrahepatic portosystemic shunt (TIPS) procedure. The interventional radiologist creates a shunt between the portal circulation and the vena cava, reducing the high portal pressures that lead to bleeding.

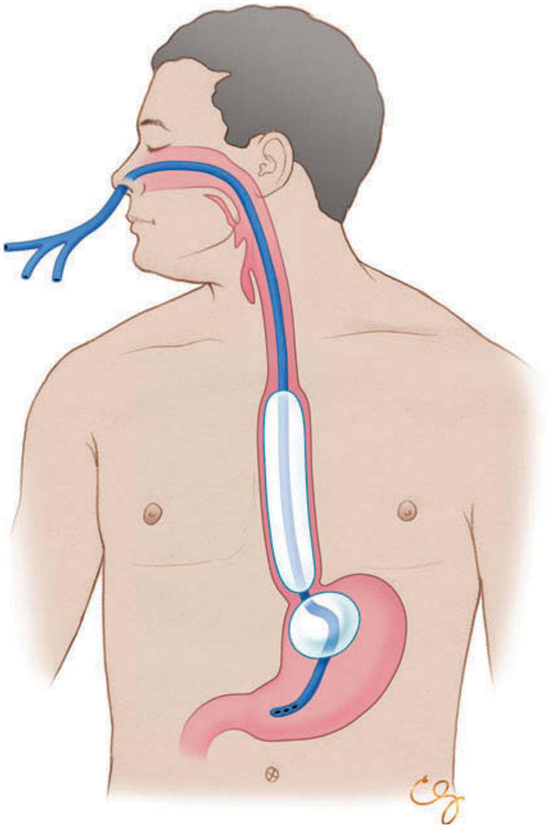

Figure 29.1
Sengstaken–Blakemore tube. © Chris Gralapp.

Surgery

Although rarely necessary, surgery is an important option for cases of severe UGIB refractory to other treatments. In patients with extreme blood loss, EGD can be performed in the operating room under general anesthesia to allow sclerotherapy or to guide surgical treatment.

Due to the risk of aortoenteric fistula, patients with aortic grafts and active GIB should be emergently evaluated by a vascular surgeon.

Lower gastrointestinal bleeding

Colonoscopy

Colonoscopy provides direct visualization of bleeding sources and the opportunity for direct therapeutic intervention. Direct epinephrine injection or electrical cauterization can stop LGIB and is considered the intervention of choice. Colonoscopy is difficult to perform emergently because it requires adequate bowel preparation, which is rarely achieved or advised in the ED. If urgent colonoscopy is anticipated, an emergent bowel preparation can be performed with NG administration of 2 L of polyethylene glycol. This can lead to volume loss by osmotic diuresis, so the patient's hemodynamic status should be followed closely, usually in the ICU setting.

Sigmoidoscopy

Sigmoidoscopy is performed to evaluate the sigmoid colon for diverticulae, polyps or tumors. It is reserved for cases of mild LGIB.

Angiography

Angiography can detect as little as 0.5 mL of GIB per minute. Although angiography is rarely utilized for UGIB (only 1% of cases), it is commonly used for LGIB. Angiography can identify the site of bleeding, but rarely identifies the cause. If bleeding is detected, vasopressin or epinephrine can be injected locally, or embolization can be performed to stop the bleeding. Complications of embolization include dye reaction, arterial dissection, or ischemia related to vasopressin. The use of angiography in the setting of GIB depends on institutional practice, availability, and operator expertise. It is usually reserved for significant, persistent, or intermittent LGIB that cannot be localized by endoscopy, and is usually performed after admission to the hospital.

Tagged red blood cell imaging

Technetium (^{99m}Tc) tagged red blood cells can detect LGIB of 0.1 mL/min. An initial scan is done, and delayed scans are compared in an attempt to localize bleeding. This scanning is rarely done in the ED setting. Tagged cell scans should be ordered in consultation with either the gastroenterology or surgical service.

Vasopressin

Vasopressin is a vasoconstrictor that affects the entire circulatory system, including the splanchnic bed. It is very potent and should be used only in an exsanguinating patient with LGIB. Colonoscopy lacks utility for the initial stabilization of LGIB, so vasopressin may be necessary until surgery or emergent preparation and subsequent colonoscopy can be performed. Use vasopressin with caution because end-organ damage may occur.

Surgery

When angiography and interventional radiological techniques fail to treat LGIB, emergent surgery may be necessary. Angiography may identify the location of bleeding, allowing for planning prior to surgery (e.g., hemicolectomy).

Indications for transfusion

Patients with hypotension or evidence of volume depletion after an initial crystalloid bolus (2 L in adults or 20–40 mL/kg in children) should be transfused. Patients with severe distress, cardiac ischemia, or massive blood loss should receive blood products as soon as possible. Patients with falling hemoglobin values and ongoing blood losses should be transfused. If type-specific blood is not available, type O blood (Rh-negative in females with childbearing potential) should be transfused a maximum volume of 4 units to avoid transfusion reactions. Type-specific and then crossmatched blood should be used as soon as possible.

Indications for plasma, vitamin K and platelets

There is growing evidence supporting the early administration of plasma, vitamin K and platelets. FFP should be administered in patients who have GIB and elevated prothrombin (PT) times, such as patients with liver disease, vitamin K deficiency, warfarin therapy, or other coagulopathies (e.g., disseminated intravascular coagulation [DIC] or hemophilia). It routinely takes about 45 minutes to thaw FFP, so it should be ordered early in the course of resuscitation. Vitamin K should be given in the ED; however, its effect is delayed and will not stop acute bleeding. Platelets should be given to most patients with ongoing bleeding and platelet counts <50,000.

Special patients

Pediatric

The most common cause of UGIB in children is esophagitis, followed by gastritis, ulcer, varices and Mallory–Weiss tears. The most common causes of LGIB in children are anal fissures, infectious colitis, inflammatory bowel disease, polyps and intussusception. Formula intolerance should be considered in infants. Intussusception should be an early consideration in children with colicky pain and lethargy. Hematochezia or "currant-jelly" stool are late findings; one should not rely on these to make the diagnosis.

The management of GIB in children is similar to the approach in adults. More often, the cause of bleeding is benign and can be managed on an outpatient basis. The emergency physician should keep in mind, however, that much smaller blood losses may result in hemodynamic instability. Children with large or ongoing blood loss, vital sign abnormalities, or comorbidities should be admitted.

Elderly

Geriatric patients are often taking medications that can mask the typical signs of shock. Geriatric patients with GIB are more likely to present atypically (e.g., a change in mental status or generalized weakness), and have higher morbidity and mortality. Elderly patients may have visual difficulties making it difficult for them to identify blood, hematochezia, or melena. GIB may lead to cardiac ischemia or respiratory compromise in elderly patients. Emergency physicians must have a low threshold for admission and further evaluation of elderly patients.

Immune compromised

Patients with malignancy, human immunodeficiency virus (HIV), and those taking immunosuppressant agents are at a higher risk for infectious complications. Patients treated with steroids have a higher risk of forming ulcers that may bleed. Chronic steroid use may also prevent a proper stress response to bleeding and hypovolemia. Supplemental corticosteroids may be necessary for such a patient in refractory shock.

Disposition

Guidelines for specialty consultation or admission

Patients with UGIB and a history concerning for aortoenteric bleeding deserve emergent evaluation by a vascular surgeon; those with UGIB and suspected ulcer perforation should be rapidly seen by a general surgeon. Any patient who is unstable or has a hemoglobin less than 10 g/dL should be admitted to the hospital and evaluated by a gastroenterologist. In the case of unstable GIB, a gastroenterologist and/or general surgeon should evaluate the patient, and the patient should be admitted to the ICU. Ill-appearing patients with inflammatory bowel disease should be admitted for antibiotics and colonoscopy. Patients with LGIB from causes other than fissures, hemorrhoids, or mild proctitis should also be admitted for further evaluation. Patients with significant GIB and comorbidities should be considered for admission.

Guidelines for emergency department observation

A patient with UGIB that has stopped, with a small to moderate amount of bleeding and a hemoglobin greater than 10 g/dL may be evaluated in an observation unit for early diagnostic endoscopy. Serial hemoglobin levels are followed, looking for a drop of 2 g/dL or more, which usually leads to admission. Patients with LGIB are not routinely evaluated in an observation setting, because colonoscopy is usually warranted and is not easily done in a short-stay observation setting.

Guidelines for discharge

Historically, all patients with GIB were admitted. With continuing pressure to decrease hospital utilization, an attempt has been made to identify low-risk patients who can be safely evaluated on an outpatient basis. Stable UGIB patients with normal hemoglobin, no comorbidities, and a small amount of bleeding that has resolved may be discharged if close follow-up (1–2 days) is available. Patients with LGIB from a benign source (hemorrhoids or fissures) and stable vital signs may be discharged with primary care follow-up.

Discharged patients should be given precautions and instructions about any further bleeding, with specific instructions regarding when they should return to the ED or contact their primary care physician. Patients should return if bleeding recurs, or if symptoms of volume depletion (dizziness, lightheadedness, syncope or near-syncope), chest pain, shortness of breath, melena or hematochezia develop. Patients should also return if they have new symptoms.

Pearls, pitfalls and myths

- The most common cause of massive LGIB is actually from an upper GI site with a brisk transit time.
- The initial hemoglobin may not reflect an actual large amount of blood loss; serial measurements are necessary.
- NG aspiration may result in falsely negative results in UGIB.
- Previous aortic aneurysm repair requires early vascular surgery consultation for possible aortoenteric fistula.
- Be very cautious with elderly patients and GIB. Comorbidities make these patients highly susceptible to associated morbidity and mortality.
- GIB is a common cause of altered mental status and generalized weakness in the elderly.

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30 Headache

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Scope of the problem

Headache is a common complaint. According to the National Hospital Ambulance Medical Care Survey published in 2008, headaches or pain in the head accounted for over 3.3 million emergency department (ED) visits in the United States in 2006 (2.8%), making it the fourth leading reason for adults (15 years and older) to seek care in an ED. Many patients present with headache as part of a constitutional illness, making it an even more common ED complaint. A patient with a headache may have a serious or minor etiology. The differential diagnosis of headache is complex and extensive. Headache can be divided into primary or secondary categories (Table 30.1). Primary headaches, such as migraines, cluster and tension-type headaches, account for 90% of headaches in clinical practice. Secondary headaches include tumors, aneurysms and meningitis; these have an identifiable, distinct pathologic process in which head pain is a presenting symptom. Most patients presenting to the ED with headache have a benign condition requiring symptomatic treatment and referral. However, a small subset of patients who present with a headache will have a life-threatening illness. The primary goal of emergency physicians is to identify these patients and provide appropriate care.

Anatomic essentials

The pain from headache can originate from extracranial or intracranial structures. Extracranial structures that can cause pain include skin, blood vessels, muscles and bone. The brain parenchyma, the majority of the dura, the arachnoid and pia mater have no pain fibers and do not produce pain. Intracranial structures with pain fibers include venous sinuses, the dura at the base of the skull, dural arteries, the falx cerebri, and large arteries at the base of the brain. The fifth cranial nerve (CN V) carries pain fibers from structures above the tentorium and supplies most of the facial area. CNs IX, X and XI, along with upper cervical nerves, carry these pain fibers below the tentorium, resulting in pain referred to the neck and back of the head. Headache may be the result of sensory input originating in locations other than the brain. For example, children (and adults) often experience headache from inflammation or irritation of pharyngeal structures (CN IX-X). Irritation of the optic nerve (CN II), retina, or facial nerve (CN VII) often causes headache. Cervical (spinal) nerves 1–3 may be responsible for head pain or referred headache. The trapezius muscle attaches to the occipital bone, and strain or inflammation of this muscle often results in headache.

Table 30.1 Major categories of headaches

Primary
Migraines
Tension
Cluster
Secondary
Head trauma
Vascular disorders (stroke, intracranial hematoma, subarachnoid hemorrhage, unruptured vascular malformation, arteritis, venous thrombosis, arterial hypertension)
Non-vascular intracranial disorder (high or low cerebral spinal fluid pressure, non-infectious inflammatory disease, intracranial neoplasm)
Substance use or withdrawal
Infection (meningitis, encephalitis, brain abscess, or acute febrile illness of any type, including pharyngitis or sinusitis)
Metabolic disorders (hypoxia, hypercapnia, other metabolic abnormalities)
Cranial–facial disorders (pathology of cranium, neck, eyes, sinuses, and other cranial–facial structures)
Neuralgias
Toxicologic (carbon monoxide)

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 30.2).

History

A detailed history is the most important part of the evaluation of a patient with a headache.

How did the pain begin (sudden vs. gradual onset) and how long has it been present?

These are two crucial questions to ask while obtaining a history. A patient with sudden onset of a severe unprecedented headache, with or without neurologic deficits, should be investigated for ruptured aneurysm or subarachnoid hemorrhage (SAH). A headache of gradual onset that has persisted for weeks or months suggests tension headaches. New headaches that worsen in intensity over weeks are suspicious for mass effect associated with increased intracranial pressure (ICP). Episodic headaches with symptom-free intervals suggest migraine or cluster headaches. Frontal or occipital headaches that begin 24–48 hours after a lumbar puncture (LP), known as post-dural (or post-LP) headaches, may be secondary to a persistent cerebrospinal fluid (CSF) leak.

Table 30.2 Headache red flags

History	Concerning diagnosis
Sudden onset of HA (thunderclap)	SAH
Worst headache of life	SAH
HA dramatically different from past headaches	SAH
HA that begins with exertion	SAH
Photophobia, neck stiffness and fever	Meningitis
HA with fever	Meningitis, brain abscess
HA with fever following brain or sinus surgery	Meningitis, brain abscess
Multiple members of same household with HA, especially winter months or cold weather	Carbon monoxide poisoning
Limitation or pain with eye movement	Cavernous vein thrombosis
Head trauma	Traumatic SAH, epidural, subdural, intracerebral hemorrhage
Acute onset of a severe HA around one eye	Acute angle closure glaucoma
HA with unilateral weakness, aphasia	CVA, intracerebral hemorrhage
HA in a patient who is immunocompromised	Aseptic meningitis, cryptococcus, toxoplasmosis, tuberculous meningitis, or cytomegaloencephalitis
Unilateral HA with blurred vision in the elderly	Temporal arteritis, acute angle closure glaucoma
Young obese female with HA and blurred vision	Idiopathic intracranial hypertension
Headache and history of cancer	Brain metastasis
Unilateral posterior HA with nausea, vomiting and ataxia	Vertebral artery dissection
New onset of HA after the age of 50 years	Glaucoma, intracranial lesion, temporal arteritis
Examination finding	**Concerning diagnosis**
Altered mental status	Meningitis, brain abscess, encephalitis, ICH
Meningeal signs, including positive Brudzinski's or Kernig's signs	Meningitis
Positive "jolt" accentuation sign	Meningitis
Focal neurologic signs	CVA, SAH, intracerebral hemorrhage, abscess, tumor
Rash	Meningococcemia
Unilateral and then bilateral proptosis with paralysis of CNs III, IV, VI	Cavernous vein thrombosis
Red eye with a fixed, mid-dilated pupil, corneal clouding, shallow anterior chamber, increased intraocular pressure	Acute angle closure glaucoma
Tenderness over temporal artery	Giant cell arteritis
Papilledema	Idiopathic intracranial hypertension, intracranial mass, intracerebral hemorrhage
Hemiparesis, ataxia, signs of brainstem or cerebellar ischemia	Vertebral artery dissection
Hemiplegia, pupillary dilation and obtundation	ICH or mass with herniation, berry aneurysm with possible rupture
Ipsilateral Horner's syndrome and contralateral hemispheric findings (aphasia, neglect, visual disturbances and hemiparesis)	Carotid artery dissection

CN: cranial nerve; CVA: cerebrovascular accident; HA: headache; ICH: intracranial hemorrhage; SAH: subarachnoid hemorrhage.

What time of day is your headache worse?

Complaints of waking up with a headache raise concern for hypertension, cluster, or neoplastic etiologies. Patients with tension headaches often awaken pain-free and develop their headache as the day progresses.

What were you doing when the pain began?

The activity or the event preceding the headache's onset often provides valuable clues to the etiology. A sudden onset of the "worst headache of my life" at rest or during any activity is highly suggestive of SAH. If the headache begins in association with motor vehicle exhaust, smoke from fires, engine fumes, or nonelectric heaters, carbon monoxide (CO) must be considered. The two most common symptoms of carbon monoxide poisoning are headache and dizziness. A headache that occurs during or immediately after sex may be a benign coital or post-coital headache, or the initial manifestation of life-threatening SAH. A patient may experience a severe but benign headache after coughing, sneezing, laughing, heavy lifting, stooping, or any Valsalva maneuver. The pain starts within a few seconds of the activity and may last for a few seconds to minutes. The sudden nature and severity of these headaches are similar to the sentinel headache of acute SAH. Headache associated with hunger, stress, sleep deprivation, menses, specific types of food ingestion, or oral contraceptive use suggests migraine. Any history of remote or recent head trauma prior to the headache must be ascertained.

What does the pain feel like?

The character of the pain is important, and may be useful in determining an etiology. Severe, intense, sudden onset, or "thunderclap" headaches may be the result of SAH. Pulsatile pain that correlates with the patient's pulse is usually vascular in origin. If the pain is pulsatile but does not correlate with the pulse, it is nonspecific. A dull, constant, band-like occipitofrontal headache is characteristic of tension headache. The intensity of pain, however, does not always correlate with the severity of underlying cause.

Where is your pain?

The location of pain may help narrow the diagnosis. Unilateral headaches are suggestive of migraines or a mass on the ipsilateral side. Unilateral facial pain is seen with trigeminal neuralgia, sinusitis and carotid artery dissection. Headaches that progress from unilateral to bilateral may be from increased ICP. Vertex headaches are seen with sphenoid sinusitis and supratentorial lesions. Orbital headaches suggest glaucoma, optic neuritis, cluster headache, or cavernous sinus thrombosis. Occipital headaches suggest cerebellar lesions, muscle spasm or cervical radiculopathy. However, an acute occipitocervical headache can be associated with intracranial pathology, especially when accompanied by other symptoms. Meningitis, encephalitis, SAH, and severe migraines are usually diffuse in their localization.

Does anything make the pain better or worse?

A headache that worsens with coughing, bending, or turning the head may be associated with a mass lesion or sinusitis. Post-dural headaches generally improve or disappear with supine positioning and worsen when the patient is upright. Pain that worsens with jaw movement suggests temporomandibular joint (TMJ) disease or temporal arteritis (jaw claudication).

What medications are you taking or have you changed any medications?

Headache is one of the most common side effects of prescribed medications. Medications that commonly cause headaches include nitroglycerine, hydralazine, calcium channel blockers, digitalis and estrogen. Patients who stop drinking coffee may develop a headache within 24–48 hours of abstinence. The headache typically resolves following ingestion of caffeine. Alcohol, marijuana and amphetamines may also induce headaches. A patient who uses cocaine may have a headache from an intracranial bleed.

Have you had the pain before?

Patients with migraine, cluster and tension headaches often have a history of similar headaches and symptoms. Migraine headaches typically start in childhood, adolescence, or young adulthood; patients often have a history of previous attacks. A significant change in intensity, location, or character from prior headaches may indicate serious new pathology, such as SAH. A recent severe headache may represent a *sentinel bleed* from a cerebral aneurysm. Patients with tension headaches may also have a previous history of similar headaches. Periodicity is the main feature of cluster headaches. An older patient complaining of a new-onset headache warrants suspicion. New headaches in patients over 50 years of age should raise concern for glaucoma, intracranial lesions, or temporal arteritis.

Associated signs and symptoms

Nausea and vomiting

Nausea and vomiting are commonly seen in patients with migraine headaches, SAH, meningitis, post-LP headaches, acute angle closure glaucoma (AACG), and increased ICP.

Photophobia

Photophobia can be due to irritation of the meninges from SAH or meningitis, and is a common complaint in patients with migraine headache. It can also arise from a pathologic problem with the eyes, such as iritis, uveitis, corneal abrasion, or AACG, although discomfort from these conditions is more likely due to light sensitivity.

Neck stiffness

Neck stiffness, particularly the inability to flex the neck or resistance to neck flexion, may be a sign of meningeal irritation from SAH or meningitis.

Fever

It is important to ask about fever, as the majority of patients with bacterial meningitis will have a fever on presentation to the ED. Fever is a nonspecific finding, but can be a secondary cause of a headache.

Other

Unilateral nasal congestion, tearing and conjunctival injection may be seen with cluster headaches. Cluster headaches are more common in young males than females or older patients, and can cause significant distress. An older patient with temporal arteritis will often complain of polymyalgia. Patients with migraine headaches may develop meningeal or visual findings, such as photophobia or scintillating scotoma. Visual field deficits, diplopia, seizures and syncope may be associated with SAH. In a patient who is pregnant, headache may be a sign of preeclampsia.

Past medical

A history of neurosurgery or malignancy (with potential for metastases) should raise concern for intracranial pathology, including obstructive hydrocephalus (from a malfunctioning indwelling shunt or mass lesion if one is present).

Family

A positive family history is present in 70% of patients with migraines. There is usually no family history with cluster headaches. A family history of SAH is a risk factor for SAH. There is a familial association of cerebral aneurysms with several diseases, including autosomal dominant polycystic disease, coarctation of the aorta, Marfan's syndrome, and Ehlers–Danlos syndrome type IV.

Physical examination

General appearance

Though a patient's general appearance is an important clinical observation that helps gauge the degree of distress, it may not differentiate life-threatening from benign conditions. A patient with a small SAH (sentinel bleed) may be comfortable, particularly if the aneurysm has not completely ruptured, as opposed to a patient with a migraine headache who may be in severe discomfort.

Vital signs

Vital signs can be abnormal in patients with headache. Tachycardia and tachypnea may be secondary to pain. An elevated blood pressure may be seen with SAH or increased ICP. An elevated temperature may indicate meningitis or encephalitis.

Head

Inspection and palpation of the head may reveal evidence of trauma. Tenderness in the area of the sinuses or teeth may be clues to the etiology; however, purulent nasal secretions and abnormal sinus transillumination are the best clinical predictors of sinusitis. The temporal artery should always be palpated in the elderly patient, as headache may be the only complaint with temporal arteritis. A tender area of the scalp that exactly reproduces the head pain may indicate neuralgia.

Eyes

A comprehensive examination of both eyes should be performed in all patients with a headache. The examination should assess visual acuity, pupil size, extraocular movements, and for evidence of photophobia. Patients with AACG may present with a headache. Signs of AACG include conjunctival injection, a mid-position dilated pupil, increased intraocular pressure, a steamy or cloudy cornea, a shallow anterior chamber, and decreased visual acuity. Evaluation of extraocular movements may reveal a CN VI deficit, which may represent a mass lesion or aneurysm. Funduscopic examination should also be performed to check for spontaneous venous pulsations (SVPs), subtle pulsations of the central retinal vein as it emerges from the optic disk. The absence of SVPs suggests (but is not specific for) papilledema, a sign of increased ICP. Subhyaloid or pre-retinal hemorrhages are seen with SAH, whereas retinal hemorrhages and exudates may be secondary to hypertensive encephalopathy.

Skin

Look for rash associated with meningococcemia (Figure 28.3), Rocky Mountain spotted fever (Figure 35.8), or vasculitis.

Neurologic

A complete neurologic examination should be performed. This includes assessment of mental status, cranial nerves, motor, sensory and cerebellar function, and deep tendon reflexes. Any change in mental status, personality, or fluctuation in level of consciousness suggests a potentially serious abnormality. Careful attention to the cranial nerves may reveal abnormal CN III or VI function, which may indicate an intracranial mass lesion, increased ICP, tonsillar herniation, or cerebral aneurysm. A headache associated with new focal neurologic deficits, seizures, or cognitive impairment mandates emergent neuroimaging.

Meningeal signs

Nuchal rigidity

A neck examination should be performed with the patient supine and relaxed, looking for involuntary resistance with passive flexion. Pain or resistance to flexion suggests meningeal inflammation, but may also be due to arthritis or neck injury.

Brudzinski's sign

While passively flexing the neck as described above, watch for flexion of the patient's hips and knees in response to this maneuver (Figure 30.1). Though the presence of Brudzinski's sign suggests meningeal inflammation, it is neither sensitive nor specific for meningitis.

Kernig's sign

Flex one of the patient's legs at the hip and knee, and then straighten at the knee. Pain or resistance to this action (Kernig's sign) suggests meningeal irritation (Figure 30.2), but is neither sensitive nor specific for meningitis.

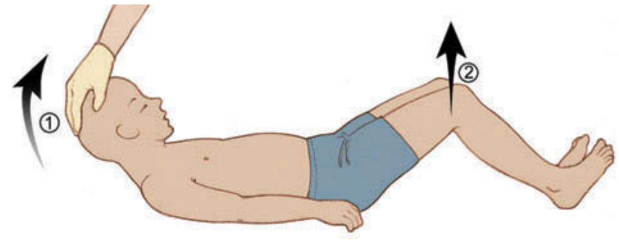

Figure 30.1
Brudzinski's sign. © Chris Gralapp.

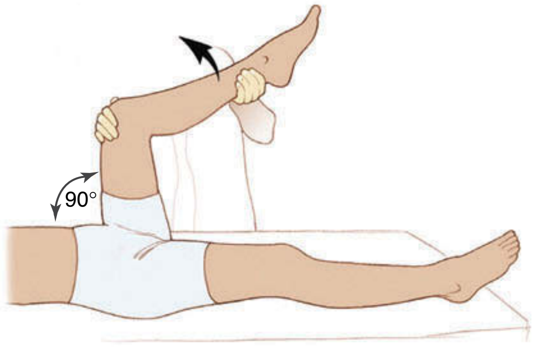

Figure 30.2
Kernig's sign. © Chris Gralapp.

Jolt accentuation test

This maneuver is performed by asking the patient to turn his or her head horizontally at a frequency of 2–3 rotations per second. Worsening of the baseline headache represents a positive sign. The jolt accentuation sign has a sensitivity of 97% and specificity of 60% for meningitis, but has only been studied in a small sample of patients.

Differential diagnosis

Tables 30.3 and 30.4 provide a list of primary and secondary causes of headache, with their symptoms signs, and work-up.

Table 30.3 Differential diagnosis of primary headache

Diagnosis	Symptoms	Signs	Work-up
Cluster headache	90% in males; no familial predisposition. Peak incidence in late 20s. Episodes occur in clusters which may last 6–8 weeks. Sharp unilateral pain, may awaken the patient from sleep. Alcohol and stress have been implicated as precipitants.	Classically unilateral nasal congestion, tearing and conjunctival injection.	No work-up necessary if the diagnosis is clear. CT of the brain may be needed to rule out CVA or hemorrhage.
Migraine	Gradual onset, often initially unilateral, severe, and associated with an aura. 40% of patients have bilateral HA. HA is throbbing and pulsatile, and associated nausea, vomiting, photophobia, phonophobia and scotomas. May be worse lying down.	Varies from mild to severe. In severe cases, patients may be prostrate to stuporous, with cold limbs and pale skin. Patients may have conjunctival injection and/or hypesthesia on affected side. Vein or artery over temple may be prominent.	No work-up necessary if the diagnosis is clear. CT of the brain may be needed to rule out CVA or hemorrhage.
Migraine variant	Uncommon. Hemiplegia is a type of aura that may include motor, sensory, or speech disturbances. Lasts >1 hour and <1 week. There is an autosomal inherited form.	As above.	No work-up necessary if the diagnosis is clear. CT of the brain may be needed to rule out CVA or hemorrhage.
Ophthalmoplegic migraine	Most common in children and adolescents. Transitory paresis of extraocular motor cranial nerves (CNs III, IV, VI). Occurs at the height of cephalgia but can persist for days to weeks. Paresis is unilateral and pain is periorbital and ipsilateral.	Paresis of extraocular muscles (CNs III, IV, VI)	No work-up necessary if the diagnosis is clear. CT of the brain may be needed to rule out CVA or hemorrhage. Prognosis for recovery is excellent. Need to rule out aneurysms or mass because of the neurologic findings.
Tension headache	Affects 75% of population, most commonly middle-aged individuals. Band-like discomfort around head that is non-pulsatile. Usually mild intensity and short duration. Anxiety and depression may coexist with chronic tension HA.	Physical examination is normal, but may find tender areas of the scalp or neck that can also be seen with migraine HA.	No work-up necessary if the diagnosis is clear. CT of the brain may be needed to rule out CVA or hemorrhage.

CN: cranial nerve; CT: computed tomography; CVA: cerebrovascular accident; HA: headache.

Table 30.4 Differential diagnosis of secondary headache

Diagnosis	Symptoms	Signs	Work-up
Acute angle closure glaucoma	Acute onset of severe HA around the affected eye. Pain may radiate to the forehead, ear, sinuses, or teeth. Patients may see halos around lights, experience blurriness or scotomas, and often complain of nausea and vomiting.	Red eye with a fixed, mid-dilated pupil, corneal clouding, and a shallow anterior chamber. IOP is >50 mmHg (normal <20 mmHg).	Slit lamp examination and tonometry are mandatory. Urgent ophthalmologic evaluation is required.
Bacterial meningitis	High fever, headache and stiff neck are common in patients over the age of 2 years. Symptoms can develop over hours to days. Other symptoms include nausea, vomiting, photophobia, confusion and sleepiness. In newborns and small infants, the classic symptoms of fever, headache and neck stiffness may be absent or difficult to detect.	Fever with photophobia and neck stiffness. Kernig's and Brudzinski's signs may be present. Focal neurologic deficits presenting in up to one-third of patients. Rash present in 22–26%. Altered sensorium may be present.	LP with CSF cultures. CT before LP is not required unless concern for space-occupying lesion.
Benign cough headache	Usually bilateral, lasting from seconds to minutes, following a paroxysm of intense coughing.	Signs depend on the etiology of the cough.	Work-up and treatment should be directed at the etiology of the cough.
Benign exertional headache	More frequent in males >40 years of age. May be precipitated by bending, lifting, sneezing, or defecating. Typically lasts a few seconds but can last for hours. It is bilateral but may be unilateral, severe and sudden in onset, having a bursting, explosive, or splitting quality.	Physical examination is normal.	History or physical examination do not distinguish this from other more serious etiologies. CT followed by an LP to rule out a life-threatening condition, particularly SAH.
Brain abscess	HA with fever; may also have vomiting.	Fever, focal neurologic findings and depressed level of consciousness.	CT, antibiotics and neurosurgical consultation
Carbon monoxide	Most common symptoms are headache, dizziness, weakness, nausea, vomiting, chest pain and confusion. High CO levels can cause loss of consciousness and death.	Signs are variable.	Must have a high index of suspicion. Diagnosis is made by co-oximetry or obtaining a CO level from a venous sample.
Carotid artery dissection	Unilateral HA may be severe and throbbing, but may be subacute and similar to previous headaches. Acute onset of severe retro-orbital pain in a patient without a history of cluster HAs is suggestive. Patients may also complain of visual disturbances, aphasia and hemiparesis.	May include ipsilateral Horner's syndrome and contralateral hemispheric findings (aphasia, neglect, visual disturbances and hemiparesis).	May be difficult to diagnose. Initial work-up includes a head CT, which may be unremarkable. Further imaging may include carotid artery duplex scanning, magnetic resonance angiography and/or carotid angiography.
Cavernous sinus thrombosis	Limitation of eye movement and facial pain. Fever, nausea, vomiting and altered level of consciousness often develop. Can be a complication of facial, periorbital, or orbital cellulitis; retrograde spread via the ophthalmic veins leads to cavernous sinus involvement.	Unilateral and then bilateral proptosis with paralysis of CNs III, IV, VI. Meningeal signs, dilation of the episcleral veins, venous engorgement of fundus, and pupillary dilation.	Blood cultures as well as culture of a draining cellulitis, if present. CT of the brain warranted. Ophthalmologic and neurosurgical consultation.
Cervicogenic headache	Unilateral and occurs with movements of the head or neck. Can occur from a trigger point in the neck, typically unilateral, that spreads to the ipsilateral shoulder or arm. Often associated with whiplash injury.	Often can elicit a trigger point in the neck or shoulders on careful examination.	Supportive care
Coital or post-coital headache	Begins as a dull bilateral ache as sexual excitement increases, becoming intense at orgasm. Many patients never seek medical attention. Four times more common in males. Risk factors include obesity, hypertension, fatigue, migraine and peripheral vascular disease.	Physical examination is normal.	Often impossible to distinguish from SAH; therefore, work-up for SAH may be necessary.
Encephalitis	HA severity is variable. May present with fever, confusion and seizures.	Altered sensorium and focal neurologic findings.	CT and LP with viral cultures

(*continued*)

Table 30.4 Differential diagnosis of secondary headache (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Epidural hematoma	Classically, a history of head trauma with LOC, followed by a return to normal mental status. As the hematoma enlarges, the patient complains of diffuse HA, and may develop hemiplegia and obtundation. This so-called "lucid interval" is neither sensitive nor specific.	Signs vary from an unremarkable examination to signs of increased ICP (hemiplegia, pupillary dilation and obtundation) as herniation approaches.	CT and neurosurgical consultation
Hypertensive headache	Not common. The rate of rise of the blood pressure is more important than the absolute value. Pain is usually diffuse and occurs in the morning upon awakening; generally improves as the day progresses.	Diastolic BP <130 mmHg is rarely the cause. With hypertensive encephalopathy, the BP is around 250/150 mmHg.	Work-up to exclude end-organ damage. Consider drug-induced hypertension, eclampsia and pheochromocytoma as alternate etiologies of HA and elevated BP.
Idiopathic intracranial hypertension	Formerly known as *pseudotumor cerebri* or *benign intracranial hypertension*. Relatively common in young obese women of childbearing age. Predisposing factors include oral contraceptives, anabolic steroids, tetracyclines and vitamin A. Visual complaints are common and can become permanent in up to 10%.	Examination may reveal papilledema and visual field defects. No localizing signs on neurologic examination.	Diagnosis confirmed with neuroimaging and ICP measurement. An opening pressure >200 mmH_2O on LP and concomitant absence of mass lesions or ventricular enlargement on neuroimaging.
Monosodium glutamate (MSG) headache	Chinese restaurant syndrome occurs 20 minutes after ingestion of MSG. HA is throbbing, bifrontal or bitemporal. Often pressure or tightness across the face and chest. May have dizziness, flushing, nausea and abdominal discomfort.	May see flushing of the skin.	No work-up needed; treatment is symptomatic.
Non-hemorrhagic stroke	HA can be seen with both ischemic and hemorrhagic strokes, and may precede, accompany, or follow the event. Presence of an HA correlates with a large artery thrombotic stroke (less with embolic and least common with lacunar strokes). HA is also more common in cortical infarcts compared with deeper lesions.	Cognitive impairment may mask the HA. Neurologic deficits may include slurred speech, cerebellar findings and hemiparesis.	CT is the study of choice in the initial evaluation of suspected stroke. Although CT may not reveal stroke in the first 24 hours, it can exclude hemorrhage prior to thrombolytics. MRI may also be helpful.
Post-concussive headache	Occurs after mild to moderate head trauma, hours to days after the incident. The syndrome is characterized by dizziness, fatigue and insomnia. Symptoms resolve spontaneously after several weeks.	Physical examination is normal.	If symptoms or HA persist, CT, MRI and neuropsychiatric testing may be warranted.
Post-dural puncture headache	Usually frontal or occipital headache within 48 hours of LP. May be associated with nausea, vomiting, dizziness, or tinnitus. The HA results from a persistent CSF leak with subsequent traction on intracranial pain-sensitive structures. Occurs following 10–30% of LPs.	Symptoms improve or disappear with recumbence.	Diagnosis is usually clear and no work-up is necessary.
Subarachnoid hemorrhage	Sudden onset of the "worst headache" of the patient's life. LOC may occur. History of an unusual HA weeks earlier (sentinel bleed) is seen in one-third of patients.	Nausea and vomiting, impaired speech, photophobia, neck stiffness and seizures. 75% of patients have a normal mental status, >50% will have ECG changes. Up to 20% will have focal neurologic findings; almost 10% will have abnormal CN III function.	CT of the brain; if negative, then LP is mandatory. If CT or LP positive, then urgent neurosurgical consultation and cerebral angiogram.
Sinus headache	HA often described as deep, dull, or heavy, made worse by shaking the head or bending forward. Often accompanied by fever and purulent nasal discharge. May have dental pain.	Tenderness over sinuses and purulent drainage may be present.	CBC and blood cultures depending on how ill the patient appears. CT of sinuses is the gold standard if imaging is warranted.

(*continued*)

Table 30.4 Differential diagnosis of secondary headache (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Subdural hematoma	Symptoms develop more slowly than with epidural bleeds. Many patients present with head trauma and HA. Symptoms range from mild HA to obtundation. Patients, particularly the elderly, may present several weeks after the initial injury with HA and personality changes.	Findings may be subtle. If there is continued bleeding, mass effect and signs of increased ICP may develop.	CT and neurosurgical consultation.
Temporal arteritis	Also known as *giant cell arteritis*. Females are more commonly affected, and the age of onset is typically the early 70s (but can occur earlier). HA can be continuous or intermittent, and tends to be worse at night or in the cold. Jaw claudication may occur. Visual loss may occur if untreated.	Physical examination may reveal tenderness and induration of the scalp arteries.	Significantly increased ESR. Diagnosis is confirmed with arterial biopsy.
Trigeminal neuralgia	Painful, unilateral, brief, shock-like pains in the distribution of one or more branches of the trigeminal nerve. Episodes last from seconds to minutes, may occur spontaneously or be invoked by talking, chewing, shaving, or even brushing teeth.	May see unilateral grimaces during episodes.	Careful neurologic examination with particular attention to the trigeminal nerve distribution. CT or MRI to rule out a mass, especially if sensory or motor dysfunction is present.
Tumor headache	HA is the most common symptom occurring in >50% of patients. Mostly metastases from lung and breast. Patients tend to be older. Pain patterns are variable depending on size and location of the mass. HA tends to be worse in the morning and may be associated with vomiting.	The neurologic examination depends on the location of the tumor.	Neuroimaging is needed to identify the size and location of the mass. Neurosurgical referral must be made.
Vertebral artery dissection	Severe unilateral posterior HA with vertigo, vomiting, ataxia, diplopia, hemiparesis, unilateral facial weakness and tinnitus.	Hemiparesis, ataxia, and signs of brainstem or cerebellar ischemia.	Same work-up as carotid artery dissection.
Viral meningitis	HA may be severe but more indolent than in bacterial meningitis.	Same as with bacterial meningitis but generally have a normal mental status.	Same work-up as bacterial meningitis. Antibiotics are not required but are often administered pending culture results.

BP: blood pressure; CBC: complete blood count; CN: cranial nerve; CO: carbon monoxide; CSF: cerebrospinal fluid; CT: computed tomography; CVA: cerebrovascular accident; ECG: electrocardiogram; ESR: erythrocyte sedimentation rate; HA: headache; HIV: human immunodeficiency virus; ICP: intracranial pressure; IOP: intraocular pressure; LOC: loss of consciousness; LP: lumbar puncture; MRI: magnetic resonance imaging; SAH: subarachnoid hemorrhage.

Diagnostic testing

Radiologic studies

If the diagnosis of migraine, cluster, tension, or post-dural puncture headache can be made from the history and physical examination, imaging studies are typically unnecessary. If the diagnosis is unclear, computed tomography (CT) of the head or magnetic resonance imaging (MRI) of the brain should be performed to delineate lesions that may be responsible for the headache. CT is preferred for the detection of acute intracranial hemorrhage, increased ICP and hydrocephalus. In a patient with a headache and human immunodeficiency virus (HIV), CT with contrast may reveal a ring-enhancing lesion suggestive of toxoplasmosis (Figure 30.3). CT can also identify approximately 90% of patients with SAH, and is best when performed in the first 24 hours following headache onset (Figure 30.4).

MRI is generally not required in the initial work-up of patients with headache; however, it is preferred for

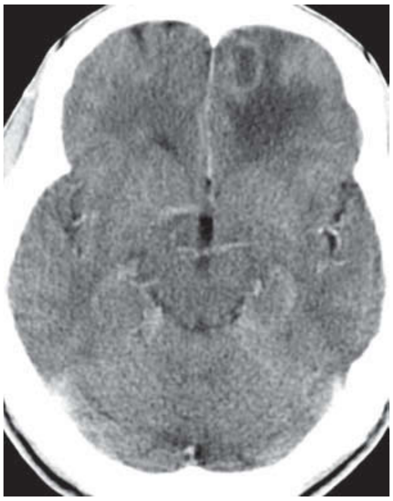

Figure 30.3
Cerebral toxoplasmosis. Head CT post-contrast enhancement demonstrates ring-enhancing lesion of left frontal lobe with vasogenic edema. Courtesy: Mahesh Jayaraman, MD.

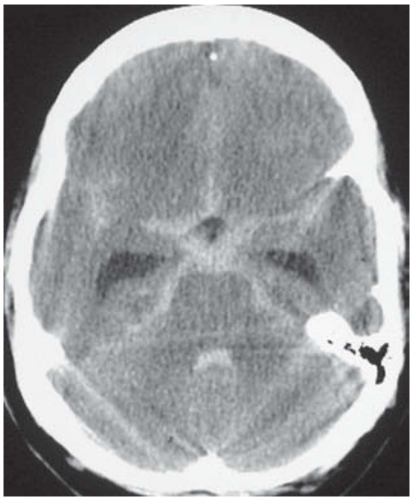

Figure 30.4
Subarachnoid hemorrhage. Non-enhanced CT of the head reveals diffuse subarachnoid hemorrhage, intraventricular hemorrhage and hydrocephalus. Courtesy: Mahesh Jayaraman, MD.

certain clinical situations. MRI has a higher sensitivity than CT for the detection of SAH after 24 hours and for identification of non-hemorrhagic strokes in the first 24–48 hours. MRI is also superior to CT for visualizing the posterior fossa, localizing tumors and focal infections, and identifying cavernous sinus thrombosis and carotid artery dissection.

Laboratory studies

Laboratory testing for headaches should be determined by the clinical scenario. If an infection is suspected, blood cultures and a complete blood count (CBC) may be required; however, patients with meningitis may have a normal white blood cell count. The erythrocyte sedimentation rate (ESR) is elevated in patients with temporal arteritis; physicians should consider this test in older patients with headache. A CO level should be obtained when there is suspicion of CO poisoning; pulse oximetry or an arterial blood gas (ABG) alone cannot rule out CO poisoning. Serum chemistries should be performed when there is concern for electrolyte abnormalities, such as in patients with profuse vomiting, prolonged diarrhea, diabetes mellitus, or seizures.

CSF analysis should be performed when there is suspicion of meningitis or encephalitis, or when SAH is suspected and CT does not demonstrate subarachnoid blood. When SAH is likely, the CSF analysis should include a cell count from the first and last tube collected. This will distinguish a traumatic LP from SAH. If the number of red blood cells (RBCs) does not significantly decrease from the first to the last tube, consider SAH. (Figure 30.5). The CSF should also be examined for xanthochromia, a yellowish discoloration caused by pigment released from the breakdown of RBCs in the CSF (Figure 30.6). This may be done visually, but is more accurate with a spectrophotometer. The presence of xanthochromia also raises concern for SAH. When performing an LP, opening pressure should be measured. An opening pressure >200 mmH_2O is consistent with idiopathic intracranial hypertension and other conditions that may have an increased ICP, such as meningitis.

General treatment principles

All patients presenting to the ED with headache should be evaluated in a timely fashion and receive symptomatic relief; some will require a comprehensive work-up. Treatment with analgesics can be initiated while diagnostic studies are performed. The patient should be placed in a quiet, dark area of the ED to avoid exacerbating symptoms.

After performing a physical examination, if the patient can tolerate oral intake and is not a surgical candidate, the

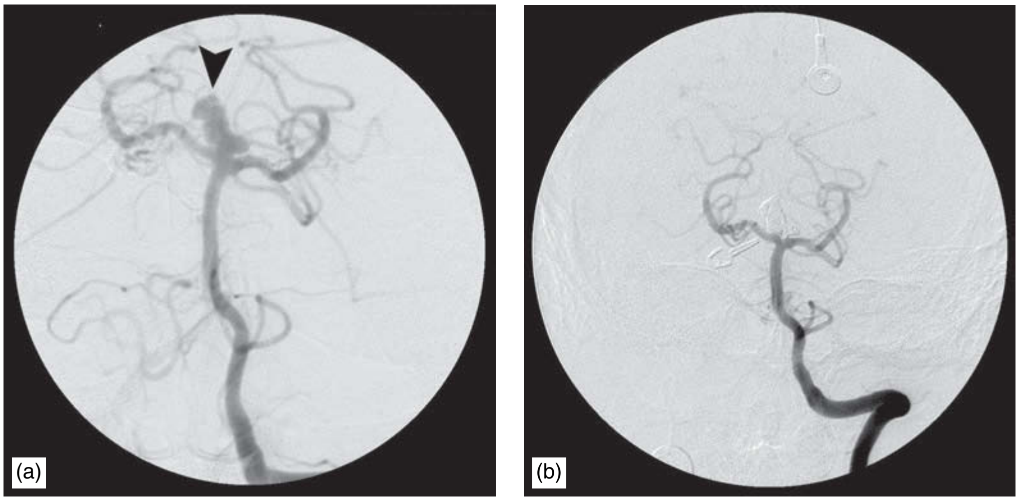

Figure 30.5
Basilar tip aneurysm. (a) Frontal projection from a left vertebral artery angiogram demonstrates a basilar tip aneurysm (black arrowhead) with a relatively narrow neck. (b) Frontal projection angiogram following endovascular coil embolization demonstrates no signification residual filling of the aneurysm. Courtesy: Mahesh Jayaraman, MD.

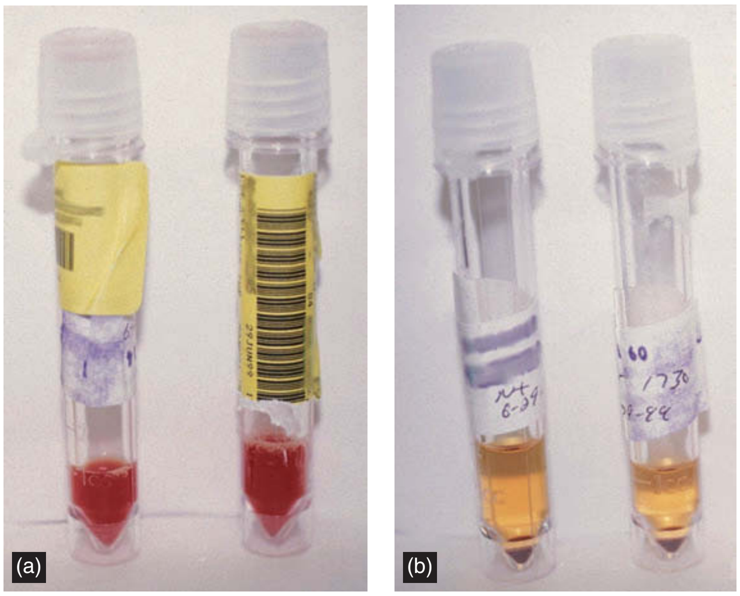

Figure 30.6
CSF tubes demonstrating: (a) bloody CSF representing subarachnoid hemorrhage (SAH); (b) xanthochromia in CSF fluid (note the golden yellow-brown appearance of the translucent CSF). Courtesy: Gus M. Garmel, MD.

patient may be given oral analgesics. If oral intake cannot be tolerated, intravenous (IV) access should be established for hydration and administration of analgesics and antiemetics.

Many secondary headaches, such as those resulting from cold stimulus, alcohol, exertion, cough, hypertension, monosodium glutamate (MSG), or side effects of medications, improve with treatment of the primary disorder or avoidance of the precipitating event or medication. Symptomatic treatment with acetaminophen may be beneficial.

Migraines

For mild to moderate attacks, acetaminophen, aspirin, or nonsteroidal antiinflammatory drugs (NSAIDs) are recommended. For moderate to severe attacks, intramuscular or subcutaneous dihydroergotamine or sumatriptan are excellent options if the patient cannot tolerate oral intake. IV prochlorperazine, chlorpromazine, or metoclopramide are also commonly used. Parenteral ketorolac, morphine or hydromorphone may be of value as well. Refractory attacks (status migrainosus) are best treated with parenteral dihydroergotamine and IV steroids (Table 30.5).

Cluster headaches

High-flow oxygen followed by subcutaneous sumatriptan are preferred treatments for this painful condition. Dihydroergotamine parenterally has also been shown effective. Intranasal application of lidocaine to produce anesthesia of the sphenopalatine region has been advocated but not proven effective. Steroids have also been used.

Tension and cervicogenic headaches

Treatment is symptomatic with analgesics, ice, relaxation techniques and trigger point injections.

Subarachnoid hemorrhage

The initial management of SAH includes resuscitation, stabilization and emergent neurosurgical consultation. The main goals are to prevent recurrent bleeding, prevent further increase in ICP, mitigate the effects of vasospasm, and treat acute medical and neurologic complications. Continued or recurrent bleeding is worsened by uncontrolled blood pressure elevations. To reduce the likelihood of an ischemic stroke, nimodipine should be started soon after the diagnosis of SAH is made. The recommended dose is 60 mg every 4 hours by mouth or nasogastric tube. Blood pressure must be monitored closely to prevent hypotension. Patients with a Hunt and Hess classification grade III or higher SAH are at risk for respiratory depression, which can lead to hypercapnia and worsening ICP (Table 30.6). These patients may require early endotracheal intubation with precautions to prevent ICP elevation. Analgesics should be used and NSAIDs, including aspirin, must be avoided because of their antiplatelet effects. Antiemetics may be required. Agents that lower the seizure threshold should be avoided, since these patients are at an increased risk for seizures. Prophylactic anticonvulsant therapy is controversial and should be instituted in consultation with neurosurgery. Most of these patients require monitoring in an intensive care setting. Elevating the head of the bed may help prevent further increases in ICP. The use of therapeutic cooling in SAH is currently being investigated.

Table 30.5 Pharmacologic options for the treatment of acute migraines and related headaches

Drug	Indications	Dose and route	Comments
NSAIDs			All contraindicated if allergy to aspirin or NSAIDs
Ibuprofen	Mild/moderate headache	600–800 mg PO	GI upset
Naproxen sodium	Mild/moderate headache	275–550 mg PO	GI upset
Indomethacin	Mild/moderate headache	25–50 mg PO or 50 mg PR	GI upset
Ketorolac	Moderate/severe headache	30 mg IV or 30–60 mg IM	GI upset and bleeding; avoid in elderly patients (or use lower dose) and those with renal insufficiency.
Serotonin agonists			
Dihydroergotamine	Moderate/severe migraine, cluster	1 mg IV or IM; may be repeated in 1 hour	GI upset (pretreat with antiemetic). Cannot be used if sumatriptan recently taken. Contraindicated with hypertension, coronary artery disease, peripheral vascular disease and pregnancy.
Sumatriptan	Moderate/severe migraine, cluster	6 mg SQ; may be repeated once in 1 hour if partial response	Chest pain, throat tightness, flushing. Contraindicated with hypertension, coronary artery disease, peripheral vascular disease and pregnancy. Cannot be used within 24 hours of ergotamine use.
Dopamine antagonists			
Prochlorperazine	Moderate/severe migraine	10 mg IV or IM; may be repeated in 30–60 minutes	Sedation and dystonic reaction
Chlorpromazine	Moderate/severe migraine	7.5 mg IV	Significant orthostatic hypotension; therefore, saline bolus should be administrated prior to use of this medication. Sedation and dystonic reaction.
Metoclopramide	Moderate/severe migraine	10 mg IV	Dystonic reaction
Steroids			
Dexamethasone	Status migrainosus, intractable episodic cluster	10–20 mg IV	Interval medication until other agents take effect. GI bleeding, infection, cataracts, aseptic necrosis, memory disturbances.
Opioids			
Hydromorphone		1–4 mg IM or IV	Last resort, most of the other regimens provide superior pain relief.
Morphine sulfate		1–10 mg IM or IV	Last resort, most of the other regimens provide superior pain relief.

GI: gastrointestinal; IM: intramuscular; IV: intravenous; NSAIDs: nonsteroidal antiinflammatory drugs; PO: per os; PR: per rectum; SQ: subcutaneous.

Table 30.6 Hunt and Hess clinical grading scale for subarachnoid hemorrhage

Grade	Condition
0	Unruptured aneurysm
1	Asymptomatic or minimal headache and nuchal rigidity
2	Moderate or severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
3	Drowsiness, confusion, or mild focal deficit
4	Stupor, moderate or severe hemiparesis
5	Deep coma, decerebrate posturing, moribund appearance

Epidural hematoma

Patients with epidural hematomas require emergent neurosurgical evaluation and intervention, as they can deteriorate rapidly. Definitive treatment is surgical evacuation of the hematoma and coagulation of bleeding sites.

Subdural hematoma

Patients with a subdural hematoma also require emergent neurosurgical evaluation. Depending on the size, location and duration of the hematoma; the resulting symptoms; and the preoperative condition of the patient, a neurosurgeon may opt to observe the patient rather than perform an immediate operation.

Giant cell arteritis

Giant cell arteritis must be treated promptly once the diagnosis is suspected due to the risk of permanent visual loss. Steroids are the mainstay of therapy. The recommended initial oral dose of prednisone is 60–120 mg/day. Therapy needs to be continued for months, although symptomatic response may be seen within several days. The ESR needs to be closely monitored. A definitive diagnosis requires a temporal artery biopsy, which should be performed within 1 week of initiating steroid therapy. A positive biopsy result may be seen up to 1 month later.

Idiopathic intracranial hypertension

Treatment involves lowering the ICP as well as symptomatic treatment of the headache. Acetazolamide decreases CSF production and lowers the ICP; furosemide may be helpful. Steroids have also been used. Repeated LPs are sometimes needed to remove CSF, but are generally not well tolerated and are difficult in obese patients. In patients with impending visual loss, a ventricular shunt may be indicated.

Tumor headache

Urgent neurosurgical consultation is required once this diagnosis is made. Treatment includes managing increased ICP and seizures. ICP elevation from a brain tumor is initially managed with high-potency steroids (like dexamethasone) to mitigate tumor-associated edema. The starting dose is 10 mg IV followed by 4 mg every 6 hours. Seizures are managed with agents such as phenytoin, valproic acid, or carbamazepine.

Meningitis

The specific treatment of the varied types of meningitis is beyond the scope of this chapter. Empiric treatment with broad-spectrum antibiotics pending cultures is prudent. IV steroids are now recommended prior to antibiotics for patients with bacterial etiologies.

Acute angle closure glaucoma

Urgent ophthalmologic consultation is required once the diagnosis of AACG is made. The initial treatment involves lowering intraocular pressure with topical beta-blockers (such as timolol maleate) and oral or IV acetazolamide. Miotics (such as pilocarpine) are used to constrict the pupil. Definitive management requires an ophthalmologist.

Trigeminal neuralgia

Carbamazepine, phenytoin and baclofen are effective in the treatment of trigeminal neuralgia. However, all of these therapies have high failure rates, and surgical management may be required.

Post-dural puncture headache

Once identified by history, the patient should be treated with hydration, bed rest and analgesics. Oral or IV caffeine can produce symptomatic relief (i.e., 500 mg of caffeine sodium benzoate in 1 liter of normal saline infused over 1 hour). When conservative methods have failed, an epidural blood patch performed by an anesthesiologist can be highly effective.

Carbon monoxide

Initial treatment is with 100% oxygen. Pregnancy, coma and significant comorbidities require aggressive measures, such as hyperbaric oxygen (HBO) therapy. Many experts advocate the use of HBO therapy based on CO levels or specific symptoms.

Carotid and vertebral artery dissection

Treatment is aimed at stroke prevention from embolization, and includes early anticoagulation followed by antiplatelet therapy.

Cavernous sinus thrombosis

Treatment requires high-dose IV antibiotics that cover staphylococcus, streptococcus and *Haemophilus influenzae*. In addition, neurosurgical, ophthalmologic, or otolaryngologic consultation are important and should be obtained in a timely fashion.

Special patients

Elderly

Older patients with headache need special consideration. Chronic disease and normal physiologic changes with aging contribute to atypical presentations in the elderly. Elderly patients can present with a mild headache or a history of minor trauma, yet have a subacute or chronic subdural hematoma. Up to 20% of patients with a chronic subdural hematoma have no identifiable precipitant. In fact, many older patients may present with symptoms up to 3 months following a known traumatic event. Certain diseases, such as temporal arteritis, present later in life. It is rare for an elderly patient to develop a new migraine headache. A thorough work-up including a low threshold for CT is therefore required for any elderly patient presenting with a headache.

Immune compromised

Immunocompromised patients can suffer from any of the headaches previously described, but require special consideration because of their immune status. This group of patients is at risk for opportunistic central nervous system (CNS) infections including mycobacteria, spirochetes, viruses (herpes simplex, herpes zoster, cytomegalovirus and HIV), fungi (*Cryptococcus*), protozoa (toxoplasma) and actinomycetes. These patients are also at risk of CNS lymphoma and Kaposi's sarcoma. Noncontrast and contrast CT must be performed on any immunocompromised patient with headache to rule out a space-occupying lesion, especially if an LP is being considered.

Pediatric

Headache is common in children and adolescents. Forty percent of children will experience a headache by the age of 7 years; 75% will experience a headache by the time they are 15 years old. Migraine is one of the most common causes of headache in childhood, occurring in 1% of children by the age of 7 and 5% of children by 15 years of age. An acute headache may accompany many infections. Nonspecific viral illnesses represent the most common diagnoses in children presenting with headaches. Care must be taken to exclude serious illnesses such as meningitis, hypertension, or SAH. Pediatric patients with meningitis usually present with a history of headache accompanied by other systemic symptoms, such as changes in behavior, lethargy, seizures and shock. Patients with mildly or severely elevated blood pressure may present with headache, and require rapid assessment to determine the etiology of their elevated blood pressure. Pediatric patients with SAH present similarly to adult patients with severe headache; they usually have neurologic abnormalities or a change in consciousness.

Pregnant

The most common type of headache occurring during pregnancy is a muscle contraction headache. Most patients have a previous history of headaches prior to the pregnancy. Eighty percent of women with classic migraine headaches have remission from attacks during pregnancy; thus nearly any headache other than a muscle contraction headache requires investigation.

Brain tumors enlarge during pregnancy and shrink temporarily postpartum. Most tumors become symptomatic during the second half of pregnancy. Brain tumors account for 10% of maternal deaths during pregnancy. Idiopathic intracranial hypertension (pseudotumor cerebri) associated with pregnancy usually occurs in obese women, and begins between the third and fifth months of pregnancy. Symptoms usually resolve spontaneously after 1–3 months.

Spontaneous SAH accounts for 10% of maternal deaths in pregnancy. The most common cause of SAH in women younger than 25 years is a berry aneurysm. For women older than 25 years, the most common etiology is an arteriovenous malformation. Nearly one-third of spontaneous SAHs are the result of bleeding disorders, bacterial endocarditis, metastatic tumors, or sickle cell disease.

Patients with eclampsia present with the sudden onset of focal neurologic deficits in addition to headache, seizure, or altered consciousness. The most common cause of death in patients with eclampsia is cerebral hemorrhage, occurring in 60% of patients who die after developing eclampsia.

Disposition

The majority of ED patients presenting with a headache can be managed as outpatients. Specific treatment modalities are discussed in Table 30.5. Table 30.7 lists indications for hospitalizing patients with headache.

Table 30.7 Indications for hospitalizing patients with headache

Central nervous system infections
Vascular diseases
Subarachnoid hemorrhage
Cerebral ischemia
Severe hypertension
Spontaneous carotid artery dissection
Temporal arteritis
Space-occupying lesions
Toxic/metabolic encephalopathy
Idiopathic intracranial hypertension not responding to usual measures
Headache associated with severe medical illness
Severe intractable headache
Continuous vomiting, electrolyte abnormalities, or inability to maintain oral hydration
Complex drug interactions
Social situations preventing appropriate outpatient management

Pearls, pitfalls and myths

- SAH should be considered in any patient who presents with the first or worst headache of their life. However, all patients will have both a first *and* worst headache at some time in their lives.
- SAH and meningitis must be considered in patients presenting with a change in the character, location, or intensity of their headache.
- A thorough examination of both eyes should always be performed on patients with headache, as their headache may be due to AACG. Identifying this condition may prevent unnecessary testing and delays in treatment.
- The typical patient with idiopathic intracranial hypertension is young, female, overweight, and has acne. Funduscopic examination typically reveals papilledema. A normal head CT and an elevated opening pressure on LP confirm the diagnosis.
- CO poisoning should be considered in a patient presenting with headache and nausea, especially if other family members have similar symptoms. Cars and faulty room heaters are often responsible.
- Giant cell arteritis should always be considered in elderly patients presenting with a headache, especially if it is unilateral.
- Current CT scanners are not 100% sensitive for the detection of SAH. A negative CT must be followed by a negative LP for exclusion of this diagnosis.
- Be cautious of making the diagnosis “new-onset migraine” in an elderly patient with a first headache.

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31 Hypertensive urgencies and emergencies

Robert Galli, MD **and Loretta Jackson-Williams,** MD

Scope of the problem

Hypertension (HTN) affects more than 20% of the adult and 3% of the pediatric populations. As the population ages, the prevalence of HTN is increasing. HTN is a process that contributes to the development of cardiovascular and renal diseases; it appears to be a polygenic, multifactorial disorder, the result of several genes interacting with environmental factors. HTN is defined by a systolic blood pressure (SBP) >140 mmHg, diastolic blood pressure (DBP) >90 mmHg, or the requirement of antihypertensive medications for control of sustained blood pressure (BP) elevations.

As a disease process, HTN was born out of epidemiologic studies that showed that chronic BP elevation decreased life expectancy; that treatment of HTN reduces stroke, coronary artery disease (CAD) and heart failure; and that most hypertensive patients require more than one agent to achieve BP control. HTN is an asymptomatic disease process with the exception of hypertensive emergency. Hypertensive emergencies and urgencies, also known as *hypertensive crises*, can cause end-organ dysfunction and require controlled yet time-sensitive management. These hypertensive crises can be viewed as a continuum of the disease process in some patients.

A *hypertensive emergency*, also known as malignant HTN, is defined as an acute elevation in BP (generally DBP >130 mmHg) with end-organ dysfunction or damage. It requires prompt parenteral treatment, with a goal of 25% reduction in mean arterial pressure (MAP) within 30–60 minutes. A *hypertensive urgency* is defined as moderately severe to severe HTN (DBP 120–140 mmHg) without presenting signs or symptoms of end-organ dysfunction or a concomitant emergency medical condition. However, these patients are at risk for imminent end-organ damage. Hypertensive urgencies require controlled reduction of BP over several days, which can generally be accomplished in the outpatient setting.

Anatomic essentials

The primary event for initiating any hypertensive crisis is an increase of arterial pressure. The triggers for this rapid rise in pressure are unknown. They likely include a combination of cellular, organ and environmental systems. It is clear that the rate of rise in arterial pressure is more important than the absolute level of pressure. Without prompt control of arterial pressure, fibrinoid necrosis of small arterioles occurs with resultant ischemia and infarction of end organs. This is followed by end-organ dysfunction and damage.

The process of fibrinoid necrosis of small arterioles begins when vessels in the capillary beds dilate to accommodate the sustained elevation in BP. Presumably, the shear force causes damage to the endothelium with resultant vascular wall injury. Response to this injury is the activation of coagulation and cell proliferation mediators. Fibrin is deposited within the damaged vascular walls. The process is continuously repeated and results in progressive narrowing and stiffening of the arterioles.

Flow through the vessels in the capillary beds is autoregulated to ensure that end organs are adequately perfused. This autoregulation occurs within specific ranges of BP in end organs. Beyond these specific ranges, autoregulation of blood flow does not occur, with ensuing hypo- and hyperperfusion of end organs. In patients with sustained elevations of BP, the specific range of autoregulation is raised. Therefore, acutely lowering the BP in hypertensive patients can result in hypoperfusion of end organs, because autoregulation of blood flow has been disrupted.

Changes in blood flow through end organs have clinical relevance. Intracerebral hemorrhage and hypertensive encephalopathy can occur from hyperperfusion of the brain. Stroke can be caused by hyper- or hypoperfusion. Angina, myocardial infarction and acute left ventricular dysfunction with resultant pulmonary edema can occur as a result of hypoperfusion through the coronary arteries. In the kidneys, renal impairment and failure result from hypoperfusion. Aortic dissection may result from hyperperfusion and its resultant shear forces.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 31.1).

History

The history of a patient with elevated BP in the emergency department (ED) should rapidly determine whether end-organ dysfunction or concomitant emergency medical conditions are present. Primary areas of focus include the neurologic, cardiovascular, pulmonary and renal systems.

What were the onset and duration of the presenting symptoms?

This information will help determine appropriate disposition and referral. Patients with longstanding HTN

Table 31.1 Hypertensive urgencies and emergencies red flags

History	Concerning diagnosis
Headache with CNS or neurologic symptoms	Stroke or CNS hemorrhage
Headache with neck pain	Carotid or vertebrobasilar artery dissection, cerebellar infarction or hemorrhage
Chest pain, shortness of breath, back pain (especially if acute, tearing in quality, or worse than usual)	Acute coronary syndrome, MI, thoracic aortic dissection, congestive heart failure
Syncope	Stroke, intracranial or cerebral hemorrhage, CNS hypoperfusion
Visual changes	Stroke, CNS event, optic nerve or retinal ischemia
Pregnancy (third trimester or recently postpartum)	Preeclampsia or eclampsia
Seizures	Toxic ingestion, amphetamine or cocaine use, intracranial or cerebral hemorrhage or tumor, eclampsia
Antihypertensive medications	Refractory or rebound hypertension
Examination finding	**Concerning diagnosis**
Focal neurologic findings	Stroke, intracranial or cerebral hemorrhage, brain tumor hemorrhage, CNS ischemia, preeclampsia or eclampsia (if pregnant or recently postpartum)
Retinal hemorrhages, nystagmus, absent spontaneous venous pulsations	CNS lesion, stroke, intracranial hemorrhage, retinal hemorrhages or ischemia, increased intracranial pressure
Pronounced PMI, ECG findings	Acute coronary syndrome, MI
Rales, rhonchi, dyspnea, air hunger, tachypnea, abnormal cardiac rhythm (atrial fibrillation, S3 gallop), murmur (particularly if new or diastolic)	Congestive heart failure, valvular emergency (including rupture)
Asymmetric extremity pulses or blood pressures, severe pain (especially if sudden onset, sharp and tearing in quality)	Thoracic aortic dissection
Gravid uterus, breast engorgement, lactation	Preeclampsia or eclampsia
Seizure activity	CNS insult, including hemorrhage or stroke, eclampsia
Ataxia, syncope	Cerebellar ischemia or hemorrhage

ECG: electrocardiogram; CNS: central nervous system; MI: myocardial infarction; PMI: point of maximal impulse.

may not present with evidence of malignant HTN because of autoregulatory mechanisms previously discussed. Hypertensive emergencies typically present with acute onset and rapid progression within minutes to hours.

Did the patient consume any unusual foods or beverages prior to the onset of symptoms?

Many foods and beverages (i.e., beer, wine, coffee) contain tyramine which can raise BP due to the release of norepinephrine. Elevations of BP may be especially prolonged or profound if a patient is taking a monoamine oxidase inhibitor (MAOI).

Does the patient have a severe headache?

In general, headache in hypertensive patients is a nonspecific sign and is not a reliable indicator of elevated BP. Sudden onset headache is concerning for subarachnoid hemorrhage. Severe headache and altered mental status may represent hypertensive encephalopathy or intracranial hemorrhage.

Does the patient have speech or gait abnormalities, focal sensory or motor deficits, or mental status changes?

These questions address the neurologic history relating to specific stroke syndromes. Focal neurologic findings are uncommon in hypertensive encephalopathy, and are more suggestive of a cerebrovascular accident.

Does the patient have visual disturbances or loss of vision?

Decreased visual acuity or the presence of visual field defects may indicate the development of retinopathy. In addition, amaurosis fugax or painless monocular blindness identifies a stroke involving the internal carotid artery and the anterior circulation of the brain.

Does the patient complain of dyspnea or chest pain?

Acute left heart failure will result in dyspnea or the sensation of breathlessness, especially with exertion. Patients with right heart failure may complain of peripheral edema. Acute coronary syndromes can be precipitated by severe elevations in BP. Acute aortic dissection should be suspected in all patients with hypertension who present with sudden onset chest pain, especially of tearing quality or radiating to the back.

Does the patient complain of abdominal or back pain?

Patients with these complaints should be assessed for an aortic dissection or abdominal aortic aneurysm.

Does the patient complain of diminished urine output or discolored urine?

Severe renal damage may result in oliguria, hematuria, or proteinuria.

Is the patient pregnant or recently postpartum?

Because of profound changes in circulatory physiology during pregnancy, hypertensive events may occur at lower-than-expected BPs. These patients should be assessed for preeclampsia. Recently postpartum patients may have preeclampsia or eclampsia.

Past medical

In general, historical information should include the patient's age, known prior diagnosis of HTN, cardiovascular or renal diseases, present or past antihypertensive medications, compliance history with those medications, and known concomitant medical conditions, such as diabetes or vasculitis. The patient should be specifically asked about the use of drugs, such as MAOIs, cocaine, amphetamines and alcohol. Alcohol withdrawal may precipitate significant elevations in BP. The abrupt discontinuation of antihypertensive drugs, especially clonidine or beta-blockers, may result in catecholamine excess and significant rebound HTN.

Physical examination

Most hypertensive patients presenting to the ED do not have distinguishing physical examination findings. As with the history, it is important to search for manifestations of end-organ dysfunction. This information will help determine whether the patient is having a hypertensive emergency requiring prompt treatment, or a hypertensive urgency requiring specific follow-up.

Vital signs

The BP should be checked more than once with an appropriately sized cuff while the patient is in the ED (Table 31.2). A cuff that is too small will produce an erroneously high reading. Most BP devices occlude the artery with an inflatable cuff and measure BP either by oscillometry or detection of Korotkoff sounds. Oscillometric or automatic devices are subject to greater error than auscultating for Korotkoff sounds by manual measurement. BP measurements can be different between arms. However, differences greater than 20 mmHg for systolic BP or 10 mmHg for diastolic BP should raise concern for a vascular abnormality. If autonomic instability is suspected, the BP should be checked in three positions – prone, upright sitting and upright standing. BP measurement from the patient with a gravid uterus should be obtained in the left lateral recumbent position. Otherwise, the BP reading can be erroneously low due to compression of blood return from the inferior vena cava by the gravid uterus. In addition to BP measurements, it is important to note whether the patient is tachypneic or hypoxic, and to measure visual acuity.

Table 31.2 Normal ranges of BP

	Blood pressure (mmHg)	
Age (years)	**Systolic**	**Diastolic**
0–2	60–110	40–65
3–6	80–120	55–70
7–10	90–130	60–75
11–15	90–140	60–80
15	90–139	60–89

Funduscopic

The retinae are examined for vascular changes, hemorrhages and exudates. The optic disc margins are examined for edema. Acute hypertensive changes include papilledema, fundal hemorrhages and vasospasm. Chronic hypertensive changes include arteriovenous nicking, hard exudates and silver wiring (Figure 31.1).

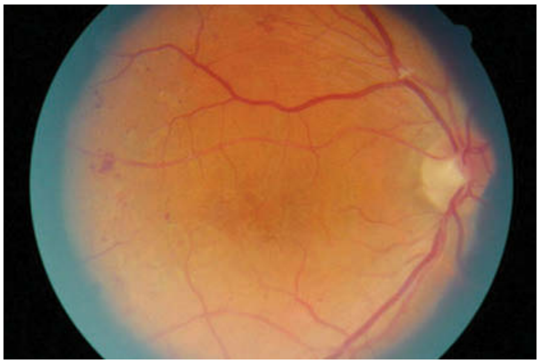

Figure 31.1
Fundus from a patient with hypertensive retinopathy demonstrates copper and silver wiring, with a small exudate seen in the upper right portion. Photo by Aaron Sobol, MD. Used with permission from Knoop KJ, Stack LB, Storrow AB, Thurman RJ, *The Atlas of Emergency Medicine*, 3rd ed., McGraw-Hill, New York, 2010.

Neurologic

The mental status examination should specifically address whether the patient is alert and aware. Alertness requires normal functioning of the reticular activating system. Awareness requires alertness plus normal functioning of cerebral hemispheres. Mental status changes may indicate hypertensive encephalopathy. Focal abnormalities of speech, cranial nerves, motor or sensory systems, or reflexes may be the result of subarachnoid hemorrhage, stroke, or preeclampsia (in a pregnant patient).

Cardiovascular

This examination should focus on identification of pulse abnormalities and heart failure. Pulses should be palpated and compared between each extremity. Diminished pulses may be present in patients with coarctation of the aorta or aortic dissection. Patients with heart failure may have tachycardia, a third heart sound (S3) or gallop, jugular venous distension (JVD), hepatojugular reflux (HJR), pulsus alternans, and/or peripheral edema.

Pulmonary

The physical findings of left heart failure include tachypnea and pulmonary rales; rhonchi and wheezing may be present secondary to airway edema, referred to as “cardiac asthma.”

Abdomen/flank

The abdomen should be examined for a bruit or palpable pulsatile mass that may indicate the presence of an abdominal aortic aneurysm (AAA). A bruit auscultated over the flank region may suggest renal artery stenosis, which can result in HTN.

Skin

The skin should be examined for evidence of poor peripheral perfusion (cool, dusky, clammy, poor capillary refill) or stigmata of chronic HTN or renovascular disease, such as pallor due to anemia. If HTN is paroxysmal, which may occur due to pheochromocytoma, the skin may be diaphoretic and flushed.

Differential diagnosis

Table 31.3 describes diagnoses associated with acute HTN.

Table 31.3 Diagnoses associated with acute hypertension

Diagnosis	Symptoms	Signs	Work-up
Acute coronary syndrome	Chest discomfort or pain, dyspnea, nausea, sweating	Diaphoresis, pallor, restlessness	ECG, cardiac enzymes
Acute pulmonary edema	Dyspnea, orthopnea, DOE, PND	Rales or crackles	ECG, CXR, BNP, cardiac enzymes
Acute renal failure	Dizziness, oliguria	Edema, decreased skin turgor	ECG, UA, serum and urine electrolytes (especially potassium), serum Cr, BUN
Aortic dissection	Chest, back or abdominal pain; dyspnea; weakness	Diminished pulses, pulse or BP differences in arms, new murmur, neurologic deficits	CXR, chest CT, ECG, aortography, transesophageal echocardiography (transthoracic may not identify), possibly MRI
Hypertensive encephalopathy	Severe headache, nausea, vomiting, confusion, seizure, stupor, decreased vision	Obtundation, retinopathy, papilledema, localized neurologic deficits without anatomic pattern	Vital signs, ECG, laboratories to rule out other end-organ damage, brain CT
Illicit drug use	Headache, chest pain, dyspnea	Diaphoresis, tachycardia, tachypnea, hyperthermia	Toxicologic studies
Intracerebral hemorrhage	Headache	Aphasia, focal paresis or paralysis, decreased consciousness	Brain CT
Medication withdrawal	Chest pain, nausea	Diaphoresis, piloerection	Ask the patient, review medical record and recent medications
Pheochromocytoma	Anxiety, palpitations, headache, chest or abdominal pain in paroxysms	Diaphoresis, pallor	ECG, 24-hr urine catecholamines
Preeclampsia/eclampsia	Headache, visual disturbances, seizure (eclampsia)	Edema, altered mental status, coma	UA, coagulation studies, CBC, renal and liver function tests
Subarachnoid hemorrhage	Headache, vomiting, syncope	Stiff neck, decreased consciousness	Brain CT, LP if CT negative

BNP: B-type natriuretic peptide; BP: blood pressure; BUN: blood urea nitrogen; CBC: complete blood count; Cr: creatinine; CT: computed tomography; CXR: chest X-ray; DOE: dyspnea on exertion; ECG: electrocardiogram; LP: lumbar puncture; MRI: magnetic resonance imaging; PND: paroxysmal nocturnal dyspnea; UA: urinalysis.

Diagnostic testing

The purpose of diagnostic testing of hypertensive patients in the ED is to identify end-organ damage and to determine the presence of concomitant diseases or complications. Commonly utilized tests include hematocrit, electrolytes, blood urea nitrogen (BUN), creatinine (Cr), glucose, urinalysis (UA), urine pregnancy test, electrocardiogram (ECG), chest x-ray and computed tomography (CT) of the brain. Selected test ordering should be presentation- and patient-specific.

Laboratory studies

Complete blood count

The complete blood count (CBC), which includes the hematocrit, serves as a general measure of a patient's health. Anemia is found in a variety of chronic diseases, including chronic renal disease or renal insufficiency from sustained elevations of BP. A peripheral smear should be obtained when suspecting hemolysis and microangiopathic anemia.

Basic metabolic panel

Abnormal potassium levels may indicate hyperaldosteronism, renovascular disease or advanced renal insufficiency. This information is useful for the primary health care provider and may guide further diagnostic testing. If renal insufficiency is acute or advanced (either the etiology of or caused by HTN), hyperkalemia may be present and requires treatment. An elevation in serum creatinine suggests underlying renal disease or renovascular injury. An abnormal glucose may suggest diabetes, which may be in part responsible for HTN and is important when selecting antihypertensive therapy.

Urinalysis

Urinalysis with microscopic examination of the urinary sediment for proteinuria, red blood cells and/or cellular casts quickly assesses renal function, and may identify the presence of renal parenchymal disease. Urine can be collected for 24 hours to assess urinary catecholamine levels, which is helpful in the diagnosis of pheochromocytoma.

Pregnancy test

Women of childbearing age with HTN should have a pregnancy test completed if appropriate.

Toxicologic testing

In any hypertensive patient in whom illicit drug use is suspected, toxicologic screening is appropriate to clarify clinical management.

Electrocardiogram

An ECG should be obtained to assess for acute cardiac ischemia and left ventricular hypertrophy (LVH) due to sustained elevations of BP. Patients with prior asymptomatic myocardial infarction and HTN should be identified because of increased risk for cardiovascular complications.

Radiologic studies

A chest x-ray may reveal acute pulmonary edema or findings suggestive of aortic dissection. A brain CT should be obtained in patients with focal neurologic signs and altered level of consciousness. For patients with elevated BP and headache, clinical judgment should determine the need for CT. A chest CT is indicated for patients with suspected aortic dissection. Abdominal ultrasound or abdominal CT is recommended if AAA is suspected. In the proper hands, focused abdominal ultrasound is acceptable and can be performed quickly and safely at the bedside.

General treatment principles

An asymptomatic, hypertensive patient presenting to the ED does not require emergency treatment. There is no evidence to suggest that acute reduction of BP in these patients reduces the complications of HTN. There is evidence, however, that stroke, deterioration in renal function, or cardiac ischemia or infarction can occur as a result of rapid, uncontrolled BP reduction. The goal of drug therapy is to return BP to normal levels with minimal side effects.

There is evidence from clinical trials supporting the use of an angiotensin-converting enzyme (ACE) inhibitor, calcium channel blocker or diuretic as first-line therapy when treating HTN. As an alternative, beta-blockers could be used if there are no contraindications (i.e., bronchospasm, bradycardia, heart block, previous sensitivity to beta-blockers). Despite this evidence, many emergency physicians are uncomfortable prescribing antihypertensive medications to patients because they produce numerous side effects. Furthermore, emergency physicians feel that HTN is a chronic condition requiring long-term medication use and close follow-up, and would rather initiate treatment with lifestyle modification (diet, salt restriction, exercise, weight loss and stress reduction). The sooner appropriate medical treatment is started, however, the greater likelihood of successful BP control which reduces complications from HTN.

Some patients with concomitant medical conditions have specific treatment recommendations. These include ACE inhibitors or angiotensin receptor blockers (ARB) in diabetics with proteinuria; diuretics or calcium channel blockers in patients with isolated systolic HTN; and beta-blockers or ACE inhibitors in patients with prior myocardial infarction.

Acute BP reduction is contraindicated for hypertensive urgencies. If a noncompliant patient had been on an antihypertensive regimen, their prior therapy could be resumed. The dose of a single medication should be increased in a compliant patient. Newly diagnosed patients should be started on monotherapy. All of these patients need to be re-evaluated within a week following the initiation of therapy to confirm compliance, to monitor adverse reactions, and to adjust therapy if necessary.

The goal of therapy in the treatment of hypertensive emergencies is to limit damage to end organs with controlled reduction in BP. The general target for pressure reduction is no more than 25% of the MAP within 2 hours. These patients must be closely monitored, ideally with invasive continuous arterial measurements. Antihypertensives used in the management of hypertensive emergencies are listed in the Tables 31.4 and 31.5.

Table 31.4 Recommended medications for specific hypertensive emergencies

Emergency condition	Drug(s) of choice	Alternative drug(s)
Acute coronary syndrome	Nitroglycerin, beta-blockers	Nitroprusside, labetalol
Acute pulmonary edema	Nitroglycerin, nitroprusside	Fenoldopam, ACE inhibitors
Adrenergic crises	Phentolamine, nitroprusside with beta-blockers	Labetalol
Aortic dissection	Beta-blockers followed by nitroprusside	Labetalol, esmolol, trimethaphan
Hypertensive encephalopathy	Nitroprusside, fenoldopam	Labetalol, nicardipine
Intracranial hemorrhage	Labetalol	Nitroprusside, nicardipine
Preeclampsia eclampsia	Hydralazine	Nicardipine, labetalol

ACE: angiotensin-converting enzyme.

Table 31.5 Pharmacologic agents in the treatment of hypertensive emergencies

Agent	Dose	Comments
Esmolol	Loading dose 250–500 mcg/kg/min infusion for 1 min followed by a maintenance infusion of 50 mcg/kg/min × 4 min; repeat loading dose and follow with maintenance infusion using increments of 50 mcg/kg/min for 4 min. May repeat up to four times as needed. Once desired BP is approached, reduce maintenance infusion from 50 mcg/kg/min to 25 mcg/kg/min or less.	Test beta-blocker safety and tolerance in patients with history of COPD, asthma or CHF who are at risk of bronchospasm from beta-blockade. When used with nitroprusside for treatment of aortic dissection, its use should precede nitroprusside to prevent reflex tachycardia and increased dP/dt. Contraindications include documented hypersensitivity, uncompensated CHF, bradycardia, cardiogenic shock and AV conduction abnormalities.
Fenoldopam	Continuous infusion only. Dose 0.1–1.6 mcg/kg/min, with usual dose 0.3 mcg/kg/min.	Primarily used in patients with renal impairment.
Hydralazine	Dose 5–20 mg IV Q 4–6 hrs PRN initial dose; increase dose PRN. Change to PO as soon as possible.	Used in the treatment of eclampsia.
Labetalol	Initial dose 20 mg IV over 2 min; follow with 20–80 mg Q 10–15 minutes until BP controlled. Maintenance dose 2 mg/min continuous infusion; titrate up to 5–20 mg/min, not to exceed a total dose of 300 mg.	Contraindications include coronary or cerebral arteriosclerosis, renal impairment or documented hypersensitivity.
Nicardipine	Initial infusion 5 mg/hr, titrate 2.5 mg/hr every 5–15 min. Maximum 15 mg/hr, maintenance 3 mg/hr. Pheochromocytoma: 0.5–2 mg boluses repeated as needed. Preeclampsia: initial dose 1 mcg/kg/min, titrate 0.5 mg/hr (typical rate 0.7 mcg/kg/min).	Contraindications include aortic stenosis, known or previous hypersensitivity to calcium channel blockers.
Nitroglycerin	Dose 5–10 mcg/min IV titrating upward to keep SBP > 90 mmHg or to decrease MAP by 25%. Continuous 0.1–1 mcg/kg/min IV infusion. Doses may reach > 100 mcg/min pending hemodynamic tolerance.	Side effects include headache, hypotension, tachycardia.
Nitroprusside	Dose 0.3–10 mcg/kg/min in D5W.	Must protect from light. Contraindications include documented hypersensitivity, IHSS, atrial fibrillation or flutter. Caution in renal or hepatic insufficiency, as levels may increase and can cause cyanide or thiocyanate toxicity, especially with prolonged use or with doses greater than 4 mcg/kg/min. Arterial invasive monitoring recommended.

ACE: angiotensin-converting enzyme; AV: atrioventricular; BP: blood pressure; CHF: congestive heart failure; COPD: chronic obstructive pulmonary edema; dP/dt: rate of rise of aortic pressure during systole; D5W: 5% dextrose in water; ED: emergency department; HTN: hypertension; IHSS: idiopathic hypertrophic subaortic stenosis; IV: intravenous; MAP: mean arterial pressure; PO: per os; Q: every; SBP: systolic blood pressure.

Special patients

Pregnant

HTN in pregnancy contributes significantly to maternal, fetal, and neonatal morbidity and mortality. In pregnancy, HTN is defined as a BP ≥140/90 mmHg, a 20 mmHg rise in the systolic BP from baseline, or a 10 mmHg rise in the diastolic BP from baseline. The threshold for the diagnosis of HTN in pregnancy can therefore be lower than in the non-pregnant patient. The hypertensive emergencies of pregnancy include preeclampsia and eclampsia. Hydralazine has classically been the antihypertensive drug of choice in hypertensive crises of pregnancy because it preserves uterine blood flow. Methyldopa is effective for chronic HTN, and its long-term safety is proven. Magnesium sulfate is effective in reducing the incidence of eclampsia in women with severe preeclampsia. Patients receiving magnesium should have their airway and reflexes monitored closely. Delivery of the infant is the only cure for eclampsia.

Pediatric

HTN in children is defined as a systolic or diastolic BP greater than the 95th percentile for age and sex. Generally, young children with significant HTN have underlying renal or renovascular causes. These children should be referred to primary care pediatricians or pediatric nephrologists for further diagnostic evaluation. Primary HTN does occur in children and represents 80% of hypertensive adolescents. Obesity is an important risk factor for future development of HTN in children.

Stroke

Patients with acute hemorrhagic stroke must have their BP monitored aggressively. Controversy exists regarding whether to reduce a patient's BP if it is not too high, as acute BP reduction might result in cerebral hypoperfusion or increase the size of the hypoperfused area. It is important to determine the patient's baseline BP before initiating treatment. Patients with an acute ischemic stroke identified for tissue plasminogen activator (tPA) administration are at increased risk of intracerebral hemorrhage (ICH) if their BP is elevated. Repeated measurements of SBP >185 mmHg or DBP >110 mmHg are a contraindication to tPA administration. BP measurements should be obtained at least every 15 minutes during and after thrombolytic treatment for the first 2 hours, with additional measurements over the first 24 hours according to protocol. Patients should be observed closely for ICH or hypotension if antihypertensives are necessary.

Disposition

All patients with identified hypertensive emergencies must be admitted to an intensive care or telemetry setting for appropriate monitoring. Patients with identified hypertensive urgencies can be referred for close primary care follow-up, preferably within 1 week. Patients with elevated BP in the ED and concomitant medical conditions should have the presenting conditions stabilized with referral to primary care for follow-up within 1–2 weeks for a BP recheck. Patients with elevated BP in the ED without concomitant medical conditions should be referred to primary care for a BP recheck within 1 month, sooner if possible.

Occasionally, patients may present with a primary complaint of elevated BP that has been noted on a screening examination. If no prior history of HTN exists, the above guidelines for referral should be followed. If a patient was treated in the past for HTN, appropriate drug therapy could be reinstated with primary care referral within a month, sooner if possible.

Pearls, pitfalls and myths

- Not all patients who present with elevated BP require emergent treatment. Evidence of acute end-organ damage defines a hypertensive emergency and the need for prompt, controlled therapy.
- When caring for patients with HTN, it is best to "treat the patient, not the number."
- Patients with evolving ischemic strokes frequently have elevated BP. A rapid reduction of BP may cause extension of neurologic damage due to decreased cerebral blood flow. Therefore, urgent lowering of the BP should be avoided. In addition, oral and sublingual medications that cannot be titrated should not be used.
- Patients with asymptomatic HTN can be managed as outpatients in a primary care setting.
- Patients presenting with HTN and heart failure should be treated for fluid overload first, with precautions for cardiac ischemia or other cardiac events.
- Pregnant patients are considered hypertensive at lower absolute BP measurements than non-pregnant patients. Preeclampsia and eclampsia are two serious conditions specific to pregnant or postpartum patients.

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32 Joint pain

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Scope of the problem

Atraumatic joint pain is a common presenting complaint of emergency department (ED) patients. All emergency practitioners should be well versed in the evaluation of the "red, hot joint." Although rarely life-threatening, joint pain may be the harbinger of serious systemic diseases and infections. Furthermore, even mild diseases of many joints can result in significant short- and long-term disability. This is especially true of the hands and weight bearing joints. Therefore, accurate evaluation and treatment may reduce the severity and duration of disability.

Anatomic essentials

In order to determine the cause of joint pain, one must understand the underlying anatomy (Figure 32.1). Sources of joint pain may be classified into two anatomic categories: articular structures (joint capsule and its contents) and periarticular structures (structures superficial to the joint capsule).

A joint is the union of the ends of two or more bones. On each bony surface lies a cushion (the articular cartilage), a compressible matrix of collagen fibers, and proteoglycans that serve to prevent bone-on-bone contact. Adherent along the margins of the articular cartilage is a synovial membrane that creates the synovial cavity. The synovial membrane also secretes high-viscosity synovial fluid, which fills the synovial cavity in order to lubricate the joints and facilitate mobility. A fibrous joint capsule encloses the synovial membrane, creating the unit known as the *joint space*.

The joint is strengthened and supported by ligaments, which are bands of fibrous tissues connecting bones or cartilages. Additional support is provided by tendons, which are cords of fibrous tissue continuous with muscle fibers. These attach the muscle to bone or cartilage. More than 150 sacs of synovial fluid, known as *bursae*, are situated throughout the body around joints where friction occurs. Examples include the *olecranon bursa* beneath the skin superficial to the olecranon process and the *subacromial bursa* between the acromion and supraspinatus muscle.

A variety of maladies may lead to joint pain, which frequently results from the disruption of the normal articular or periarticular anatomy. Mechanical trauma may lead to imbalance of the anabolic and catabolic processes maintaining normal joint homeostasis. Inflammatory reactions may be triggered by various stimuli, including bacteria, crystals, trauma, or systemic inflammatory conditions. Inflammation of either the articular or periarticular structures is associated with movement of polymorphonuclear cells (PMNs) into the synovial cavities, resulting in a decrease in the viscosity of the synovial fluid. Lysosomal enzymes released from these PMNs attack joint structures, invoking a severe inflammatory response. Unlike cartilage, synovium is rich in pain receptors. Therefore, even minor inflammation may lead to severe pain and disability.

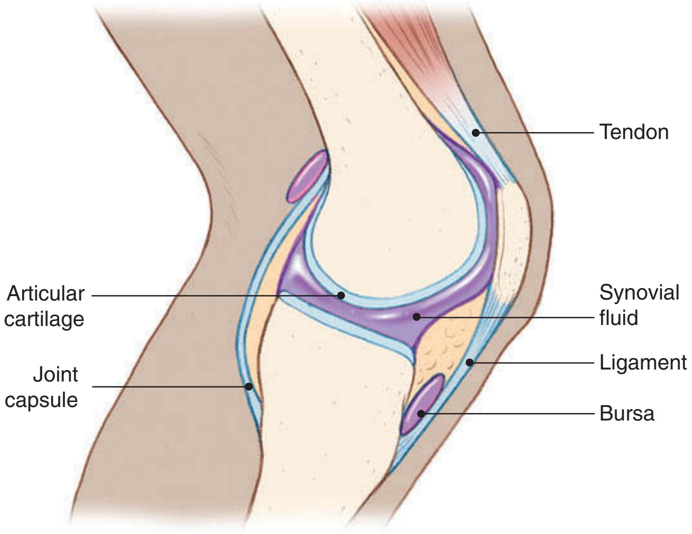

Figure 32.1
Joint anatomy. © Chris Gralapp.

Table 32.1 Joint pain red flags

History	Concerning diagnosis
Infants, elderly, immunosuppression	Septic arthritis
Injection drug use	Septic arthritis
Fever or recent infection	Septic arthritis, rheumatic fever
History of or risk for cancer	Pathologic fracture
Chest pain	Acute rheumatic fever
Tick bite	Lyme disease
Examination finding	**Concerning diagnosis**
Ill-appearing or toxic	Septic arthritis
Heart murmur	Endocarditis, rheumatic fever
Limitation of range of motion	Septic arthritis, fracture
Monoarticular joint effusion, erythema, warmth	Septic arthritis
Pustular or hemorrhagic skin lesion	Gonococcal arthritis
Urethritis/cervicitis	Gonococcal arthritis, Reiter's syndrome
Erythema marginatum, subcutaneous nodules	Acute rheumatic fever
Erythema chronicum migrans	Lyme disease
Oral ulcerations	Reiter's syndrome

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 32.1).

History

Obtaining key historical information from a patient with joint discomfort allows determination of whether the pain involves the joint capsule (articular) or the structures surrounding the joint capsule (periarticular). If the pain is articular, the number and distribution of joints helps guide the differential diagnosis (Table 32.2).

Table 32.2 Causes of joint pain

Monoarticular	Polyarticular	Periarticular
Gout Hemarthrosis Osteoarthritis Pseudogout Septic arthritis Trauma	Acute rheumatic fever (ARF) Drug-induced arthritis Gonococcal arthritis Immune complex Lyme disease Reiter's syndrome Rheumatoid arthritis (RA) Seronegative spondyloarthropathies Systemic lupus erythematosus (SLE) Viral arthritis	Bursitis Cellulitis Tendonitis

Where is the pain located?

Articular pain is often described as a generalized sensation of joint pain because the inflammatory process affects all parts of the joint. Periarticular pain, in contrast, is more readily localized to a specific site of inflammation.

Is the pain referred from another location?

A site distinct from the joint itself may be the actual cause of pathology. Additionally, coexisting axial pain of the neck and/or back is more commonly associated with osteoarthritis (OA) than rheumatoid arthritis (RA).

When did the pain begin?

Acute pain may be secondary to an injury, infection or inflammatory process, or may be due to an acute exacerbation of a chronic condition. Patients with chronic arthritic conditions will often provide a history of recurrent acute episodes.

What makes the pain worse?

Because synovial tissue is rich in stretch receptors, articular pain is often exacerbated by both active and passive motion of the joint. It may even occur at rest, as seen in arthritic conditions (e.g., inflammatory or septic arthritis). Periarticular pain is usually exacerbated with active or passive movement involving the affected muscles or tendons. It is commonly seen with overuse conditions, in which repetitive motion results in inflammation of the involved tissues (e.g., shoulder bursitis or tendinitis in a baseball player).

How many joints are involved?

Periarticular disease (e.g., bursitis, tendonitis, cellulitis) typically involves a single joint. Articular disease may be monoarticular or polyarticular. Examples of monoarticular arthritides include septic and gouty arthritis. Polyarticular arthritis may be acute (e.g., viral arthritis, Lyme disease, or gonococcemia) or chronic (e.g., systemic lupus erythematosus [SLE], psoriatic arthritis, or dermatomyositis). Polyarticular arthritis may also be symmetric (e.g., drug-induced arthritis, RA) or asymmetric (e.g., rubella, gonococcal arthritis, acute rheumatic fever).

Which joints are involved?

Certain arthritic conditions are often recognized by joint pain in distinctive locations. Osteoarthritis commonly affects the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints of the hand; RA commonly affects the metacarpophalangeal (MCP) and PIP joints of the hand. Gout commonly affects the great toe metatarsophalangeal (MTP) joint (known as *podagra*), the ankle and the knee. Symmetric arthritis is typical of RA and SLE; asymmetric arthritis is typical of psoriatic and Lyme arthritis. Occasionally, a patient may present with joint pain, particularly in the hip, that may be referred pain from a bony metastatic lesion in the setting of an underlying malignancy.

Associated signs and symptoms

Eliciting other specific extra-articular information will help guide the differential diagnosis of a patient with joint pain. A low-grade fever is not unusual with inflammatory conditions of a joint. However, the presence of high fever and chills must alert clinicians to the possibility of septic arthritis. Constitutional symptoms are commonly seen not only with infection but also with other systemic or autoimmune disorders (e.g., RA). Skin lesions should increase suspicion for a systemic or autoimmune disease (e.g., track marks of injection drug users in endocarditis, malar rash of SLE). Ocular findings (such as episcleritis in RA or anterior uveitis in ankylosing spondylitis) may provide clues to the final diagnosis. Tick bites may cause Lyme disease (and Lyme arthritis) in endemic areas. Purulent urethritis suggests gonococcal disease. The combination of conjunctivitis and urethritis suggests Reiter's syndrome. Uric acid nephrolithiasis may be associated with gouty arthritis.

Past medical

Obtaining information about the past medical history may yield additional clues to the etiology of joint pain. One must inquire about autoimmune disease, gout, known arthritic conditions, hemophilia and malignancy. The medication history may prove useful, as medications may precipitate or be used as treatment for arthritides. Thiazide diuretics increase uric acid levels and may precipitate gout. Isoniazid, procainamide and hydralazine therapy may lead to a lupus-like syndrome with arthritis. Chronic arthritides have a familial predisposition, so family history may prove helpful. Social history is often essential in making a definitive diagnosis. Inquiries about overuse activities, sports, injection drug use and sexual history should be made. Vocational history is also important, as certain joint conditions are due to distinct injuries or repetitive stress, commonly associated with specific occupations (e.g., anterior cruciate ligament tear in a football player or prepatellar bursitis in a housemaid). Hiking or other outdoor activities in areas endemic with Lyme disease may suggest Lyme arthritis from a tick bite.

Physical examination

Once a thorough history has been elicited, physical examination is important to allow practitioners to generate a differential diagnosis. The basic principles of physical examination of the musculoskeletal system are inspection, palpation and range of motion.

General appearance

Patients with acute inflammatory or infectious joint conditions will present with varying degrees of pain and distress. However, an acutely ill or toxic-appearing patient with joint pain will likely have a significant infection or an underlying systemic condition that may or may not have been previously identified.

Vital signs

In the presence of a high fever and joint pain, it is imperative to look for evidence of infection. Other signs suggestive of infection may include tachycardia or hypotension, if the infection is systemic. Inflammatory conditions typically present with a normal temperature or low-grade fever, and possibly mild tachycardia or tachypnea due to pain.

Musculoskeletal examination

Inspection

Redness of the overlying skin is often associated with periarticular processes (e.g., cellulitis of the skin, bursitis or tendinitis from overuse or injury), and should alert clinicians to the possibility of infection of the underlying joint. This condition warrants immediate orthopedic evaluation.

Swelling within the joint (an effusion) may be secondary to blood from an injury, purulence from infection,

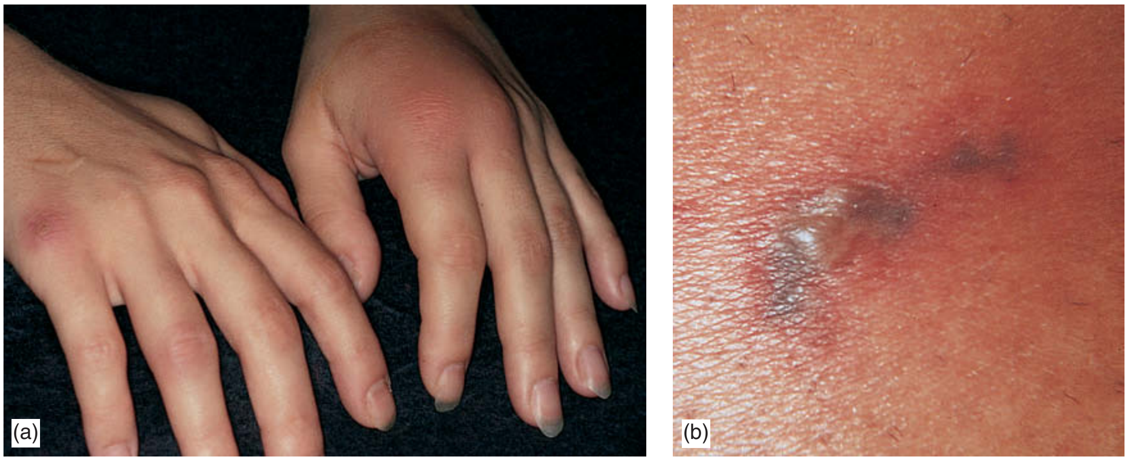

Figure 32.2
Disseminated gonococcal infection. (a) Erythema and swelling of the joints of the left hand; a single vesicle is present on the right hand; (b) more advanced lesion with a hemorrhagic and necrotic base; the central hemorrhagic area is the embolic focus of the gonococcus. Reprinted from Habif TP, *Clinical Dermatology*, 4th ed., p. 333, Copyright 2004, with permission from Elsevier.

or excessive amounts of synovial fluid from synovitis. A joint effusion is usually palpable. However, fluid may also be periarticular, originating from similar causes and involving tissues surrounding the joint (e.g., prepatellar bursitis).

Wounds may be superficial, indicating a minor injury, or contiguous with the joint space, necessitating urgent orthopedic intervention to prevent or treat a potential infection within the joint. Pustular lesions may indicate a potential systemic infection, such as gonococcemia (Figure 32.2).

Muscle atrophy is evidence of disuse due to pain in the involved joint. *Deformities* frequently enable an immediate diagnosis to be made, whether due to a fracture or dislocation, or a specific condition (e.g., tophaceous lesions of gouty arthritis or Heberden's nodes of OA).

In chronic arthritic conditions, the *distribution* of joint involvement often suggests a specific diagnosis. For example, OA has a predilection for the first carpometacarpal joints, the first MTP joints, the DIP joints, and the knees, hips, cervical and lumbosacral spine. In contrast, RA affects the wrists, MCP joints, PIP joints and feet. *Symmetry* of deformities on both sides of the body is typical of RA.

Palpation

Warmth suggests inflammation, and should be compared to the surrounding tissues or the opposite joint. It may be due to arthritis, infection, or acute trauma. *Tenderness* often reveals the underlying pathology if localized to a specific anatomic structure. Generalized tenderness may imply involvement of the entire joint. *Swelling*, particularly due to a joint effusion, may be palpable if not easily visualized on inspection.

Range of motion

Range of motion varies from person-to-person, but no musculoskeletal examination is complete without moving a joint through its full range of motion. Decreased range of motion may be present secondary to pain from degenerative joint disease (DJD), inflammation, infection, or injury, even if no abnormalities are identified on inspection or palpation. Decreased range of motion may also be seen with increasing age. Increased range of motion may occur with joints that are unstable from ligamentous injury, or in association with hyperelastic connective tissue (i.e., Ehlers-Danlos syndrome). Symmetry in range of motion should be identified and documented.

The presence of pain with range of motion is not a specific finding, as it may be found with numerous pathologies involving the joint. It is usually more helpful if the pain can be localized to a specific structure. Involvement of periarticular structures commonly results in pain during active range of motion. Another maneuver to assess range of motion is pain with axial-loading or weight bearing, which is more likely seen with arthritic conditions or an injury to the joint.

Head to toe

In addition to a focused examination of the joint, a thorough examination involving the remaining organ systems may be required in the presence of other patient complaints, signs or symptoms (Table 32.3). A detailed examination will help identify evidence of an underlying systemic disease that may not have been suspected initially based on the chief complaint of joint pain (e.g., skin tightening in scleroderma, heart murmur of endocarditis, pharyngitis or eye findings of associated arthritides, or skin rashes of SLE or Lyme arthritis [Figure 32.3]).

Table 32.3 Clues to specific arthritic diseases

	Findings	Diseases
Skin	Pustules (Figure 32.2b) Malar rash Rash on elbows, knees ECM (Figure 32.3) Hyperkeratotic lesions Tophi Track marks Erythema marginatum Subcutaneous nodules	Gonococcemia SLE, dermatomyositis Psoriasis Lyme disease Reiter's syndrome Gout Injection drug use Rheumatic fever Rheumatic fever, rheumatoid arthritis
Eyes	Iritis, uveitis Conjunctivitis Scleral icterus	Seronegative spondyloarthropathy Reiter's syndrome Hepatitis
Mouth	Ulcerations	Reiter's syndrome
Cardiac	Friction rubs Murmurs	Rheumatic fever, rheumatoid arthritis, SLE Rheumatic fever, rheumatoid arthritis
Pulmonary	Pleuritis	SLE, rheumatoid arthritis
Gastrointestinal	Enlarged, tender liver	Hepatitis
Genitourinary	Purulent urethral discharge	Reiter's syndrome, gonococcemia

ECM: erythema chronicum migrans; SLE: systemic lupus erythematosus.

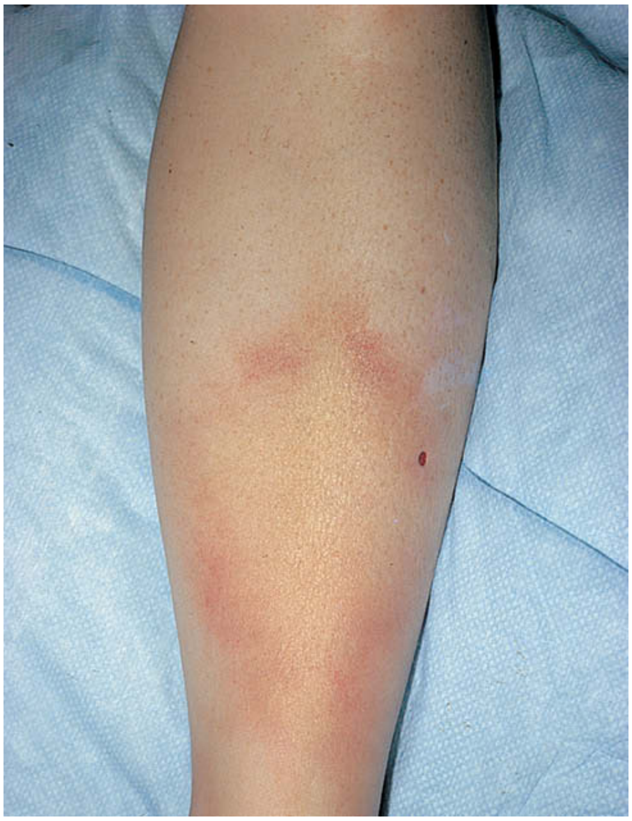

Figure 32.3
Erythema chronicum migrans of Lyme disease. Broad oval area of erythema that has slowly migrated from the central bite puncture. Reprinted from Habif TP, *Clinical Dermatology*, 4th ed., p. 519, Copyright 2004, with permission from Elsevier.

Differential diagnosis

Table 32.4 describes causes of joint pain.

Diagnostic testing

Laboratory studies

Laboratory testing, with the exception of synovial fluid analysis, is rarely useful in evaluating a patient with an atraumatic cause of joint pain. Clinicians generally order a complete blood count (CBC), looking for an elevated white blood cell (WBC) count (indicative of an infectious disease) or anemia (suggestive of systemic rheumatic disease). The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are commonly ordered to screen for inflammatory and infectious conditions; however these tests do not demonstrate adequate sensitivity or specificity to assist with the final diagnosis. Because septic arthritis is typically the result of hematogenous spread of bacteria to the joints, blood cultures may have some utility. If gonococcus (GC) is a suspected pathogen in septic arthritis (particularly in younger patients), cultures for GC should be taken from the pharynx, urethra, rectum and cervix. Antistreptolysin O (ASO) titers or a throat culture may provide evidence of antecedent streptococcal infection in acute rheumatic fever. In acute gout, serum uric acid levels are often normal.

If the practitioner has suspicion for a systemic rheumatic disease, as would be the case for patients with polyarthritis of more than 6 weeks duration or with an inflammatory synovial fluid, the following lab tests are recommended: CBC, ESR, anti-nuclear antibody (ANA), rheumatoid factor (RF), creatinine, urinalysis and ASO titer. If the clinician is suspicious of a viral infectious etiology, as seen with polyarthritis of less than 6 weeks duration, the following lab tests are suggested: CBC with differential, liver function tests (to assess for the presence of hepatitis), hepatitis B and C serologies (if indicated by abnormal liver function tests), and parvovirus serology. Additional studies, such as Lyme serologies, may be sent as indicated by the clinical picture and pretest probability. In atraumatic hemarthrosis, an evaluation for hemophilia should be considered, including prothrombin time (PT),

Table 32.4 Differential diagnosis of joint pain

Diagnosis	Symptoms	Signs	Work-up
Acute rheumatic fever	Polyarticular (symmetric or asymmetric) arthritis, usually affecting the large joints; may accompany carditis, valvulitis, rash, or chorea after a group A β-hemolytic streptococcal infection of the pharynx.	Patients may demonstrate *erythema marginatum*, a pinkish, non-pruritic rash with central clearing on the trunk and proximal limbs. *Subcutaneous nodules* are firm and nontender, and overly bony prominences. In addition to the symmetric polyarthritis, patients may have evidence of cardiac disease, including pericarditis, CHF, or valvular abnormalities. The major neurologic manifestation is chorea with sparing of sensory function.	Laboratory testing should include ESR, CRP, ASO titer, and pharyngeal cultures. The synovial fluid is inflammatory with a negative culture. ECG may show evidence of carditis or pericarditis. Echocardiography may demonstrate valvular dysfunction.
Drug-induced arthritis	Polyarticular, symmetric arthritis, associated with lupus-like systemic symptoms and a history of procainamide, hydralazine, or isoniazid treatment.	Mild to moderate arthritis and synovitis with effusions, symmetrically distributed. Other systemic signs of the lupus-like syndrome may be present.	Synovial fluid shows a noninflammatory picture, although cell counts may suggest an inflammatory picture in severe cases.
Gonococcal arthritis	Polyarticular (symmetric or asymmetric) arthritis, affecting one to several joints – typically the knee, ankle, or wrist. Associated with rash, fever, chills, and rarely urethritis or cervicitis.	Fever often present. The rash (70%) is characterized by hemorrhagic, necrotic pustules starting on the distal extremities. Acute inflammation in the joints of the knee, ankle and wrist, or the tendon sheaths of the hands or wrists is common.	CBC may show elevated WBC; ESR may be elevated. Blood cultures are positive for *N. gonorrhoeae* <50% of the time, but should be sent. Cultures for *N. gonorrhoeae* should be sent from the pharynx, rectum, cervix and urethra. Synovial fluid analysis shows an inflammatory fluid. Gram stain is positive more often than cultures.
Gout	Monoarticular symptoms, with >75% occurring in the MTP joint of the great toe. Pain is severe at onset. Systemic symptoms are uncommon.	Affected joints are erythematous, warm, and exquisitely sensitive to touch or movement. Fever is uncommon.	Serum uric acid may be normal in acute attacks. Synovial fluid analysis demonstrates negatively birefringent, needle-shaped crystals and high WBC counts in the fluid. Septic arthritis must be ruled out by joint fluid Gram stain and culture, as the presence of gout crystals does not rule out septic arthritis.
Lyme disease	Polyarticular (symmetric or asymmetric) arthritis is a late manifestation of this disease, caused by a spirochete, *Borrelia burgdorferi*, from the deer tick *Ixodes dammini*. The tick bite leads to rash, fever, malaise, myalgias and arthralgias. Occasionally, the patient may have neurologic (Bell's palsy or other mononeuritis) or cardiac symptoms (syncope).	Arthritis of the large joints occurs in more than half of patients. Large joint effusions are common. The rash of *erythema chronicum migrans* appears at the site of the tick bite, and may be followed by spread to the thigh, axilla and groin.	Lyme serologies may not be positive for 6 weeks following initial exposure, but are often positive by the time arthritis develops. Synovial fluid demonstrates inflammatory changes.
Osteoarthritis	Monoarticular arthritis with acute flare-ups in a joint with long-standing chronic disease. Typically no systemic symptoms. The hands, knees and first MTP joints are most commonly affected.	Patients are usually >50 years old, and have crepitus and swelling in the affected joints. Bouchard's nodes (PIP) and Heberden's nodes (DIP) are osteophytic spurs often present in the hands.	CBC is often normal; ESR may be slightly elevated. Radiographs demonstrate formation of osteophytes and joint space narrowing. Synovial fluid analysis typically shows a noninflammatory picture, with cell counts <2,000 cells/mm^3.
Pseudogout	Monarticular arthritis usually occurring in the knee, wrist, ankle and elbow. More than one joint may be involved. Typically less acute and less severe than gout.	Affected joints are erythematous, warm and exquisitely sensitive to touch or movement. Systemic systems are less common than with gout.	WBC count is often elevated, as is ESR. Radiographs sometimes show calcifications (chondrocalcinosis) in joints, tendon insertions, ligaments and bursae. Synovial fluid shows rhomboidal, weakly positive birefringent crystals of calcium pyrophosphate dehydrate. Septic arthritis must be ruled out by joint fluid Gram stain and culture.

(*continued*)

Table 32.4 Differential diagnosis (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Rheumatoid arthritis	Polyarticular, usually symmetric, arthritis with acute flare-ups occurring in the context of chronic disease. The hands (MCP and PIP joints), wrists, elbows and feet (MTP joints) are most often affected. Patients often complain of fatigue, malaise and joint pain lasting months.	Joints are warm, tender, and swollen. Long-term changes of rheumatoid arthritis include severe deformities such as *swan neck* and *boutonniere* deformities in the hand, and muscle atrophy and Baker's cysts in the knees. Subcutaneous nodules may be present.	CBC may show mild anemia. ESR is often elevated. Rheumatoid factor, an antibody against IgG, is positive in 85% of patients but is nonspecific. Radiographs demonstrate soft tissue swelling and joint space narrowing. Synovial fluid is typically inflammatory.
Septic arthritis	Monoarticular joint pain, warmth, and swelling. More than two-thirds of cases involve the knee, hip, or shoulder. Joint symptoms are associated with fever (80%), chills (20%) and malaise.	Affected joints are erythematous, warm, and exquisitely sensitive to touch or movement. Fever may be present.	WBC count is often, though not always, elevated. ESR is commonly elevated, although nonspecific. Blood cultures may grow etiologic agents (50%). Synovial fluid analysis shows a markedly elevated WBC count with a predominance (>75%) of polymorphonuclear leukocytes. Gram stain may demonstrate organisms (50–70%), although synovial culture is the gold standard. X-rays are not usually helpful.
Seronegative spondyloarthropathies	Polyarticular, symmetric arthritis, often associated with other symptoms such as psoriatic rash, low back pain from sacroiliitis, urethritis, or uveitis. Family history of similar illnesses (ankylosing spondylitis, psoriatic arthritis, arthropathy of IBD, Reiter's syndrome and other reactive arthritides) often present. Onset is subacute (may occur over several months). Morning stiffness may be a component of all of these.	In addition to polyarticular, symmetric arthritis, a variety of clinical signs related to the underlying condition (psoriatic rash or uveitis) may be present.	Laboratory analysis shows a negative rheumatoid factor, although CBC may show mild anemia with elevated WBC. ESR is often elevated. 80% of patients have the HLA-B27 marker. Radiographs may demonstrate sacroiliitis and "bamboo spine" in ankylosing spondylitis. Synovial fluid is typically inflammatory.
Trauma/hemarthrosis	Monoarticular joint pain and swelling, with history of known trauma. In the case of coagulopathy, a history of trauma may be absent. Systemic symptoms are absent, although a personal or family history of bleeding diatheses may be present in patients with coagulopathy.	Pain and swelling in the affected single joint. Signs of inflammation are minimal acutely. Systemic signs, such as fever, are typically absent.	Laboratory testing (PT, PTT, bleeding time, platelet count) may demonstrate evidence of coagulopathy if the hemarthrosis is atraumatic. Radiographs may demonstrate a fracture if traumatic etiology. The synovial fluid is bloody, with fewer than 10,000 WBC mm^3; presence of marrow elements possible in trauma.
Viral arthritis (immune complex disease)	Polyarticular (symmetric or asymmetric) concomitantly or following viral syndrome, typically rubella, hepatitis B, parvovirus, mumps, or adenovirus. Typically, the PIP, knee, ankle and MCP joints are most commonly affected. Other symptoms dependent on the temporal relationship of the arthritis to the primary viral infection; fever with swollen lymph nodes may be the only other symptoms.	Fever may be present, and signs of hepatitis or other specific viral illness may be noted. The arthritis is typically severe.	Elevated WBC count may be noted. Liver function tests may be abnormal in hepatitis, and should be followed with serologies for specific viral etiologies. The synovial fluid is typically noninflammatory, with cell counts <2,000 cells/mm^3. May develop inflammatory characteristics as the disease progresses. Rubella cultures may be performed on the synovial fluid.

ASO: antistreptolysin O titer; CBC: complete blood count; CHF: congestive heart failure; CRP: C-reactive protein; DIP: distal interphalangeal; ECG: electrocardiogram; ESR: erythrocyte sedimentation rate; IBD: inflammatory bowel disease; IgG: immunoglobulin G; MCP: metacarpophalangeal; MTP: metatarsophalangeal; *N. gonorrhoeae*: *Neisseria gonorrhoeae*; PIP: proximal interphalangeal; PMNs: polymorphonuclear cells; PT: prothrombin time; PTT: partial thromboplastin time; RF: rheumatoid factor; WBC: white blood cell.

Table 32.5 Synovial fluid analysis

	Normal	Noninflammatory	Inflammatory	Septic	Traumatic
Color	Colorless	Yellow	Yellow	Yellow	Red
Appearance	Clear	Clear	Cloudy	Cloudy	Cloudy
WBC/mL	<200	<2,000	2,000–100,000	>100,000	
% PMNs	<25	<25	>50	>95	
Crystals	None	None	May be present	None	None
Culture	Negative	Negative	Negative	Positive	Negative, though bone marrow elements may be present with fracture
Typical conditions		OA, trauma, viral infection, drug-induced.	Crystal-induced arthritides, Lyme disease, acute rheumatic fever, RA, JRA, SLE, spondylo-arthropathies, viral infection, sarcoidosis.	Bacterial, including GC arthritis	Fracture, ligament injury, hemophilia

GC: gonococcus; JRA: juvenile rheumatoid arthritis; OA: osteoarthritis; PMNs: polymorphonuclear cells; RA: rheumatoid arthritis; SLE: systemic lupus erythematosus; WBC: white blood cell.

partial thromboplastin time (PTT), bleeding time and platelet count.

Synovial fluid analysis

The most useful diagnostic test for patients with acute joint pain is often synovial fluid analysis. Aspiration and examination of synovial fluid is indicated for patients with joint effusion and/or signs of inflammation. Arthrocentesis involves needle aspiration of synovial fluid from a joint space. Careful aseptic technique must be ensured. The absence of fever should not discourage clinicians from performing this procedure urgently, because arthrocentesis is necessary to confirm or disprove septic arthritis. This procedure is contraindicated if there is suspicion or evidence of infection overlying the landmarks for arthrocentesis. Typically, synovial fluid analysis includes cell count and differential, Gram stain and culture, and crystal analysis. Glucose analysis and viscosity may be obtained as well, but are less diagnostic. Synovial fluid should be collected in EDTA tubes (lavender top) for cell count and differential, heparin tubes (green top) for crystal analysis, and standard tubes (red top) for chemistries, serologies and viscosity. Cultures should be plated as soon as possible following aspiration, especially if GC is a suspected pathogen. A positive Gram stain is diagnostic for septic arthritis, but a negative Gram stain does not rule it out. Crystal analysis is performed using polarizing microscopy, with monosodium urate crystals in gout appearing as 2–10 micron, needle-shaped crystals that are negatively birefringent. In contrast, calcium pyrophosphate crystals in pseudogout appear as polymorphic (often rhomboid) positively birefringent crystals. High white blood cell (WBC) counts are suggestive but not diagnostic of infectious etiologies. A low WBC count does not rule out septic arthritis but makes it more unlikely. Always use caution when interpreting these results, as significant overlap exists between categories (Table 32.5). Synovial fluid cell count may also be estimated on a wet mount preparation; two or fewer WBCs per high-power field (hpf) suggests a noninflammatory effusion, whereas greater than 20 WBCs/hpf indicates severe inflammation or infection.

Electrocardiography

Electrocardiography is indicated in patients with clinical symptoms suggestive of cardiac involvement, including chest pain, palpitations, or shortness of breath. Acute rheumatic fever may cause carditis, which leads to prolongation of the PR interval, and pericarditis, which leads to diffuse ST-segment changes and shortening of the PR interval. Both of these findings are part of the Jones criteria for diagnosis of acute rheumatic fever (Table 32.6).

Table 32.6 Revised Jones criteria for the diagnosis of acute rheumatic fever

Major criteria	Minor criteria
Carditis Chorea Erythema marginatum Polyarthritis Subcutaneous nodules	***Clinical findings*** Arthralgia Fever ***Laboratory findings*** Elevated acute phase reactants (CRP or ESR) Evidence of previous group A β-hemolytic streptococcal pharyngitis (positive throat culture or rapid antigen test, elevated ASO or other streptococcal antibody titer, or recent scarlet fever) ***ECG findings*** Prolonged PR interval

Note: Diagnosis of acute rheumatic fever is made when two major or one major and two minor criteria are present. Evidence of previous group A β-hemolytic streptococcal pharyngitis is also necessary. However, the diagnosis of rheumatic fever can be made in patients with only confirmed streptococcal pharyngitis and chorea.

ASO: antistreptolysin O titer; CRP: C-reactive protein; ECG: electrocardiogram; ESR: erythrocyte sedimentation rate.

Radiologic studies

Plain radiographs of painful joints provide the most information in patients with chronic arthritides and late septic arthritis. In the acute setting, X-rays are indicated for patients with a history of significant trauma, point tenderness over an area, gross deformity, limited range of motion, inability to bear weight, or skeletal immaturity. In a patient with acute arthritis, the most likely finding is soft tissue swelling; therefore, radiographs cannot be used to "rule in" or "rule out" acute septic arthritis. Acutely, images should be reviewed for evidence of fracture, neoplasm, osteomyelitis, avascular necrosis (including Legg-Calvé-Perthes disease), slipped capital femoral epiphysis (SCFE), or other bone disease. In cases of cellulitis, careful examination of the soft tissues should be performed to rule out foreign body. Radiographs may show loss of joint space, subchondral bone destruction, and periosteal new bone formation as soon as 1 week after the onset of septic arthritis.

Additional imaging may be done in order to clarify abnormal findings or to further evaluate the complaint of joint pain. Ultrasonography may be useful in confirming a joint effusion in a deeply situated joint (such as the hip) and can also be used to guide joint aspiration. Computed tomography better defines the bony anatomy, whereas magnetic resonance imaging (MRI) is extremely useful for defining soft tissue pathology.

General treatment principles

As with all emergency patients, treatment begins with the ABCs (airway, breathing, circulation). The main goals of treatment are physiologic stabilization, symptom relief, proper utilization of diagnostic tests and appropriate referral.

Pain relief

Given the severity of pain associated with acute synovitis of any etiology, rapid and effective pain relief is critical in treating joint pain. Patients may require parenteral opioid analgesics, such as morphine or hydromorphone, to manage their pain. Adding antiemetics to this regimen decreases the nausea and vomiting that often accompany the administration of these agents. Nonsteroidal antiinflammatory drugs (NSAIDs), such as parenteral ketorolac or oral ibuprofen, effectively reduce the pain of acute synovitis. In OA and RA, acetaminophen has been repeatedly demonstrated to decrease pain at recommended dosages. Salicylates are effective for acute rheumatic fever and RA.

In crystal arthropathies, acute synovitis is often treated with a combination of NSAIDs (indomethacin is preferred for gout) and colchicine, a microtubule inhibitor that reduces inflammation in the synovium. Colchicine may be used parenterally for acute relief, but care must be taken to prevent infiltration. Colchicine is not as effective for pseudogout as for gout, but may still prove a useful adjunct. In resistant cases, a prednisone taper or intramuscular adrenocorticotrophic hormone (ACTH) may prove useful. ACTH may also be useful in patients who cannot take NSAIDs. Uric acid-lowering agents have no role in the acute management of gouty arthritis.

Immobilization

Non-pharmacologic therapies include rest, ice, compression and elevation (RICE). Simple splinting of the affected joints often significantly reduces the pain of synovitis, since the synovial receptors are exquisitely sensitive to stretch. However, once the pain has been controlled pharmacologically, patients should be encouraged to remove splints and begin range-of-motion exercises to avoid the loss of function, stiffness, and muscle atrophy that occur with prolonged splinting.

Drainage

Arthrocentesis may be both diagnostic and therapeutic. Patients with septic or gonococcal arthritis require drainage of the affected joints. With bacterial arthritis, this may best be accomplished in the operating room by open incision and drainage, especially if a large or major joint such as the knee or hip is involved. For smaller joints, or in the case of gonococcal arthritis, repeated daily aspirations with a large bore needle or arthroscope may be necessary. This therapeutic decision is best made by the orthopedic or infectious disease consultant(s) managing the patient. If the patient has osteomyelitis or a joint prosthesis, or is resistant to conservative therapy, then open incision and drainage in the operating room should be performed. Therapeutic drainage should also be considered when a joint effusion is large enough to result in significant pain or significant limitation in range of motion or weightbearing.

Antibiotics

Outcomes after septic arthritis, the most serious cause of acute joint pain, are improved with rapid diagnosis and administration of intravenous antibiotics. Specific antibiotic selection should be made with regard to the likely microbial pathogens. It should always include vigorous coverage for *Staphylococcal* species, given their frequency of occurrence. Ceftriaxone works well against *Neisseria gonorrhoeae*. For early Lyme disease, patients should be treated with 20–30 days of oral doxycycline, amoxicillin, or cefuroxime. Erythromycin is less effective. More severe disease requires intravenous penicillin or ceftriaxone at high doses for several weeks. Acute rheumatic fever is best treated with benzathine penicillin G intramuscularly, or oral penicillin V for 10 days.

Special patients

Pediatric

Septic arthritis in the pediatric population is the result of hematogenous spread from another site, or the result of direct invasion from an area of osteomyelitis prior to growth plate closure. Clinicians must maintain a high degree of suspicion for this diagnosis in febrile children with any joint complaint, even in the presence of an alternative source of fever. The diagnosis of septic arthritis in infants is particularly challenging, because infants often present only with irritability and possibly fever. Only the most astute clinicians and parents are likely to notice a decrease in mobility of an extremity in this age population. *Staphylococcus aureus* remains an important etiologic agent across all age ranges. Neonates are at increased risk for infection due to *Escherichia coli* and *Group B Streptococcus*. In toddlers, *S. aureus*, and *Kingella kingae* are the etiologic agents. Fortunately, vaccination has reduced the incidence of *H. influenzae* septic arthritis by more than 95%. *Pneumococcus* and *S. aureus* are the most likely organisms throughout the remainder of the pediatric population, with *N. gonorrhoeae* becoming more common during the adolescent years. In children, additional consideration must also be given to conditions such as SCFE, osteomyelitis, or Legg-Calvé-Perthes disease (avascular necrosis of the femoral head) that also present with a painful joint.

Elderly

Older adults differ in several ways from the general population with respect to joint pain. First, geriatric patients have a higher incidence of chronic arthritides, which may flare, causing acute joint pain. It is also difficult to identify acute illness in the setting of chronic joint changes. With regard to pain control, the number of analgesic options may be limited. Drug–drug interactions, an increased risk of gastrointestinal bleeding with NSAIDS (particularly in association with other anticoagulants), and concerns about constipation and falls from narcotic analgesics should be considered. Older patients have a higher incidence of septic arthritis, and many have prosthetic joints. Elderly patients frequently lack the classic symptoms and signs of septic arthritis, making the diagnosis more difficult. Additionally, the prevalence of osteopenia and osteoporosis in this age group makes the likelihood of joint pain due to fracture (traumatic or pathologic) much more common. Therefore, any trauma resulting in new joint pain or a change in intensity or character of chronic joint pain should prompt consideration of plain film imaging, especially if the patient's baseline range of motion is altered. In these circumstances, lifestyle modifications (e.g., fall prevention, exercise) and nutritional assessment (e.g., calcium, Vitamin D, bisphosphonate stores, caloric intake) are unique treatment considerations requiring urgent follow-up with the patient's primary care physician.

Immune compromised

Injection drug users, with or without HIV infection, comprise an increasing proportion of patients presenting with septic arthritis. Typically, septic arthritis in the injection drug user affects joints of the axial skeleton more often than the peripheral skeleton. The most common organisms are *Pseudomonas aeruginosa*, *S. aureus*, *Enterobacter* and *Serratia marcescens*. Oncology and transplant patients have a higher rate of septic arthritis, as well as iatrogenic (drug-associated) and gouty arthritis. Increased attention must be given to complaints of joint pain in these individuals, as the clinical signs of acute inflammation are often mild or absent despite the presence of significant infection. In addition to considering a broader spectrum of bacterial pathogens in immune-compromised patients, synovial fluid cultures should include evaluation for fungi and mycobacteria. Patients with chronic arthritis, especially RA and crystal arthritis, often take immunosuppressive medications that increase their risk for septic arthritis.

Disposition

Patients suspected of having septic arthritis require confirmation of this diagnosis. For those with the diagnosis of septic arthritis established, and those in whom it can't be ruled out, orthopedic consultation and admission for intravenous antibiotics pending culture results is appropriate. Once a noninfectious etiology for joint pain is established, patients can be discharged if their pain can be adequately controlled. If this is not possible, patients may require admission for parenteral analgesics. Visiting home nursing care, assisted living, or skilled nursing facilities may be required if an episode of acute arthritis leaves a patient unable to perform activities of daily living or increases their risk for falls.

Pearls, pitfalls and myths

- Assume septic arthritis in patients presenting with monoarticular arthritis until proven otherwise, especially if systemic signs of toxicity are present.
- The number and distribution of joints involved in patients presenting with joint pain helps generate the differential diagnosis, and is a significant clue to the final diagnosis.
- Patients with a single, painful joint require a detailed physical examination to rule out evidence of systemic disease.
- Never perform arthrocentesis through an area of cellulitis or infection, as this can introduce infection into the joint.
- The white blood cell count of synovial fluid analysis must be interpreted cautiously, as overlap exists between etiologies.
- Pain caused by synovitis should neither be underestimated nor undertreated.

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33 Low back pain

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Scope of the problem

Low back pain affects up to 80% of the general population at some time during their lives, and is second only to the common cold as a reason to see a physician. Most patients have no serious underlying disease and are termed "uncomplicated." A few patients will have life-threatening or potentially disabling conditions requiring emergent intervention. The process of differentiating those with serious disease from the vast majority of patients with uncomplicated back pain can be challenging.

Anatomic essentials

Acute low back pain refers to pain, stiffness or tension felt in the lumbosacral spine and paraspinal areas. The pain may originate from lumbosacral structures such as bones (lumbar vertebrae, sacrum and coccyx), intervertebral discs, joints (facet, sacroiliac), soft tissues (muscles, tendons, ligaments), vascular structures, or nervous tissue (spinal cord, nerve roots). Low back pain may also be referred from pelvic, retroperitoneal and abdominal structures due to shared innervation.

There are five lumbar vertebrae. Between each vertebral body lies a shock-absorbing intervertebral disc, which supports the vertebral column while allowing spinal mobility. The spinal nerves exit the spinal column through the intervertebral foramina, paired openings between vertebrae. Herniation of the intervertebral discs may compress the spinal nerves, producing *sciatica* (a sharp or burning pain that radiates down the posterior or lateral leg beyond the knee). Ninety-five percent of disc herniations occur at the L4–5 and L5–S1 disc spaces, leading to radicular pain and motor and/or sensory loss in the L5 and S1 dermatomes. Understanding the lumbar dermatomes and myotomes is important for following the progression or resolution of neurologic symptoms, as well as effective documentation (Table 33.1).

The spinal cord is housed within the vertebral column, and surrounded by the dura mater and a series of potential spaces (where infection and tumors may seed). The adult spinal cord ends at the L1–L2 junction, giving rise to the *cauda equina* (horse's tail), a mass of spinal nerve roots. Compression of the cauda equina can result in urinary incontinence, saddle anesthesia (decreased sensation over the buttocks, perineum and proximal medial thighs) and bilateral sciatica. Compressive lesions above the cauda equina produce a cord compression syndrome.

The spinal canal is bounded by the vertebrae, intervertebral discs and ligamentum flavum. Degenerative changes in the spine and age-related thickening of the ligamentum flavum and can lead to *spinal stenosis*, which typically causes back and leg pain that is relieved by sitting or spine flexion.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 33.2). The key is to identify those who require immediate surgical evaluation or who suffer from serious causes (Figure 33.1).

History

How long have you had the pain?

Acute low back pain refers to pain lasting less than 6 weeks, whereas *chronic* low back pain typically lasts

Table 33.1 Selected dermatomes and myotome

Site of herniation	Nerve root	Dermatome	Myotome	Reflex
L5–S1	S1	Posterior calf Lateral foot	Plantar flexors	Achilles
L4–5	L5	Anterior calf Medial foot First web space ± great toe	Dorsiflexors	None
L3–4	L4	Medial calf and foot ± great toe	Quadriceps	Patellar
Cauda equina (massive central anterior prolapse)	Multiple	Any or all of the above, usually bilaterally Saddle[a] anesthesia	Multiple, including any or all of the above Bladder/bowel dysfunction	Any or all of the above Anal wink Cremasteric

[a] Saddle area is that part of the buttocks that sits on a traditional bicycle.

Table 33.2 Low back pain red flags

History	Concerning diagnosis
Symptoms for >6 weeks	Malignancy, spinal infection, ankylosing spondylitis
Age <18 years	Malignancy, spinal infection, congenital anomalies, spondylolysis, spondylolisthesis
Age >50 years	Malignancy, fracture, AAA
Neurologic complaints, bowel or bladder incontinence	Cauda equina syndrome, spinal cord compression
Sciatica	Disc herniation, malignancy, spinal infection, spondylolysis, spondylolisthesis
Syncope; acute back, flank or testicular pain	AAA rupture/expansion
Trauma, osteoporosis, chronic corticosteroid use	Fracture
History of cancer	Spinal metastases
Fevers, chills, night sweats, weight loss	Malignancy, spinal infection
Anticoagulation or coagulopathy	Cauda equina syndrome, spinal cord compression
Injection drug use, immune compromise	Infection
Night pain or unrelenting pain despite rest	Malignancy, spinal infection, ankylosing spondylitis
Examination finding	**Concerning diagnosis**
Fever	Malignancy, spinal infection
Pulsatile abdominal mass, diminished lower extremity pulses	AAA rupture/expansion
Anal sphincter laxity, saddle anesthesia	Cauda equina syndrome
Motor weakness	Cauda equina syndrome, spinal cord compression, herniated disc
Abnormal reflexes	Cauda equina syndrome, spinal cord compression, herniated disc
Positive SLR or CLSR	Disc herniation with radiculopathy
Percussion bone tenderness	Fracture, infection
Babinski sign present	Upper motor neuron disease (spinal cord compression)
Bilateral neurologic deficits	Cauda equina syndrome, spinal cord compression
Unilateral neurologic deficit	Disc herniation with radiculopathy

AAA: abdominal aortic aneurysm; CSLR: crossed straight leg raise; SLR: straight leg raise. Adapted from Della-Giustina, Kilcline BA, Denny M. Back pain: Cost-effective strategies for distinguishing between benign and life-threatening causes. *EM Practice* 2000;2(2).

more than 12 weeks. Ninety percent of low back pain episodes resolve within 6 weeks.

Where is the pain? Does it radiate?

Pain localized to the paraspinal musculature without dermatomal radiation most likely represents muscular strain. Radiation of pain in a dermatomal distribution, especially below the knee, suggests nerve root compression. Pain radiating to both legs can be seen with cauda equina syndrome. Pain radiating to the groin may be seen with abdominal aortic aneurysm (AAA) or renal calculi. Pain radiating to the abdomen should raise suspicion for a possible visceral cause or sacroiliac disease.

How would you describe the pain?

Deep somatic pain from structures such as the muscles, tendons and ligaments is often described as a deep, dull, aching or throbbing. Sciatica is often described as a sharp, shooting or burning pain radiating down the posterior or lateral aspect of the leg. The pain associated with a ruptured AAA is often acute, severe and constant.

How did the pain begin (sudden versus gradual)? Did you sustain trauma?

The sudden onset of pain suggests disc prolapse, crush fracture, or ruptured AAA, whereas the gradual onset of pain over years suggests degenerative disease. Pain onset over days to weeks is not reliable in distinguishing the cause. Major trauma, such as motor vehicle collisions or falls from a height, as well as minor trauma in at-risk patients (i.e., elderly, osteoporotic) should raise concern for vertebral fractures.

What makes the pain better or worse?

Mechanical (uncomplicated) back pain improves with rest and is exacerbated by movement and standing. Radicular pain (nerve root compression) is worse with coughing, sneezing, or changes in position. Constant pain unchanged by rest or position is worrisome for a serious cause, such as cancer or spinal infection. Similarly,

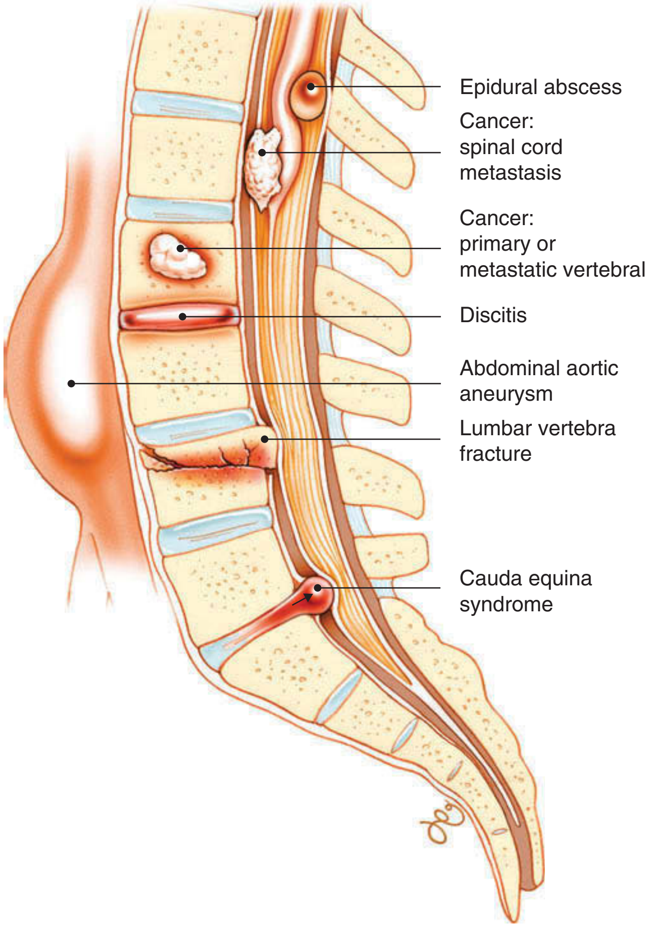

Epidural abscess
Cancer: spinal cord metastasis
Cancer: primary or metastatic vertebral
Discitis
Abdominal aortic aneurysm
Lumbar vertebra fracture
Cauda equina syndrome

Figure 33.1.
Serious causes of low back pain. 1. Epidural abscess; 2. Cancer: spinal cord metastasis; 3. Cancer: primary or metastatic vertebral; 4. Discitis; 5. Abdominal aortic aneurysm; 6. Lumbar vertebra fracture; 7. Cauda equina syndrome. © Chris Gralapp.

persistent pain despite adequate analgesia raises concern for serious disease. Unrelenting pain despite rest or pain that worsens at night raises concern for malignancy or infection. Morning stiffness and pain that improves with exercise suggests an inflammatory arthritis (e.g., ankylosing spondylitis), whereas pain worsening over the course of the day suggests osteoarthritis. Lower extremity pain exacerbated by walking or standing, and relieved by sitting or lying (*neurogenic claudication*) may be due to spinal stenosis.

Have you had any tingling, numbness, weakness, or loss of control of your bowel or bladder?

The presence of tingling, numbness or weakness suggests nerve root compression or spinal cord involvement. Bladder dysfunction or fecal incontinence in association with low back pain is very serious, suggesting cauda equina syndrome or higher spinal cord compression. Ask the patient about symptoms of urinary retention, increased frequency, or overflow incontinence.

Do you have a history of cancer or injection drug use?

The most powerful predictor of cancer as a cause of back pain is a current or prior history of cancer (especially breast, lung and prostate). Metastatic tumors of the vertebrae are 25 times more common than primary tumors, such as multiple myeloma. Metastases to the spinal column and cord may also result in epidural cord compression.

Injection drug use places patients at high risk for spinal infections, such as osteomyelitis, discitis, or epidural abscess. Other patients at increased risk for spinal infections include those with diabetes, chronic renal failure, alcoholism, or recent spinal surgery. Some authorities recommend that these patients be promptly and aggressively evaluated for spinal infections. Failure to rapidly diagnose and treat an expanding epidural abscess can result in permanent neurologic deficit.

Associated symptoms

The presence of fevers, chills, night sweats, or unexplained weight loss in patients with back pain suggests malignancy or infection. Urinary tract, pulmonary or gastrointestinal (GI) symptoms may identify a referred cause of low back pain.

Past medical

A previous history of back surgery or back problems should be elicited. Any patient with a history of chronic low back pain should be asked about previous motor, sensory or reflex abnormalities. A history of an aneurysm or aortic graft repair raises concern for a leaking AAA. A previous history of or risk factors for osteoporosis (i.e., female, age >50 years, steroid use) may predispose the patient to fractures. The dates of the last menstrual period and the possibility of pregnancy should be ascertained, as a normal or ectopic pregnancy can cause low back pain.

Details regarding the patient's profession, workman's compensation status and pending litigation are important if the injury occurred on the job. A family history of inflammatory arthritis such as ankylosing spondylitis could suggest a potential etiology.

Corticosteroids may increase the risk of spinal infections (due to immune suppression) and fractures (due to osteoporosis). Anticoagulation or thrombocytopenia may place the patient at risk for an epidural hematoma (especially following trauma, lumbar puncture (LP), epidural anesthesia, or spinal surgery). Spontaneous retroperitoneal hemorrhage in patients on warfarin or with bleeding disorders may present with low back pain.

Physical examination

The physical examination of the patient with low back pain is focused and intended to identify patients with possible serious etiologies.

General appearance

Cachectic patients are at risk for spinal infections from being immunocompromised and vertebral fractures from osteoporosis. Consider whether the underlying cause of cachexia is from injection drug use, human immunodeficiency virus (HIV), or an underlying cancer that has metastasized to the spine.

Vital signs

Fever suggests an infectious cause of back pain; however, many patients with spinal infections do not have a fever. Patients with AAA, sepsis, or ruptured ectopic pregnancy may present with hypotension. This finding may be transient and lead to syncope as a presenting complaint.

Abdomen

Gently palpate the abdomen for a pulsatile mass, present in 50% of patients with AAA. Also note any abdominal tenderness or findings that suggest a visceral etiology (e.g., appendicitis or ectopic pregnancy) with pain referred to the back or flank. For low back pain referred to the groin or buttocks, the hips should be assessed for pain and range of motion (ROM). Rotation of the hip is painful with osteoarthritis but not spinal stenosis.

Back

Examination of the back includes inspection, palpation, and assessment of ROM. The patient should be assessed for abnormal posture, spinal contour, or pelvic tilt. Such abnormalities may be structural or in response to pain or weakness. The skin overlying the back should be examined for bruising, swelling, or other lesions such as herpes zoster, which can cause lateralized back pain. Midline skin abnormalities may indicate underlying developmental spinal abnormalities.

The lumbar paravertebral muscles should be palpated for tenderness or spasm. Palpate each spinal vertebral process and the sacroiliac joints to identify areas of localized tenderness. Midline bony percussion tenderness is unusual in patients with uncomplicated back pain, and suggests a focal lesion such as a fracture, cancer, or infection. Pain on percussion of the costovertebral angles suggests the presence of kidney pathology such as pyelonephritis.

Active ROM is assessed and described by having the patient flex, extend and bend laterally to each side. Normal ROM is 90 degrees flexion, 15 degrees extension, and 45 degrees lateral flexion.

Rectal

This part of the examination should generally be done with a chaperone. Examine for perianal and saddle sensation by asking the patient "Does the skin in this region feel normal or numb?" Check the rectal tone by having the patient voluntarily contract their anal sphincter after inserting your gloved and lubricated finger. Saddle anesthesia and abnormal sphincter tone suggest a serious cause of low back pain, such as cauda equina syndrome.

Neurologic

The neurologic examination is the most informative part of the physical examination. The legs should be evaluated for sensory changes in a dermatomal distribution by comparing one side to the other (Figure 33.2). Similarly, motor deficits should be sought in a systematic manner, generally starting at the toes and moving to the hips. Each side should be compared against the other. Evidence of major motor weakness is suggested by abnormalities of knee extension (quadriceps weakness), ankle plantar flexion and eversion, and ankle dorsiflexion (footdrop). It is important to differentiate true neurologic weakness from pain-related motion avoidance. Documentation is best done with descriptive words rather than any of the variety of scoring systems. For example: "Left great toe dorsiflexion with mild weakness

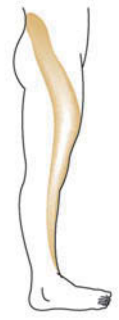
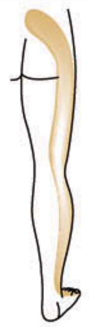
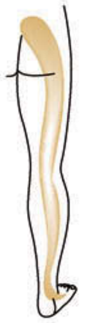
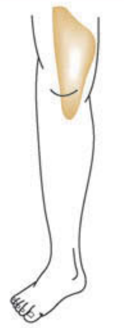
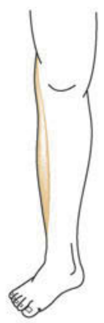
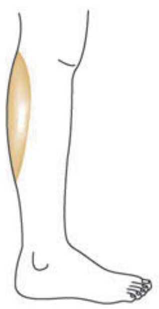
Nerve root	L4	L5	S1
Pain			
Numbness			
Motor weakness	Extension of quadriceps	Dorsiflexion of great toe and foot	Plantar flexion of great toe and foot
Screening examination	Squat and rise	Heel walking	Walking on toes
Reflexes	Knee jerk diminished	None reliable	Ankle jerk diminished

Figure 33.2
Testing for lumbar nerve root compromise. Reprinted from Bigos S, Bowyer O, Braen G, et al. *Acute Low Back Problems in Adults*, Clinical Practice Guideline No. 14, AHCPR Publication No. 95–0642, Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services, Rockville, MD, December 1994.

compared with right side," is better than: "2+/3 or 4/5 or + + + left great toe." Muscle atrophy can be detected by comparing the circumference of the thighs or calves.

Check for an abnormal Achilles reflex (predominantly the S1 nerve root) and patellar reflex (predominantly the L4 nerve root). As reflexes typically diminish with age, it is critical to identify asymmetry. The presence of clonus, hyperreflexia, or upgoing toes in response to plantar stimulation indicates an upper motor neuron lesion, such as spinal cord compression.

Assessing the patient's gait is very important. Check for difficulty with heel-walking (ankle and toe dorsiflexors innervated by the L5 and some L4 nerve roots) or toe-walking (calf muscles, mostly the S1 nerve root). If the patient cannot heel-walk, ask him to squat; inability to do so indicates an L4 abnormality. Inability to ambulate despite adequate analgesia raises concern for serious pathology. A patient who cannot walk should not be discharged from the emergency department (ED), unless this situation is chronic and unchanged.

Clinical tests for radiculopathy

The straight leg raise (SLR) test is the standard method of eliciting clinically significant nerve root compression (Figure 33.3). With the knee extended, the leg is elevated until pain is elicited. The SLR test detects tension on the L5 and/or S1 nerve root. The SLR test reproduces posterior or posterolateral leg pain by stretching nerve roots irritated by disc herniation. Pain that is felt below the knee at less than 70 degrees of straight leg elevation, aggravated by dorsiflexion of the ankle and relieved by ankle plantar flexion or external limb rotation, is highly suggestive of tension on the L5 or S1 nerve root from disc herniation. The reproduction of back, buttock or thigh pain with SLR testing does not indicate significant nerve root tension. The SLR is often negative in patients with spinal stenosis.

The crossed straight leg raise (CSLR) test is positive if SLR testing of the opposite (asymptomatic) limb elicits sciatic pain in the symptomatic limb. A positive CSLR is a stronger indication of nerve root compression due to disc herniation than pain elicited from SLR testing in the symptomatic limb. The reverse straight leg raise (RSLR) test or *femoral stretch test* detects tension in the L2–L4 nerve roots. With the patient prone, pain elicited in the anterior thigh with extension of the hip or flexion of the knee confirms a positive RSLR test.

The sitting knee extension test (SKET) also tests sciatic nerve root tension (Figure 33.4). The patient with

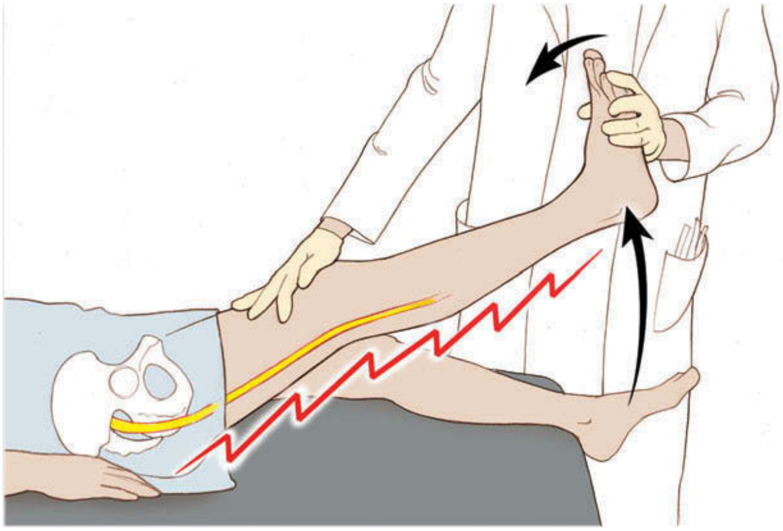


Figure 33.3
Straight leg raise (SLR) test. SLR instructions include: (1) Have the patient lie as straight as possible on a table in the supine position. With one hand placed above the knee of the leg being examined, exert enough firm pressure to keep the knee fully extended. Ask the patient to relax. (2) With the other hand cupped under the heel, slowly raise the straight limb. Tell the patient, "If this bothers you, let me know and I will stop." (3) Monitor for any movement of the pelvis before complaints are elicited. True sciatic tension should elicit complaints before the hamstrings are stretched enough to move the pelvis. (4) Estimate the degree of leg elevation that elicits complaint from the patient. Then determine the most distal area of discomfort: back, hip, thigh, knee, or below the knee. (5) While holding the leg at the limit of straight leg raising, dorsiflex the ankle. Note whether this aggravates the pain. Internal rotation of the limb also increases the tension on the sciatic nerve roots. © Chris Gralapp.

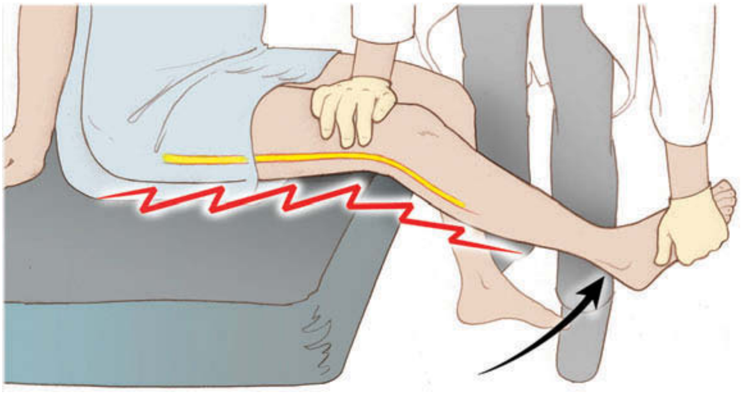


Figure 33.4
Sitting knee extension test (SKET). SKET instructions include: With the patient sitting on a table, both hip and knees flexed at 90 degrees, slowly extend the knee as if evaluating the patella or bottom of the foot. This maneuver stretches nerve roots as much as a moderate degree of supine SLR. © Chris Gralapp.

significant nerve root irritation tends to complain or lean backward to reduce tension on the nerve. A negative SKET in a patient with a positive SLR is inconsistent and raises concern for malingering.

Skin

Search for signs of peripheral vascular disease (e.g., skin or hair loss over the toes or distal lower extremities, ulcers, absent or weak distal pulses, or the presence of vascular grafts) to distinguish neurogenic from vascular claudication. Inspect the skin for lesions suspicious for malignancy (e.g., erythema nodosum), infection (e.g., hidradenitis), or needle marks. The presence of café au lait spots may indicate neurofibromatosis.

Cancer screening

As a general screen for cancer, examine the testicles and prostate in men, noting any irregularities. In women, examine the breast for lumps or skin findings that may be associated with cancer. Breast cancer affects women of *all* ages. Lymphadenopathy may be a sign of HIV disease, lymphoma, leukemia, or metastatic cancer.

Differential diagnosis

The etiologies of low back pain can be classified as mechanical or non-mechanical (Table 33.3). Although the differential diagnosis is broad, the classic error is to attribute the pain to a benign condition (i.e., lumbar strain) when a serious cause is responsible (Table 33.4). Renal colic is the most common incorrect diagnosis given to patients ultimately found to have AAA.

Diagnostic tests

The goal of diagnostic testing is to confirm or exclude serious pathology. Testing should be based on the results of a focused history and physical examination. The overriding principle is aggressive imaging of patients who have "red flag" findings on history or physical examination, while arranging outpatient follow-up for all other patients.

Post-void residual

A post-void residual should be checked in any patient with symptoms of urinary retention or incontinence. This volume may be measured by urinary catheterization or non-invasive ultrasound. A post-void residual volume >100–200 mL is highly suggestive of a neurogenic bladder due to cauda equina syndrome.

Laboratory studies

The routine use of the complete blood count (CBC), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) as screening tools is without merit. Numerous studies have shown that these tests lack both sensitivity and specificity when applied to the low back pain population. The ESR may be elevated in patients with malignancy, inflammatory conditions, or infection.

A urinalysis can screen for pyuria or hematuria. A urine pregnancy test can exclude pregnancy prior to

Table 33.3 Differential diagnosis of low back pain

	Non-mechanical etiologies	
Mechanical spine etiologies	**Spinal disorders**	**Visceral disorders**
Lumbar strain or sprain[a]	Neoplasia	Pelvic organs
Degenerative disease	Metastatic carcinoma	Prostatitis
Discs (spondylosis)	Multiple myeloma	Endometriosis
Facet joints[b]	Lymphoma or leukemia	Pelvic inflammatory disease
Diffuse idiopathic skeletal hyperostosis[c]	Primary spinal cord or vertebral tumor	Renal disease
Spondylolysis[b,c]	Retroperitoneal tumors	Nephrolithiasis
Spondylolisthesis[d]	Infection	Pyelonephritis
Intervertebral disc herniation	Vertebral osteomyelitis	Perinephric abscess
Spinal stenosis	Septic discitis	Vascular disease
Fracture	Paraspinal or epidural abscess	Abdominal aortic aneurysm
Traumatic	Herpes zoster (shingles)	Aortoiliac disease
Osteoporotic	Inflammatory arthritis	GI disease
Congenital disease	Ankylosing spondylitis	Pancreatitis
Severe kyphosis	Reiter's syndrome	Cholecystitis
Severe scoliosis	Psoriatic spondylitis	Perforated bowel
Transitional vertebrae	Inflammatory bowel disease	
Internal disc disruption (discogenic pain)[b]	Paget's disease	
	Scheuermann's disease (osteochondrosis)	

[a] Lumbar strain or sprain can be considered due to nonspecific (idiopathic) musculoligamentous etiology.
[b] The relationship between symptoms and objective findings for these conditions is not clearly established.
[c] Spondylolysis is a defect in the pars interarticularis without vertebral slippage.
[d] Spondylolisthesis is anterior displacement of one vertebra, typically L5, over the one beneath it.
Adapted from Atlas SJ, Nardin RA. Evaluation and treatment of low back pain: An evidenced-based approach to clinical care. *Muscle Nerve* 2003;**27**:265–84.

Table 33.4 Serious causes of low back pain

Disease	History	Examination	Comments
Abdominal aortic aneurysm	Elderly. Patients with vascular disease (e.g., diabetic, smoker). Pain may radiate to the flank or groin.	Feel for an abdominal pulsatile mass. Check for symmetric lower extremity pulses.	Best diagnosed with CT or US. Hematuria common, often mistaken for renal colic.
Cancer	Consider in age >50 years or history of cancer (even remote). Continuous pain not relieved with rest. Recent unexplained weight loss. Night sweats.	Look for spinal tenderness. Look for signs of cancer elsewhere (perform breast or prostate examination, feel for enlarged lymph nodes).	True emergency if spinal cord compression. X-rays may miss the diagnosis. The best tests are MRI or CT.
Infections: epidural abscess, vertebral osteomyelitis, discitis	Recent skin or urinary tract infection. Recent GI/GU manipulation. IV drug use. Sickle cell disease. Diabetes. Immunosuppresion.	Look for fever (absent 20% of the time). Localized spinal tenderness. Neurologic deficits are late findings.	Difficult to diagnose. If suspected, obtain MRI. X-rays may miss this diagnosis.
Spinal fracture	Acute onset. Trauma. Elderly. Osteoporosis. Corticosteroid use. Cancer.	Spinal tenderness. May have radicular pain.	Plain films are generally adequate for screening in any patient with a question of a fracture. CT useful for evaluating extent of the injury (encroachment into the spinal canal).
Inflammatory arthritis (e.g., ankylosing spondylitis)	Young adult males (<40 years) with pain and stiffness in the morning, not relieved when supine. Improvement with exercise. Family history.	Pain and stiffness throughout back and chest. May have decreased ROM depending on degree of advanced disease.	X-ray may be helpful. MRI better to look for inflammation of the sacroiliac joint. Serum HLA-B27 antigen.
Spinal cord compression or cauda equina syndrome	Urinary retention or dribbling (overflow incontinence). Fecal incontinence. Bilateral leg weakness.	Lower extremity weakness with hyporeflexia. Diminished rectal tone. Saddle anesthesia. Post-void residual <100 mL makes diagnosis unlikely.	Anything that compresses the spinal cord can produce this syndrome. MRI is the gold standard diagnostic test. CT provides useful bony information.
Spinal stenosis	Elderly patient with long history of low back pain. *Neurogenic claudication* (pain, numbness/tingling and weakness aggravated by standing or walking and relieved by sitting or lying).	Signs of peripheral vascular disease or hip osteoarthritis absent. Absence of pain when seated. Thigh pain with sustained lumbar extension (30 sec). Wide-based gait and/or positive Romberg sign with normal cerebellar exam.	Narrowing of the spinal canal. Diagnosed by CT or MRI.

CT: computed tomography; GI: gastrointestinal; GU: genitourinary; IV: intravenous; MRI: magnetic resonance imaging; ROM: range of motion; US: ultrasound.

radiography or as a potential etiology of low back pain (e.g., ectopic pregnancy).

Radiologic studies

Although plain lumbosacral spine radiographs may screen for vertebral fractures (Figure 33.5) and spondylolisthesis (which may occur in athletes involved in hyperextension sports like golf or gymnastics), these studies have limitations. Lumbosacral plain films cannot diagnose herniated intervertebral discs or spinal stenosis, and are frequently negative in patients with cancer or spinal infections. Routine lumbar spine films do not diagnose most serious causes of low back pain and could lead to potentially harmful radiation exposure. In one study, plain lumbosacral radiographs revealed unexpected findings in only 0.04% of patients under the age of 50 years. A complete lumbosacral plain film series exposes the patient to nearly 2,000 times the gonadal radiation as a single chest X-ray (CXR).

Emergent computed tomography (CT) or magnetic resonance imaging (MRI) is indicated in patients with a history, physical examination, or prior tests that strongly suggest a serious cause for back pain. These include cauda equina syndrome, AAA (Figure 33.6), vertebral fracture, infection, or tumor. Noncontrast CT is indicated when bony abnormalities (i.e., fracture, degenerative changes) are suspected, or when MRI is contraindicated. Radiologists favor MRI for suspected radiculopathy compared with CT, as it provides better resolution, no ionizing radiation, and superior ability to diagnose other inflammatory, malignant, or vascular

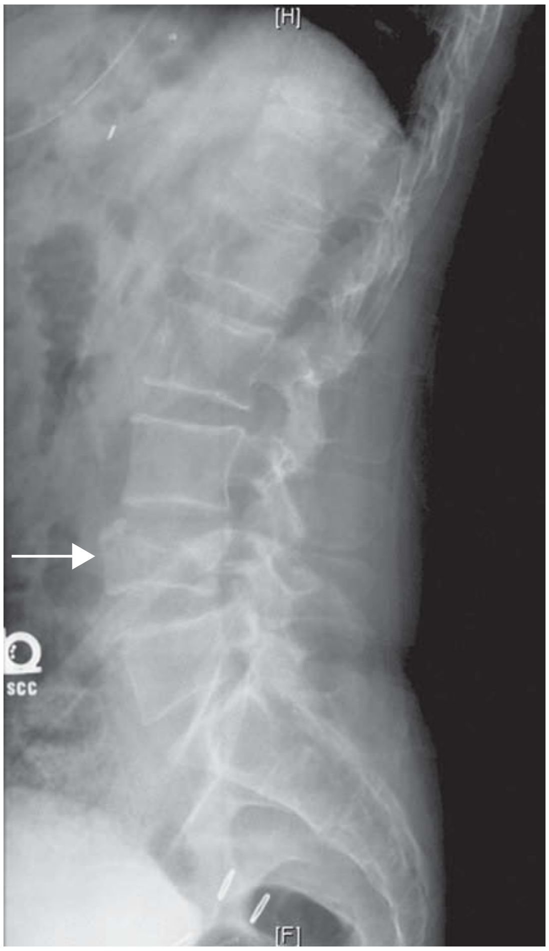


Figure 33.5
Lumbar compression fracture. Lateral radiograph of the lumbar spine demonstrating a compression fracture of the L4 vertebra, with probable retropulsion of bony fragments. Courtesy: Kathryn Stevens, MD.

conditions (Figure 33.7). For patients with sciatica likely due to a herniated disc or spinal stenosis, and who lack major neurologic abnormalities, imaging can be deferred 4–8 weeks, as the majority of these patients will improve with conservative therapy.

The clinical significance of any abnormalities detected on imaging studies must be carefully interpreted in the context of the patient's presenting symptoms. It is well known that plain films, CT and MRI may reveal abnormalities in asymptomatic patients, including lumbar disc degeneration, spondylosis (osteophytes), facet joint arthritis, lumbar disc herniation, spinal stenosis and spondylolisthesis (anterior displacement of the vertebra). This is especially true in older patients.

General treatment principles

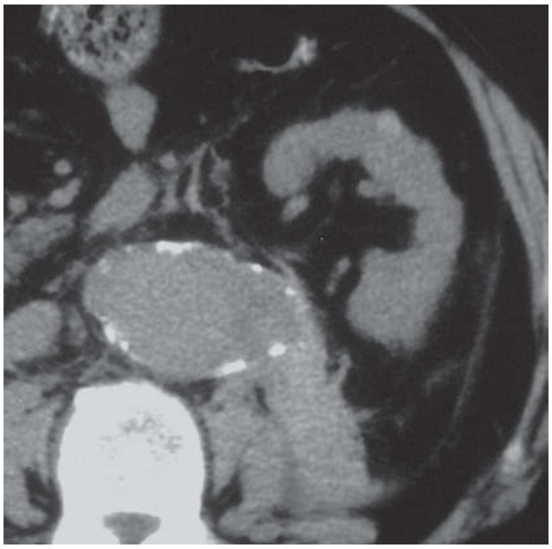


Figure 33.6
Noncontrast CT of ruptured abdominal aortic aneurysm (AAA). Note high attenuation left peri-aortic hemorrhage adjacent to calcified AAA. Courtesy: R. Brooke Jeffrey, MD.

In the majority of patients without red flags, the principles of therapy include analgesia and return to normal activity as quickly as possible, with lifting modifications and instructions on correct posture, lifting techniques and simple exercises. In contrast, table 33.5 provides initial treatment recommendations for several serious causes of LBP.

Analgesia

Analgesia should be adequate to allow patients to comfortably return to normal activity as quickly as possible. Analgesia should be given on a timed interval initially, then on an "as required" basis as the pain resolves. Hydrocodone/acetaminophen combinations are commonly used and generally effective: one or two tablets of 5/500 strength every 4 hours for short courses may be effective. For mild pain, acetaminophen alone in doses of 15 mg/kg provides excellent pain relief. Tylenol with codeine is generally a poor choice because it has many GI side effects and little efficacy over acetaminophen alone. Musculoskeletal back pain is rarely inflammatory; therefore, nonsteroidal antiinflammatory drugs (NSAIDs) such as ibuprofen do not offer any particular advantage. However, ibuprofen doses of 400–600 mg every 6 hours may provide some relief. Any prescription for NSAIDs should be for short courses, because GI ulceration and hemorrhage is an increasingly recognized side effect (especially in the elderly), even after relatively short exposure.

Muscle relaxants

There is no doubt that many patients with uncomplicated back pain have muscle spasm. However, there is controversy regarding the effectiveness of muscle relaxants. Although diazepam at doses of 2–10 mg every 4–6 hours may be effective, many other choices exist. The addictive potential of diazepam, other muscle relaxants and narcotics is real, but when used for acute pain and for short durations, the risk of addiction is small.

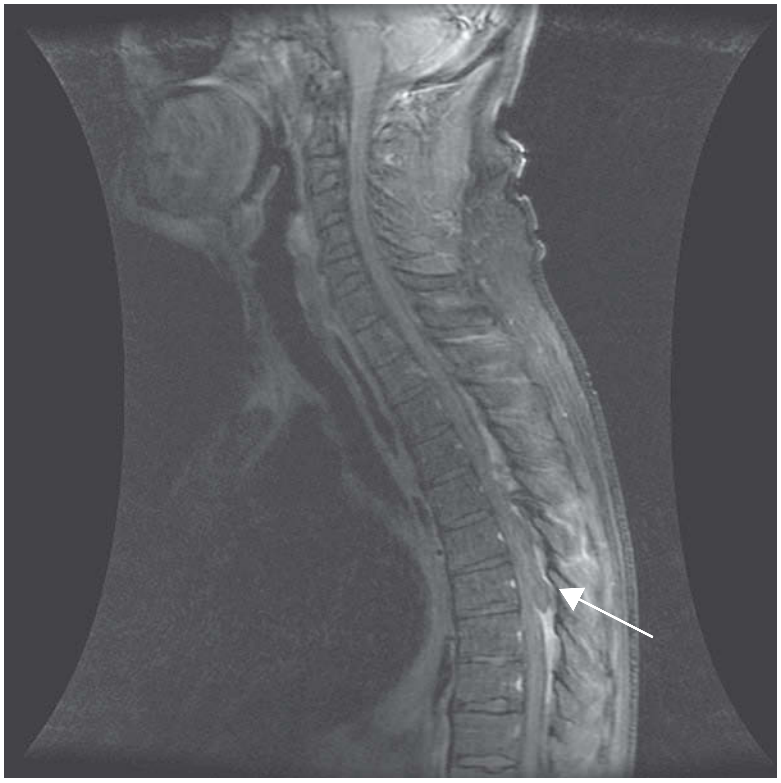


Figure 33.7
Epidural abscess. Sagittal T1-weighted MRI (post-gadolinium) reveals epidural enhancement and abscess collection (arrow). Courtesy: Mahesh Jayaraman, MD.

Table 33.5 Initial treatment of serious causes of low back pain

Disease	Initial treatment and consultation
Abdominal aortic aneurysm	Two large bore IVs. Six units of blood for crossmatch. Immediate vascular surgery consultation. Age-appropriate preoperative laboratories, including hematocrit and creatinine.
Cancer	Aggressive analgesia. CBC, CXR (looking for metastases), and CT or MRI to determine extent of disease.
Cauda equina or cord compression syndromes	Immediate neurosurgical consultation. Imaging with MRI or CT myelography. Dexamethasone. Age-appropriate preoperative laboratories.
Epidural abscess or spinal infection	Blood cultures × 3. IV antibiotics, generally a combination of anti-staphylococcal and aminoglycoside agents. Neurosurgical consultation. Age-appropriate preoperative laboratories.
Fractures	Appropriate analgesia. CT to define the extent of injury. Orthopedic or neurosurgical consultation (often institution-specific). Additional work-up needed if fractures are atraumatic.

CBC: complete blood count; CT: computed tomography; CXR: chest X-ray; IV: intravenous; MRI: magnetic resonance imaging.

Steroids

Although corticosteroids are prescribed for patients with musculoskeletal LBP (especially with radiculopathy), evidence supporting their use is lacking. However, all patients with suspected epidural compression syndromes (e.g., cauda equina syndrome, spinal cord compression) should be treated with steroids before obtaining confirmatory diagnostic tests.

Other therapies

Early ambulation and avoidance of activities that provoke pain should be encouraged. Patients should avoid prolonged sitting or standing and should get up at regular intervals to walk and stretch their backs. Bed rest should be limited to periods of severe pain, and never for more than a few days at a time.

Evidence recommending routine heat or cold therapy, physical therapy, or chiropractic manipulation is lacking. Because most episodes of acute low back pain resolve in 2–4 weeks, these therapies are best reserved for patients not responding to initial conservative management.

Prevention

Back exercises during the acute phase of low back pain should be avoided, as they may exacerbate symptoms. However, education on specific back strengthening exercises and proper lifting techniques may reduce subsequent episodes of pain or injury. In general, the best prevention is maintaining an appropriate body mass index (i.e., weight loss for most patients), exercise promoting cardiovascular fitness (without specific back exercises), and smoking cessation (this probably does not reduce back pain episodes substantially but is good for overall health and reduces the risk for lung cancer). Abdominal core muscle strengthening is helpful in minimizing low back pain as well.

Special patients

Elderly

Low back pain at the extremes of age should always cause concern. Elderly patients with low back pain can harbor multiple pathologies, including AAA and cancer. The most immediately life-threatening condition is AAA. In this age group, cancer is frequently diagnosed in patients who present with low back pain but without an obvious mechanism of injury.

Pediatric

In children, controversy exists about the seriousness of acute low back pain. Some authors note that it is a rare complaint, suggesting significant pathology, whereas others have demonstrated the opposite. Suspect serious pathology when children complain of nocturnal pain or limit their activity secondary to pain. Etiologies to consider in a patient younger than 10 years include infections (e.g., osteomyelitis, discitis) and tumors. In children older than 10 years, consider spondylolysis, spondylolisthesis, infection, tumor, herniated disc and ankylosing spondylitis. All children who remain undiagnosed following their ED visit need close follow-up and reevaluation.

Athletes

Athletes of all ages involved in hyperextension sports (e.g., baseball pitchers, golfers, gymnasts) can develop pars interarticularis fractures and subsequent spondylolisthesis. These injuries are more likely found in those involved at the competitive level. In such cases, referral to an orthopedist or neurosurgeon is recommended.

Pregnancy

Low back pain in pregnancy is an extremely common complaint. There are many potential causes, resulting from changes in maternal weight, center of gravity, posture, and ligamentous stability. Although pregnant patients are susceptible to serious causes of low back pain, it is generally true that most pain resolves following delivery. Due to the potential effects of ionizing radiation and medications on the growing fetus, additional consideration should be given prior to diagnostic imaging or therapy in these patients.

Malingering

The patient who embellishes medical history, exaggerates pain, or provides inconsistent responses on physical examination can be particularly challenging. Anatomically "inappropriate" physical signs of LBP may be thought of as "yellow flags" (Table 33.6). "Pain behaviors" such as amplified grimacing, distorted gait or posture, moaning, and rubbing of painful body parts may cloud medical issues and evoke responses from the clinician. Rather than interpreting these inconsistencies or pain behaviors as malingering, the clinician should view them as a plea for help or an attempt to enlist the clinician as an advocate. In patients with recurrent back problems, these behaviors and inconsistencies may simply be habits learned during previous medical evaluations. In working with these patients, the clinician should attempt to identify psychologic or socioeconomic pressures that might influence the presentation. The overall goal should always be to facilitate the patient's recovery and return to work or normal activities, without the development of chronic low back disability.

Table 33.6 "Yellow flags": Waddell's non-organic physical signs in low back pain

Patient overreaction during the physical examination
Inappropriate superficial tenderness
Pain with simulated axial loading
Inconsistent straight leg raise in the seated and supine positions
Non-dermatomal distribution of sensory loss and weakness
Inconsistency in formal motor testing and spontaneously observed behavior
Jerky movements or sudden giving way with motor examination

Adapted from Waddell G et al. Nonorganic physical signs in low back pain. *Spine (Phila Pa 1976)* 1980;5(2):117–25.

Disposition

Patients with uncomplicated low back pain can generally be discharged home. Patients with severe pain or social circumstances that make self-care difficult may require a short hospital or skilled nursing facility stay until pain is controlled. Discharge instructions should encourage the return to usual activities and the appropriate use of analgesia. Serious or worsening symptoms (i.e., fever, progressive unremitting pain, loss of bowel or bladder function, and progressive impairment of neurologic function such as sensory loss that expands or motor weakness that progresses up or down nerve roots) should prompt a return visit.

Patients with serious etiologies of low back pain should be admitted to the appropriate inpatient service with rapid consultation. AAA is a vascular emergency; the vascular surgeon should be called as soon as the diagnosis is suspected. Cauda equina syndrome, other spinal cord syndromes, and spinal infections require immediate neurosurgical consultation and possible surgical decompression. Although some lumbar spine fractures are stable, it is generally safest to have an orthopedic or neurosurgical consultant assist with treatment and disposition. Cancer is a serious cause of low back pain, although not all cancer patients require admission. Cancer patients who are candidates for discharge include those without evidence of neurologic involvement, whose pain is controlled, and for whom rapid outpatient follow-up can be obtained. This plan, of course, assumes that appropriate arrangements have been made to follow the patient and expedite further evaluation.

Pearls, pitfalls and myths

- Patients with life-threatening diagnoses (e.g., ruptured AAA) may present with low back pain (LBP).
- Patients with spinal cord threatening diagnoses (e.g., cauda equina) may present with LBP without neurologic deficits.
- LBP in a patient with a history of cancer is cancer-related until proven otherwise.
- LBP in an injection drug user is an infection (e.g., discitis, epidural abscess, osteomyelitis) until proven otherwise.
- Constant, unremitting pain is worrisome for a serious cause of LBP (e.g., malignancy, spinal infection).
- Patients with spinal infections may not have a fever.
- Failure to perform a thorough physical examination or differentiate true weakness from pain-related motion avoidance could lead to missed diagnoses.
- In patients with suspected cauda equina syndrome, check a post-void residual.
- Not all patients with LBP need imaging. A patient with LBP and no red flags does not need any diagnostic tests.
- Lumbar plain films provide significant radiation without diagnosing most serious causes of LBP.
- If you suspect a serious cause of LBP (e.g., AAA, cauda equina, fracture, infection or tumor), obtain an emergent CT or MRI, and consult a specialist.
- Never presume a patient with LBP is drug-seeking or malingering.
- Never discharge patients with LBP who can't walk or urinate on their own.
- Most LBP resolves in 4–6 weeks. Patients with a suspected herniated disc do not require imaging with MRI unless they fail to improve in that time frame.
- Patients with lumbosacral strain or sciatica should continue their routine activities as tolerated rather than resting in bed. They should avoid back exercises during their acute phase of pain.
- All children with LBP who remain undiagnosed after their ED visit need close follow-up.

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34 Pelvic pain

Peter G. Kumasaka, MD

Scope of the problem

Pelvic pain is a common emergency department (ED) condition and is the second most common gynecologic complaint. The pain may be acute or chronic, vague or defined, and may be referred to other regions of the body. Etiologies of pelvic pain may be reproductive, gastrointestinal (GI), vascular, or urinary. Although some causes of pelvic pain do not require emergent diagnosis or treatment, other diagnoses may pose an immediate or delayed threat to life (e.g., ectopic pregnancy), or may result in serious reproductive sequelae, such as infertility (e.g., salpingitis, ovarian torsion).

Anatomic essentials

Pelvic pain may originate from the reproductive organs (uterus, fallopian tubes, ovaries) or local organs, such as the appendix, ureters, bladder, sigmoid colon or rectum (Figure 34.1).

Visceral pelvic pain is usually caused by distention of hollow organs by fluid or gas, or capsular stretch of solid organs secondary to edema, blood, cysts or abscesses. It is less commonly the result of ischemia or inflammation. It is often the earliest manifestation of a specific disease process. Visceral pelvic discomfort is often poorly characterized and hard to localize, varying from a steady ache or vague pain to excruciating or colicky pain. Examples include distention of the fallopian tube in ectopic pregnancy, uterine contractions in dysmenorrhea, or stretch of the round ligament with advancing stages of pregnancy.

Parietal (somatic) pelvic pain is caused by irritation of the parietal peritoneum. This is typically the result of infection, chemical irritation, or other inflammatory processes. Parietal pain is usually sharp, knife-like and constant. In contrast to visceral pain, it can be localized to the dermatome directly above the painful stimulus. Parietal pain is responsible for the physical examination findings of tenderness to palpation, guarding, rebound and rigidity. Examples include salpingitis and endometritis.

Referred pain is defined as pain felt at a distance from the involved organ. Pelvic pain may be referred to the buttocks, back, groin, perineum, legs, or upper abdomen.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 34.1).

History

Clues gleaned from a careful history direct the examination and subsequent diagnostic testing. Patients should always be asked about their sexual history and the possibility of pregnancy. Personal questions should be asked in a non-judgmental manner and in strict privacy. Spouses, parents, friends or significant others should be politely asked to leave the room. This will encourage patients to

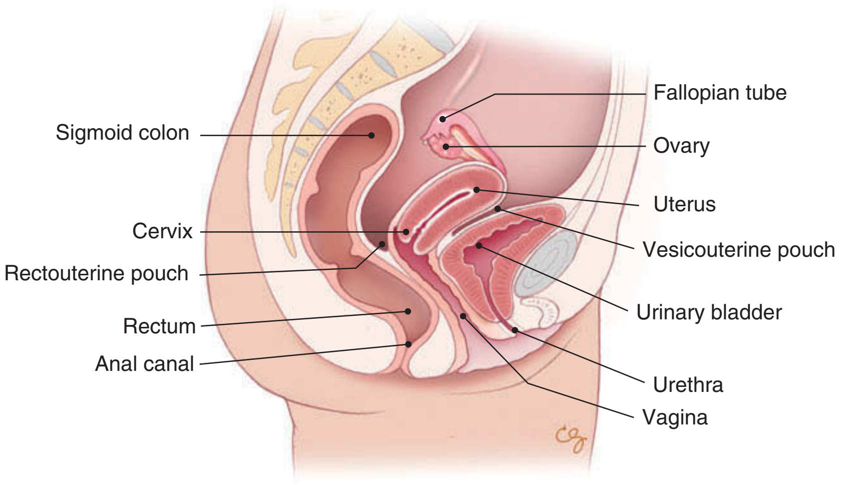

Figure 34.1
Pelvic anatomy (sagittal). © Chris Gralapp.

Table 34.1 Pelvic pain red flags

History	Concerning diagnosis
Sexually active, childbearing age, fertility drugs, artificial insemination, known to be pregnant (without confirmatory intrauterine pregnancy by US)	Ectopic or heterotopic pregnancy
Fever, vaginal discharge, multiple sexual partners, prior history of sexually transmitted infection	Salpingitis, PID, TOA, septic miscarriage, endometritis
Sudden onset pain	Ruptured ectopic pregnancy, ovarian torsion, ruptured ovarian cyst, appendicitis, diverticulitis, abscess, placental abruption
Elderly (older)	Ruptured abdominal aortic aneurysm (AAA), ovarian cancer, metastasis
Syncope	Ectopic pregnancy, vascular catastrophe, AAA
Recent uterine procedure (D&C, IUD insertion, delivery)	Endometritis, uterine perforation
Third-trimester pregnancy	Placental abnormality (previa or abruption), round ligament syndrome, arthritis, pubic diastasis, hernia, incarcerated hemorrhoid
Hypertension, sympathomimetic abuse in third trimester	Placental abruption
Crampy intermittent pain, rush of vaginal fluid	Imminent delivery
Examination finding	**Concerning diagnosis**
Hypotension, tachycardia, abnormal vital signs, shock	Ruptured ectopic pregnancy or AAA, trauma, infectious complication due to appendicitis, TOA, diverticular abscess, sepsis
Pelvic mass	Ectopic pregnancy, ovarian or uterine mass, ovarian cyst, TOA, ovarian torsion, incarcerated uterine fibroid (leiomyoma)
Peritonitis, cervical motion or uterine tenderness, cervical discharge	Ectopic pregnancy, TOA, PID, salpingitis, cervicitis, endometritis
Vaginal bleeding	Ectopic pregnancy, miscarriage
Foul-smelling vaginal discharge	Endometritis, cervicitis, septic miscarriage, PID, TOA
Hemorrhagic skin findings	Ruptured ectopic or AAA, pelvic fracture, intraperitoneal hemorrhage (often late finding)
Fetal presenting parts	Imminent delivery
Acute abdominal tenderness and gravid	Placental abruption

AAA: abdominal aortic aneurysm; D&C: dilation and curettage; IUD: intrauterine device; PID: pelvic inflammatory disease; TOA: tubo-ovarian abscess; US: ultrasound.

provide honest answers to questions. Even so, patients may still not be truthful in their responses.

How did the pain begin? How long have you had it? Have you had it before?

Sudden onset of pain may indicate a disease with significant morbidity and mortality, such as perforation of a hollow viscus, intrapelvic hemorrhage or vascular compromise (ovarian torsion). Chronic or recurrent pain can be associated with endometriosis, recurrent ovarian cysts, or a persistent ovarian mass. If the patient suffers from chronic pain, it is important to determine why she came in for help. The answer may reveal a new problem or a significant change of a chronic ailment.

Is there a pattern to the pain?

It is helpful to ascertain if the pain follows a cyclical pattern, including its relationship to the menstrual cycle. The pain of endometriosis usually accompanies the onset of menses, whereas pain associated with ovulation (Mittelschmerz) typically occurs during mid-cycle.

How would you describe the pain?

Sharp pain is more likely to be related to peritoneal irritation, whereas dull or crampy pain suggests contraction of an organ (e.g., uterus) or obstruction of a viscus (fallopian tube or ureter). Steady, progressive pain is associated with inflammatory or neoplastic causes. Burning pain of a chronic nature may indicate a neuropathic origin.

Where is the pain?

The ability to localize the pain may be extremely helpful. Lateral pelvic pain may be traced to the fallopian tube or ovary; central pelvic pain suggests a process involving the bladder or uterus. Diffuse pain can occur with pelvic inflammatory disease (PID) or peritonitis.

How severe is the pain?

Pain severity is subjective and not always helpful in directing the diagnostic evaluation. The patient complaining of “severe” pain whom you see laughing with a friend is clearly not the same as the grimacing, tearful patient. Pain can wax and wane, however, and different individuals assess, tolerate and respond to pain differently.

Does the pain radiate?

Pain radiating to the low back may indicate ovarian or uterine pathology; radiation to either flank may be due to ureteral problems. Radiation of pain to the rectum can be caused by blood or fluid collecting in the cul-de-sac.

Does the pain occur during intercourse?

Sudden onset of pain during intercourse *(dyspareunia)* often accompanies a ruptured ovarian cyst. Most other causes can be evaluated on an outpatient basis. These include gynecologic tumors, pelvic adhesions, adenomyosis, endometriosis, fibroids and uterine retroversion. Dyspareunia may also be the result of vaginal inflammation, cysts or abscesses of the vagina, or cervical lesions.

What is your sexual history?

Obtaining a good sexual history, including a history of sexually transmitted infections (STIs), birth control devices and sexual partners is imperative. Keep in mind that a monogamous patient does not mean a monogamous partner.

Are you pregnant? When was your last normal menstrual cycle? Describe any previous (especially abnormal) pregnancies?

The possibility of pregnancy may significantly alter the differential diagnosis, as well as raise concern for possible adverse effects of diagnostic testing or therapeutic intervention. All women of childbearing age should be assumed pregnant until proven otherwise, despite claims of celibacy, the use of birth control (including tubal ligation), or reports of “normal” menstrual periods. If pregnant, risk factors for ectopic pregnancy should be identified (Table 34.2).

Table 34.2 Risk factors for ectopic pregnancy and pelvic inflammatory disease (PID)

Risk factor	Ectopic pregnancy	PID
Previous ectopic pregnancy	Yes	No
History of PID	Yes	Yes
Tubal surgery	Yes	No
Prior abdominal surgery or inflammatory condition (e.g., appendectomy or inflammatory bowel disease)	Yes	No
Endometriosis	Yes	No
Multiple sexual partners	Yes	Yes
Presence of IUD	Yes	Yes
Assisted fertility	Yes	No

IUD: intrauterine device; PID: pelvic inflammatory disease.

Do you have any vaginal bleeding and/or discharge?

Describe any vaginal discharge. How does the discharge smell and look, and has this changed? How much bleeding are you experiencing? Can you quantify the amount of bleeding in terms of number of pads or tampons per hour? Is the bleeding occurring near the time of your menses? In a pregnant patient, ectopic pregnancy should be considered first, as this condition may be life-threatening. Other etiologies of vaginal bleeding in a pregnant patient include spontaneous miscarriage, threatened miscarriage or complications of the later stages of pregnancy, such as placenta previa or placental abruption. In a non-pregnant patient, vaginal bleeding may be associated with a number of conditions (e.g., dysmenorrhea, dysfunctional uterine bleeding, cervical or uterine cancer). Vaginal discharge may be associated with vaginitis, cervicitis and PID.

Are urinary symptoms present?

Ask about urinary frequency, urgency, dysuria and flank pain. Dysuria or other urinary tract symptoms may point to a simple cystitis or STI; however, these symptoms may occur from irritation of the bladder as a consequence of ectopic pregnancy or free peritoneal fluid. A history of frequent urinary tract infections or kidney stones (including a family history) should raise the possibility of these conditions.

Associated symptoms

Fever, nausea, vomiting, syncope

Presence of a fever suggests an infectious cause of pain, such as PID. Nausea or vomiting is more common with

GI processes, but may also be seen in ureteral stones and ovarian torsion. Syncope should raise the suspicion for blood loss (e.g., ruptured ectopic pregnancy or hemorrhagic ovarian cyst), volume depletion from any cause, or sepsis (e.g., PID or perforated appendix).

Past medical

A detailed medical history is imperative, and should include past pregnancies and associated problems. A history of abdominal or obstetric-gynecologic operations should be elicited. Systemic diseases also need to be reviewed. Recent procedures or surgeries raise concern for iatrogenic problems. Remote surgeries increase the likelihood of adhesions causing bowel obstructions. The diabetic or immunocompromised patient may be unable to mount a normal response to infection. More liberal laboratory testing and imaging studies should be used in evaluating these patients compared with patients having normal immune status.

Medications

Antibiotic (recent or current) or steroid use may influence a patient's presentation by masking infectious processes. Birth control pills do not preclude pregnancy. Many patients do not consider oral contraceptives or over-the-counter medications as "medications" when asked by a nurse or physician.

Social

Alcoholics are relatively immunocompromised, may be unreliable for follow-up, and often have coagulopathies. Smokers have an increased risk of cancer and vascular disease (e.g., mesenteric ischemia and aneurysms). Patients with drug addiction may be infected with the human immunodeficiency virus (HIV), may have lifestyles that put themselves at risk for STIs, and may be unreliable for follow-up.

Physical examination

General appearance

Begin your physical examination upon walking into the room, with inspection of the general appearance and overall impression of the patient and any visitors. Always start by evaluating the patient's ABCs (airway, breathing, circulation). If you think the patient is or may be unstable, seek additional help immediately. If the patient is speaking comfortably, her airway is protected and her breathing likely adequate. Feel the patient's pulse as you talk with her. Can you feel one? Is it thready? Is it fast? Does the patient look pale or diaphoretic? Is she mentating properly? Abnormal findings suggest that she is in shock. Does she feel warm? If her oral temperature is normal, consider checking a rectal temperature if you are concerned about a fever.

Vital signs

Attention to the patient's vital signs should be noted on initial assessment of the patient. Significant tachycardia or hypotension in a patient with vaginal bleeding, pelvic or abdominal pain should prompt aggressive measures, such as immediately starting intravenous (IV) fluids. Paradoxically, the patient may be bradycardic in the presence of intraperitoneal bleeding. When in doubt about the patient's stability, seek immediate assistance and additional help.

Abdomen

A detailed discussion of the abdominal examination is found in Chapter 10. Key aspects of the examination with respect to pelvic pain are described below.

Inspection

Is the abdomen distended or gravid-appearing? Is ecchymoses present? Grey Turner's sign (flank ecchymosis) or Cullen's sign (periumbilical ecchymosis) may indicate retroperitoneal or intraperitoneal bleeding. Check for the presence of hernias (umbilical or inguinal). If the patient is pregnant, assess for fetal movement.

Auscultation

Listen for abdominal bruits, bowel sounds, or fetal heart tones if the patient is pregnant.

Palpation

Can the pain be localized to a specific area? Gentle palpation starting in a nontender area or an area distant from the pain is recommended. You may be able to palpate masses (gravid uterus or distended bladder) or hernias. The patient may need to Valsalva or cough to make a hernia apparent. It is important to identify the presence of guarding (voluntary or involuntary), rebound, referred pain, or rigidity (indicative of peritoneal irritation).

Percussion

Gentle percussion may elicit tympany or distention, which may represent obstruction. Percussion tenderness over a specific area may identify a local area of inflammation or irritation.

Pelvic

Both male and female physicians are advised to have a chaperone present during the pelvic examination.

The chaperone should preferably be female, especially in the case of a male physician, and familiar with the examination techniques and the equipment needed. The patient should be allowed to empty her bladder or remove a tampon prior to the examination. Position and drape the patient in a manner that allows her to see the examining physician during the examination. She should also be made as comfortable as possible during the entire examination. Explain and describe each step of the examination and let her know what she may feel. Ask the patient to mention if the examination is different or more painful than previous pelvic examinations. Monitor the patient's reaction and behavior during the examination. Always be gentle and describe in advance what will be done and why.

Equipment

All necessary equipment should be set up beforehand, so the examination can be performed as efficiently as possible. You and the chaperone should be familiar with the equipment in advance, and check to make sure it is in working condition. Good lighting, a vaginal speculum of appropriate size, water-soluble lubricant, and materials for any anticipated bacteriologic studies, wet mount and pathology specimen collection should be available prior to the examination.

External genitalia

Inspect the external genitalia for evidence of inflammation, trauma, ulceration, discharge, swelling or nodules. A Bartholin cyst may present as a vulvar mass or swelling. A vesicular rash may represent genital herpes. Gently insert one finger into the patient's vagina and milk the urethra from the inside outward. Any discharge from the urethral orifice should be collected for culture.

Speculum

Warming the speculum by rinsing it with warm water prevents "surprise" and reduces discomfort for the patient. The speculum should be lubricated prior to insertion. Begin with the speculum in an oblique position, such that the handle hangs at 4 or 8 o'clock. It should then be slowly advanced, with gentle pressure posterior and inferior, while rotating the speculum so the handle hangs straight down. Take care to avoid the more sensitive anterior vaginal wall and urethra. Open the blades gently and note the cervix. If the cervix is not visible, try repositioning the speculum by slowly withdrawing it. This maneuver often results in the cervix "dropping into view."

The appearance of the cervix should be noted. In pregnancy, the cervix may have a bluish hue (Chadwick's sign). The cervix and the cervical os should be inspected for blood, polyps, masses, inflammation, ulceration, signs of infection, and the presence of the string of an intrauterine device (IUD). The presence and description of any cervical discharge should be noted. Is it bloody, frothy, purulent or mucoid? Samples of any discharge or tissue should be sent for appropriate testing.

In the presence of bleeding and early pregnancy, assess the patency of the cervical os. Gently insert the ring forceps into the os. If the forceps can be inserted more than a centimeter or so, the internal os may be open, indicating an incomplete or inevitable miscarriage. If a patient in her third trimester presents with vaginal bleeding, do *not* attempt to assess if the cervical os is open. Severe hemorrhage may result if placenta previa is present. Many third-trimester pregnancy patients who present with vaginal bleeding are managed in the operating suite with a double set up in case emergent Cesarean section is necessary.

Prior to and while slowly withdrawing the speculum, inspect the mucosa and walls of the vagina for signs of infection, trauma, lesions or foreign bodies, as well as blood, tissue or discharge.

Bimanual

Next, perform a gentle bimanual examination. Although the diagnostic accuracy of the bimanual examination has been questioned, every woman with pelvic pain should have a bimanual examination. Gently insert a lubricated middle and index finger into the patient's vagina. Palpate the anterior vaginal wall to differentiate bladder or urethral problems from gynecologic etiologies. Palpation of the posterior vaginal wall may identify rectal or sphincter pathology. With two fingers in the vaginal vault and the opposite hand on the pubic region, palpate the uterus for size, tenderness or masses. Place the cervix between the two fingers in the vault, and gently move it side to side. Exquisite pain or discomfort from this maneuver is known as *cervical motion tenderness* (CMT). Though CMT has classically been associated with PID, its presence is nonspecific and may represent irritation from an inflamed appendix, ruptured cyst or ectopic pregnancy. CMT is an insensitive finding, as it may be mild or absent in patients with PID.

Next, sweep the fingers to the adnexal regions. Start by examining the patient's nontender side. With the external hand, pull the fingertips firmly but gently downward and feel for adnexal masses and tenderness. Adnexal masses include ovarian cysts, tumors, or swollen fallopian tubes associated with PID or tubal pregnancy. In a patient with severe discomfort, adnexal palpation may be facilitated by the administration of an analgesic (e.g., morphine).

Lastly, a rectovaginal examination is performed with one finger in the vagina and one in the rectum. Sweep these fingers back and forth in a horizontal plane to feel for any masses, fullness or areas of discomfort. The rectovaginal examination allows for the evaluation of the posterior wall of the uterus, the posterior cul-de-sac (ovarian masses), and the ureterosacral ligaments (metastatic nodules or ectopic endometriosis).

Rectal

The rectal examination is commonly performed during or immediately following the pelvic examination. The rectal examination can reveal the presence of gross blood, nodularity or mass in the rectum. Ask the patient if the examination was painful or just uncomfortable.

Breast

Breast engorgement or tenderness may indicate pregnancy or a change related to menstrual cycle.

Lymph

Femoral and inguinal nodes should be evaluated for size and tenderness.

Differential diagnosis

Common gynecologic sources of pelvic pain are shown in Figure 34.2 and described in Table 34.3. Non-gynecologic sources are listed in Table 34.4.

Diagnostic testing

Laboratory studies

Pregnancy test

This test should be ordered for any female patient of childbearing potential presenting with pelvic pain. The history of birth control use, tubal ligation and abstinence should not preclude testing for pregnancy. Although infrequent, failure may occur with all forms of birth control. The incidence of ectopic pregnancy is increased when IUD or tubal ligation fails to prevent pregnancy. Quantitative beta-human chorionic gonadotropin (β-HCG) testing may be helpful when the presence of a normal intrauterine pregnancy (IUP) is in question. In early (normal) pregnancy, the level of β-HCG doubles approximately every 48 hours.

Progesterone level

Several studies have examined the use of a serum progesterone level to rule out ectopic pregnancy. One strategy involves using a two-tiered cut-off, with low levels (<5 ng/mL) associated with an abnormal pregnancy (but not specifically ectopic pregnancy); levels >25 ng/mL indicate a normal IUP with a high degree of specificity (levels less than this can be found in normal pregnancies, however). This still leaves the gray area between these cut-offs, where there is insufficient sensitivity and specificity to differentiate normal from abnormal pregnancy. In such cases, a common strategy is to combine the β-HCG level or transvaginal (endovaginal) ultrasound (US) result with the progesterone level. Currently, there does not appear to be consistent agreement about the utility of progesterone levels in the ED, and they may not be readily available.

Urinalysis

Virtually all patients with pelvic pain should have a urinalysis, although further confirmatory testing should be guided by clinical suspicion. Hematuria (presence of red blood cells) has been noted in patients with several conditions, including abdominal aortic aneurysm (AAA) and kidney stones. Pyuria (presence of white blood cells) can be present in inflammatory conditions adjacent to the bladder or ureter (e.g., appendicitis), as well as in urinary tract infections.

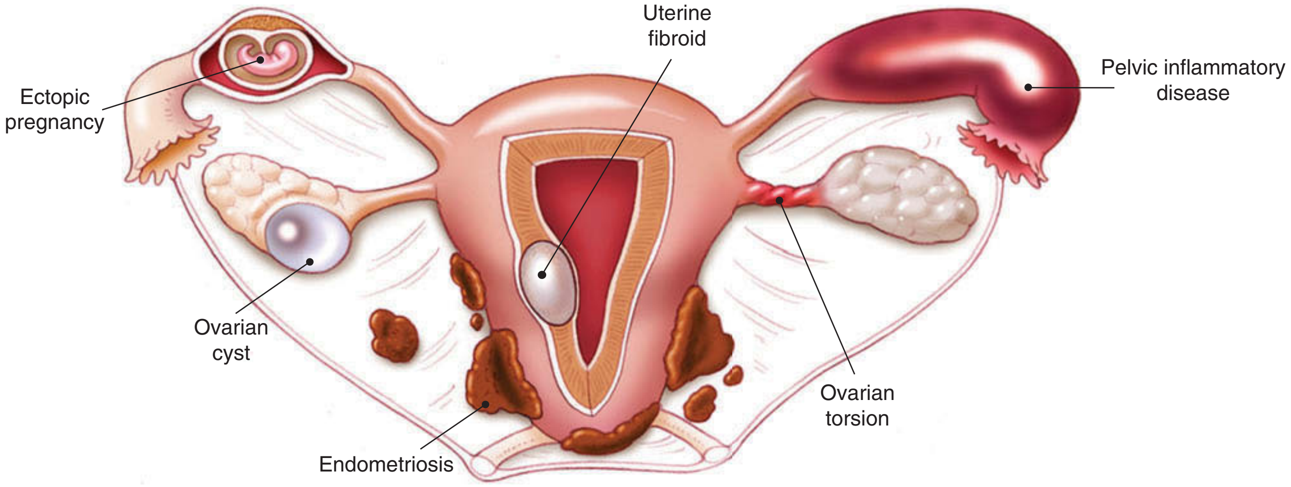

Figure 34.2
Common gynecologic sources of pelvic pain. © Chris Gralapp.

Table 34.3 Differential diagnosis of pelvic pain

Diagnoses	Symptoms	Signs	Work-up
Cancer – ovarian, uterine, cervical	Typically would not present with symptoms until late in course. Vague bloating/cramping, with or without abnormal bleeding. Dyspareunia.	Ascites. Pelvic mass.	Work-up to exclude other etiologies of pain. CT for staging. If ascites present, paracentesis for fluid analysis.
Degeneration of uterine fibroids	Acute, severe pain. Poorly localized. May have history of dysfunctional uterine bleeding/ fibroids.	Large uterus on pelvic examination.	US or CT. IV hydration. Pain control.
Dysmenorrhea	Crampy pain around and during menses. Cyclical occurrence.	Stable. Nontender or nonspecific tenderness.	Test for pregnancy; otherwise, no other testing necessary.
Ectopic pregnancy	Pain and/or vaginal bleeding within 10–12 weeks gestation (though dates can be off and misleading). Pain lateralizes to one of the adnexal regions, but can be anywhere in the abdomen. May become lightheaded or weak with significant intraperitoneal hemorrhage or vaginal bleeding.	Normal vital signs to significant tachycardia (may exhibit paradoxical bradycardia) and hypotension. Tenderness with or without mass in adnexal region. May have peritoneal signs and/or CMT.	β-HCG lower than expected for dates, needed for interpretation of US findings. Transvaginal US to evaluate for adnexal mass and presence/absence of IUP. Consider serum progesterone and other markers. Immediate Ob/Gyn consultation.
Endometriosis	Cyclical pain that can occur anywhere in the abdomino-pelvic region. Worse around the time of menses. Dyspareunia.	Pain localized to the sites of endometriomas, which may be palpable on pelvic examination. Otherwise, may have a normal examination.	No specific testing. Clinical history and response to hormonal therapy may be diagnostic. Laparoscopy can be diagnostic and/or therapeutic.
Endometritis	Fever, significant pelvic pain, with or without discharge. Nausea and vomiting. History may include recent delivery, uterine manipulation/procedure (including IUD, D&C).	Tender uterus. May be enlarged. Foul smelling, with or without bloody discharge.	Triple antibiotic therapy. Pain medication. Ob/Gyn consultation. May need D&C if retained products.
Labor	Cramping, intermittent pain, with or without rush of fluid (rupture of membranes).	Uterine contractions. Cervix examination shows dilation and effacement. Crowning. Fetal descent into canal.	Tocodynamometry shows regular contractions. Sterile pelvic examination. Vaginal fluid pH ~ 7; presence of *ferning*. Prepare for delivery. Follow fetal heart rate for evidence of distress. Transfer to labor and delivery if not imminently delivering.
Miscarriage/ threatened miscarriage	Crampy pain. Vaginal bleeding. May be passing tissue.	Rarely associated with significant vital sign abnormalities. Uterine tenderness.	US varies from an IUP to no evidence of pregnancy, to blighted ovum. Check Rh status if bleeding present.
Mittelschmerz	Vague, aching pain 2 weeks before menses.	Stable. Tender on examination, lateralizing to one adnexal region.	Test for pregnancy; otherwise, no other testing necessary.
Ovarian cyst – ruptured	Sudden onset of severe pain. May have had prior pain related to cyst (see below). Lightheaded/syncope if severe and hemorrhage is present. Localizes to one side. Corpus luteal cysts rupture around 6–8 weeks from last menses.	Hemodynamically stable, unless severe hemorrhage. Tachycardic, but may be paradoxically bradycardic. Unilateral tenderness on pelvic examination, and may have peritoneal signs; CMT.	IV fluids. Pain control. Type and screen/cross. Transabdominal US can assess for intra-abdominal fluid. Transvaginal US may demonstrate ruptured cyst.
Ovarian cyst – unruptured	Aching pain, typically cyclical with periods.	Hemodynamically stable, but may present with tachycardia due to pain. If palpable, examination will show ovarian fullness/mass.	US not necessary, unless to rule out other problems (e.g., torsion).

(*continued*)

Table 34.3 Differential diagnosis of pelvic pain (*cont.*)

Diagnoses	Symptoms	Signs	Work-up
Ovarian torsion	"Classic" symptoms: acute (59%), crampy (44%), severe pain in lower quadrant (90%). Patient will find the position of most comfort. May have had several similar occurrences. Nausea and vomiting.	Stable vitals. Pelvic examination reveals unilateral tenderness. Mass may or may not be noted (47%).	High index of suspicion needed, as may be missed. Doppler US to evaluate ovarian blood flow. Often a mass present (especially cyst) in the involved ovary, but presence of mass is not necessary. Ob/Gyn consultation for possible surgery to preserve ovary.
Pelvic congestion	Cyclical. About 7–10 days before menses. Worse with upright positioning. Often with musculoskeletal symptoms (e.g., back and leg pain).	Uterine tenderness.	No testing needed except to exclude other etiologies (e.g., PID). NSAIDs are treatment.
PID with or without TOA	Vaginal discharge, abdominal pain. Multiple sexual partners.	Fever. Hypotension may be present if septic. Discharge present. May have CMT.	IV fluids, cervical cultures, antibiotics and pain medication. US may be needed to assess for TOA. Ob/Gyn consultation if TOA.
Placental abruption	Third-trimester pregnancy. Crampy pain, usually accompanied by bleeding. History of trauma, hypertension, or cocaine use.	Vital signs may show cardiovascular collapse due to bleeding, but may be normal. Patient can exsanguinate into the uterus without any external evidence of bleeding (concealed hemorrhage). Enlarging abdomen. Evaluate for contractions and fetal distress. Petechiae or other evidence of DIC. Fetal heart rate may drop (indicating stress).	Hemoglobin may be normal to very low. As large amounts of bleeding consume up coagulation products, DIC may develop (elevated INR, D-dimer and fibrin degradation products; low fibrinogen). US shows hemorrhage between placenta and uterine wall, but may be normal. Immediate Ob/Gyn consultation.
Round ligament pain	Mid-pregnancy, aching, nonspecific pain. May be unilateral or bilateral.	Stable vitals. No bleeding or discharge. Relatively unremarkable pelvic examination.	No work-up needed except to exclude other etiologies of pain.
Septic miscarriage	Fever, severe pain.	Peritonitis. Foul smelling, bloody discharge.	US shows retained products. IV hydration through large-bore catheter. Broad-spectrum antibiotics. Immediate Ob/Gyn consultation for D&C.
Septic thrombophlebitis	Pain and fever (if develops from infection). Lower extremity swelling/pain indicating DVT. Any symptoms that may indicate PE. History may suggest DVT, especially surgical or puerperal.	Fever. May have CMT/uterine tenderness. Peri-adnexal masses (abscesses) may present with fullness on examination. Evidence of pelvic infection.	Contrast CT or US may help define infections or thrombosis (contrast CT). MRI has been used to document pelvic DVT. Heparin. IV antibiotics.
Uterine fibroids	Intermittent pain that is cyclical. Associated with heavy bleeding.	Pelvic examination reveals an enlarged uterus.	US not necessary, but will reveal discrete masses within the uterine myometrium.
Uterine perforation	Abdomino-pelvic pain. Fever if infection present. History of recent uterine procedure (e.g., D&C).	Peritonitis. Uterine/adnexal tenderness. Fever.	Antibiotics for infection. CT or US for evaluation of abdomino-pelvic pathology. Ob/Gyn consultation.

β-HCG: beta-human chorionic gonadotropin; CMT: cervical motion tenderness; CT: computed tomography; D&C: dilation and curettage; DIC: disseminated intravascular coagulation; DVT: deep venous thrombosis; INR: international normalized ratio; IUD: intrauterine device; IUP: intrauterine pregnancy; IV: intravenous; MRI: magnetic resonance imaging; NSAIDs: nonsteroidal antiinflammatory agents; Ob/Gyn: obstetrics/gynecology; PE: pulmonary embolism; PID: pelvic inflammatory disease; TOA: tubo-ovarian abscess; US: ultrasound.

Table 34.4 Non-gynecologic causes of pelvic pain

Gastrointestinal etiologies

- Appendicitis
- Mesenteric ischemia
- Bowel obstruction
- Diverticulitis
- Inflammatory bowel disease
- Volvulus
- Cancer
- Constipation
- Gastroenteritis/enteritis
- Mesenteric adenitis
- Colitis
- Hernias

Urinary tract etiologies

- Pyelonephritis
- Ureteral stone
- Cystitis
- Urethral syndrome

Toxic/metabolic etiologies

- Lead toxicity
- Acute porphyria
- Diabetic ketoacidosis

Vascular/other etiologies

- Aneurysm (aortic, iliac, femoral)
- Deep vein thrombosis (pelvic veins)
- Mesenteric vein thrombosis
- Sickle cell anemia

Complete blood count

Though the white blood cell (WBC) count is often ordered, many studies have shown that this test has limited clinical utility. The WBC count is only useful if it is extremely high or extremely low. The hemoglobin and hematocrit may be helpful in patients who are bleeding heavily or have evidence of hemodynamic compromise. These values may not reflect changes due to acute hemorrhage, and may not adequately reflect the patient's current circulating blood volume.

Basic metabolic panel

Electrolytes will only occasionally assist with the work-up of a patient with pelvic pain. Measuring the creatinine may be helpful for diagnostic testing purposes (e.g., contrast-enhanced computed tomography [CT] scan).

Cervical cultures

Cultures for *Gonorrhea* and *Chlamydia* as well as a wet prep for *Trichomonas*, *Gardnerella vaginalis* and yeast should be obtained when PID is suspected, or if a patient complains of vaginal discharge.

Other

Some patients will require blood products, and anticipating this by ordering appropriate laboratories is helpful. The critically ill patient may require O negative or type-specific uncrossmatched blood. Other patients may need a *type and crossmatch* if vital signs are unstable or blood transfusion is likely. A *type and screen* is indicated for patients who are bleeding and may need blood on short notice. *Rh factor* should be ordered for all patients who are pregnant and bleeding. Some institutions require checking the Rh factor on all pregnant patients with vaginal bleeding, even when the patient claims to know their Rh status or records are available confirming the patient's Rh status. This policy may reduce medical error in the rare instance of obtaining the wrong patient's record. Additionally, in some EDs, the misuse of someone else's identification card is not uncommon. Disseminated intravascular coagulation (DIC) profiles should be ordered for a patient with suspected placental abruption. Prothrombin time (PT), partial thromboplastin time (PTT), and international normalized ratio (INR) should be ordered for patients with bleeding disorders, those taking anticoagulants, in cases of suspected or potential DIC, or patients who are hemodynamically unstable and require massive blood transfusion.

Radiologic studies

Pelvic ultrasound

US can be very helpful in the work-up of pelvic pain. Transvaginal (endovaginal) US is the test of choice to evaluate for IUP, ovarian lesions, ovarian torsion and tubo-ovarian abscess (TOA) (Figure 34.3). Furthermore, it can provide information on the presence of free pelvic fluid, uterine masses, or retained products of conception.

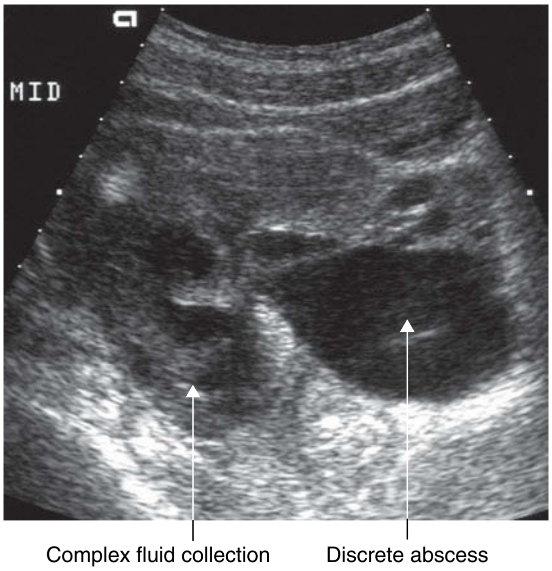

Figure 34.3
Tubo-ovarian abscess. Transverse sonogram reveals complex fluid collection posterior to uterus in the cul-de-sac. A more discrete abscess is located along left pelvic sidewall. Courtesy: R. Brooke Jeffrey, MD.

Of note, for first-trimester bleeding, the usefulness of US must be assessed in conjunction with the patient's clinical situation and laboratory values. Transvaginal US can typically detect the presence of an IUP at a β-HCG level of approximately 1,500 mIU/mL (around the fifth or sixth week by dates). This *discriminatory zone* varies by institution, and may lie between 1,000 and 2,000 mIU/mL. At levels above the discriminatory zone, a viable IUP should be visible by transvaginal US. Above this discriminatory zone, if an IUP is not visualized, then a non-viable pregnancy, ectopic pregnancy or inevitable miscarriage is likely.

Although rare, an ectopic pregnancy may coexist with an IUP (*heterotopic pregnancy*), especially if the patient has undergone assisted fertilization. The incidence of heterotopic pregnancy is typically cited at 1 in 30,000. However, recent data suggest it may be 1 in 3,000–7,000, and much higher (1 in 100) with infertility medications or in vitro fertilization. Clinical suspicion should guide the work-up in these patients, with early Ob/Gyn consultation.

At levels below the discriminatory zone, if an IUP is not seen on transvaginal US, then a normal pregnancy, threatened miscarriage or ectopic pregnancy are possible. The Ob/Gyn service should be consulted if clinical suspicion for an ectopic pregnancy is high. Otherwise, a reliable patient should be scheduled for a repeat β-HCG and close follow-up in 48 hours. A detailed explanation of indications to return immediately to the ED is essential (e.g., lightheadedness, syncope or near-syncope, heavy bleeding, fever, or severe pain). Patients should also be instructed to bring any tissue they pass (products of conception) to the ED or their Ob/Gyn provider.

Computed tomography

CT is another modality for assessing abdominal and pelvic structures. CT can identify ureteral stones, AAA, and other areas of inflammation (e.g., pelvic abscesses or appendicitis). Some CT studies require IV contrast, which may limit use in patients with renal failure or allergies to IV contrast. Oral and/or rectal contrast may be required as well (although less commonly needed), and its ability to pass through the GI tract may affect the timing and quality of the study.

Although CT utilization is increasing as a result of greater availability and improved sensitivity and specificity, US is generally considered the initial imaging study of choice in patients with pelvic pain. This is especially true in young or pregnant patients, as CT exposes the reproductive organs to significant amounts of radiation. US can delineate most pelvic inflammatory conditions, may demonstrate appendicitis, and can provide information regarding blood flow to the ovaries if ovarian torsion is a concern.

General treatment principles

Stabilization

As with any patient, assessment and stabilization of the ABCs are the first priorities of the emergency physician. This may require intubation, assisted ventilation, IV fluids, or the use of blood products.

Intravenous access/fluids

Two large-bore IVs should be placed in patients exhibiting hemodynamic compromise, or when a strong suspicion of significant hemorrhage (e.g., ectopic pregnancy) or sepsis (e.g., endometritis, TOA) exists. Patients unable to tolerate oral fluids and those who need to be kept NPO (if surgery is a possibility) may benefit from IV fluids. Furthermore, patients who may require multiple doses of parenteral medication, blood and blood products, or IV contrast require IV access.

Pain relief

Relieving pain is one of the primary responsibilities of emergency physicians. Narcotic administration does not interfere with making the correct diagnosis. Withholding pain medications is inhumane, and this practice should be condemned. Other medications such as nonsteroidal antiinflammatory drugs (NSAIDs) may relieve prostaglandin-mediated pain, such as that created by smooth muscle contraction (e.g., uterine cramping). If the patient is unable to tolerate fluids, or needs to be NPO, ketorolac may be useful. Studies have not shown a faster or stronger effect from intramuscular (IM) ketorolac injection versus oral ibuprofen.

Antibiotics

The diagnosis, patient condition and other patient-related issues guide antibiotic coverage and routes of administration. Factors influencing the choice of therapy include the patient's condition, comorbid conditions, likelihood of compliance, local resistance patterns, pregnancy status, age, known medication allergies, and previous successful (or unsuccessful) treatment regimens if the condition is recurrent.

Antibiotic selection for treating STIs should cover *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. For simple cervicitis, one-time oral regimens are available and often given in the ED, which helps assure compliance. PID can be managed on an outpatient basis if the patient's pain can be controlled, peritoneal signs are absent, adequate follow-up is ensured and the patient can tolerate oral medications. Otherwise, parenteral antibiotics and admission are indicated. Unlike simple cervicitis, PID appears to be a polymicrobial disease. Organisms other than *N. gonorrhoeae* and *C. trachomatis* may cause PID, including streptococci, Gram-negative organisms, anaerobes and *Trichomonas*. A choice of broad-spectrum antibiotics or combination of antibiotics such as a third-generation cephalosporin plus doxycycline, or fluoroquinolone (not ciprofloxacin) plus metronidazole is appropriate. Inpatient parenteral therapy is indicated if the patient is immunocompromised, pregnant or has failed outpatient therapy (Table 34.5).

Other pelvic infections, such as endometritis, require broad coverage of Gram-negative, Gram-positive and anaerobic organisms. Broad-spectrum antibiotics include regimens such as a synthetic penicillin/beta-lactamase

Table 34.5 Antibiotic selection for pelvic infections

Diagnosis	Pathogen	Antibiotic
Bacterial vaginosis	*G. vaginalis* Generally a disruption of normal vaginal flora	Metronidazole 500 mg PO BID × 7 days *or* Metronidazole gel 0.75%, 1 applicator intravaginally QD × 5 days *or* Metronidazole 2g PO × 1 dose *or* Clindamycin cream 2%, 1 applicator (5 gm) intravaginally QHS × 7 days **Alternative therapy:** Clindamycin 300 mg PO BID × 7 days *or* Clindamycin ovules 100 mg intravaginally QHS × 3 days *or* Tinidazole 2 g PO QD × 2 days *or* Tinidazole 1 g PO QD × 5 days
Mucopurulent cervicitis	*N. gonorrhoeae/C. trachomatis* Note: treat for BOTH organisms in area of high prevalence N. gonorrhoeae (>5%) or confirmed N. gonorrhoeae alone	***C. trachomatis:*** Azithromycin 1 gm PO × 1 dose *or* Doxycycline 100 mg PO BID × 7 days **Alternative therapy:** Ofloxacin 300 mg PO BID × 7 days *or* Levofloxacin 500 mg PO QD × 7 days *or* Erythromycin base 500 mg PO QID × 7 days *or* Erythromycin ethylsuccinate 800 mg PO QID × 7 days ***N. gonorrhoeae:*** Ceftriaxone 250 mg IM × 1 dose *or* Cefixime 400 mg PO × 1 dose *PLUS treatment for Chlamydia*
Pelvic inflammatory disease (oral regimen)	*N. gonorrhoeae/C. trachomatis, M. hominis,* anaerobes, *Enterobacteriaceae*	Ceftriaxone 250 mg IM × 1 dose *or* Cefoxitin 2 g IM × 1 dose with Probenecid 1 g PO × 1 dose *Plus* Doxycycline 100 mg PO BID × 14 days *Plus/minus* Metronidazole 500 mg BID × 14 days (if suspicion exists for *Trichomonas*)
Pelvic inflammatory disease (parenteral regimen)	*N. gonorrhoeae/C. trachomatis, M. hominis*, anaerobes, *Enterobacteriaceae*	Cefotetan 2 gm IV Q 12 hrs *or* Cefoxitin 2 g IV Q 6 hrs *plus* Doxycycline 100 mg PO/IV Q 12 hrs
Pelvic inflammatory disease (alternative parenteral regimen)		Clindamycin 900 mg IV Q 8 hrs *plus* Gentamicin 2 mg/kg IV load, then 1.5 mg/kg IV Q 8 hrs *plus* Doxycycline 100 mg IV/PO Q 12 hrs
Pelvic inflammatory disease (second alternative parenteral regimen)		Ampicillin-Sulbactam 3 g IV Q 6 hrs *plus* Doxycycline 100 mg IV/PO Q 12 hrs
Trichomoniasis	*T. vaginalis*	Metronidazole 2 g PO × 1 dose *or* Tinidazole 2 g PO × 1 dose **Alternative therapy:** Metronidazole 500 mg PO BID × 7 days

Note: As of 2007, CDC guidelines no longer recommend quinolones for the treatment of gonorrhea and associated conditions, such as PID, due to resistance in the United States. Local resistance patterns vary.

BID: twice a day; *C. trachomatis: Chlamydia trachomatis; G. vaginalis: Gardnerella vaginalis*; IM: intramuscular; IV: intravenous; *M. hominis: Mycoplasma hominis; N. gonorrhoeae: Neisseria gonorrhoeae*; PO: per os; QD: once daily; QHS: every night at bedtime; QID: four times a day; TID: three times a day; *T. vaginalis: Trichomonas vaginalis.*

inhibitor (e.g., ticarcillin/clavulanate) plus doxycycline, or clindamycin plus a third-generation cephalosporin.

Blood products

A blood transfusion is indicated in a patient who is hemodynamically unstable from hemorrhage and does not respond to crystalloid boluses. A type and screen or crossmatch should be sent, depending on clinical urgency. Fresh-frozen plasma (FFP) may be given to a patient with DIC, which can develop in septic patients and those with severe hemorrhage from placental abruption. FFP may be needed for patients on warfarin. Platelet transfusion may be needed for thrombocytopenic states such as idiopathic thrombocytopenic purpura (ITP), although consultation with Ob/Gyn and/or hematology is recommended.

Rh immune globulin (Rho-GAM)

Rho-GAM is indicated for any Rh-negative pregnant patient presenting with vaginal bleeding, suspected or proven placental abruption, and trauma with the potential for fetal–maternal transfusion or proven fetal–maternal transfusion (by *Kleihauer–Betke* testing). Dosing is based on gestational age. Low dose Rho-GAM (MicRho-GAM 50 mcg) is given for miscarriages, pregnancy, or ectopic pregnancy termination to Rh-negative mothers when between 0 and 12 weeks gestational age. Beyond 12 weeks and for threatened miscarriage at any gestational age, full dose Rho-GAM (300 mcg) is given. In the situation of fetal–maternal hemorrhage, 300 mcg is given for every 15 mL of fetal red blood cells (or 30 mL of fetal whole blood) that the mother has been exposed to based on Kleihauer–Betke test results.

Medications

Other than analgesics and antibiotics, other medications include antiemetics for intractable or problematic nausea and vomiting. Methotrexate can be used in selective patients for the treatment of ectopic pregnancy, in consultation with Ob/Gyn. Misoprostol has been used in patients with incomplete or inevitable miscarriages.

Special patients

Pediatric

Emergency physicians must diligently search for a cause of pelvic pain in a young patient. Due to social, cultural and parenteral pressures, these patients and their parents may be scared or embarrassed to answer questions about sexuality and genitalia, much less undergo a pelvic examination. All children who are perimenarchal or sexually active should be assumed pregnant until proven otherwise.

Prepubertal children are most likely to present with pelvic pain secondary to vaginal foreign body, urinary tract infection or sexual abuse. A pelvic examination is still indicated in these patients to assess for evidence of abuse. A modified approach can be used, but a full pelvic examination may be required under procedural sedation or general anesthesia. Proper collection of samples and precise documentation are required for evidence if a criminal case is suspected. Most local EDs and police departments have developed a sexual assault kit for these purposes. Child protective services should be consulted in cases of suspected or proven abuse or STI. Ob/Gyn and/or sexual assault teams may be involved according to local practice, policy and availability.

All states allow minors to consent to evaluation and treatment of STIs and drug abuse without parental consent. The legal status of the *emancipated* and the *mature minor* vary from state to state. These statutes allow these minors certain rights, including seeking and consenting to medical care without the authorization and notification of their parents. Any emergent care deemed necessary takes precedence over parental rights.

Geriatric

Older patients may not be willing or able to communicate their problems. Nursing home workers, paramedics and family, as well as the patient's medical chart may be extremely helpful, particularly if they provide information on any associated signs or symptoms. In general, the older patient should be considered relatively immunocompromised, and more likely to have other comorbid problems that can affect both the presentation and diagnostic evaluation.

Older patients with pelvic pain need a complete gynecologic examination. Often, the geriatric patient will not present with "classic" symptoms of an acute inflammatory process such as appendicitis or diverticulitis. They may not develop a fever, mount an increased WBC count or have peritoneal signs. Older patients with pelvic or abdominal pain are more likely to have significant pathology. As a result, the morbidity and mortality of abdominopelvic complaints in the elderly patient is significant.

The physiologic drop in estrogen levels with age can cause vaginal irritation and thinning of the mucosa. On the other hand, unopposed estrogen levels secondary to decreased progesterone can lead to endometrial hyperplasia and possibly endometrial cancer. Uterine prolapse is common, as are cystoceles, rectoceles and urethroceles. Furthermore, a normal ovary should not be palpable five years after menopause; any enlargement is abnormal and mandates further investigation.

Chemically dependent or impaired

Patients who are intoxicated can present with pain that may be overlooked. Their ability to provide a coherent history may likewise present problems for clinicians. Despite these issues, such patients may be at higher risk to develop certain diseases (e.g., STIs, ectopic pregnancy) and are less likely to schedule and keep follow-up appointments, purchase or take their medications, or return if the problem worsens.

Assisted reproductive therapy

Today, more patients are undergoing some form of assisted reproductive therapy (ART). This has raised the incidence of *ovarian hyperstimulation syndrome* (OHSS), as well as the presence of multiple gestations. OHSS can be mild to severe. In its mild form, patients may feel some distension and pain. When severe, the patient may present with extremely large ovaries (>10 cm) and large amounts of free fluid in the abdomen (ascites). Electrolyte abnormalities, hypotension, pleural effusions and oliguria may ensue.

The use of ART has increased the prevalence of multiple gestation pregnancies. Along with the rise in multiple gestations, the risk for *heterotopic pregnancy* has greatly increased. In these patients, the presence of an ectopic pregnancy should be considered even if an IUP is confirmed with US. Consultation with Ob/Gyn is appropriate.

Communication and cultural barriers

Patients who are unable to communicate clearly (e.g., language barrier or mentally handicapped) deserve a thorough and sensitive investigation. Even a mentally challenged patient can contract STIs and develop ectopic pregnancies. The non–English-speaking patient may also present more of a challenge due to cultural issues or anatomic problems (e.g., female circumcision).

Disposition

Obstetric/gynecologic consultation

Ob/Gyn should be consulted for all ectopic pregnancies. If a definitive IUP is not identified and the possibility of an ectopic or heterotopic pregnancy exists, consultation should also occur. Ob/Gyn consultation is prudent for any pregnant patient receiving ART. Definitive follow-up should be arranged for a repeat evaluation and β-HCG determination within 48 hours. Other cases requiring consultation include ovarian torsion, TOA, ruptured ovarian cyst with hemodynamic compromise, placental abruption, placenta previa (these patients typically do not have pain), and active labor. All of these patients generally require admission or observation. Patients also require admission for significant pelvic infections, uncontrolled pain, and inability to tolerate fluids or oral medications.

Ob/Gyn should be contacted regarding a patient with problematic ovarian cysts, follow-up of pelvic infections, any complications of pelvic procedures, and ongoing or threatened miscarriage. Heavy menstrual bleeding, especially causing symptomatic anemia, should result in Ob/Gyn consultation. The patient may not need to be seen immediately, but input from the Ob/Gyn consultant should be obtained and follow-up arranged. When follow-up is uncertain, or the patient is unreliable, input from the Ob/Gyn consultant should be sought.

Admission

Admission is the general rule for acute life- or fertility-threatening diseases. The patient who is at risk for failing outpatient therapy due to noncompliance, chemical dependency, social circumstances, comorbid diseases, or associated conditions (e.g., vomiting) should be deemed a candidate for inpatient treatment. Any patient involved in sexual assault or abuse should not be sent home until all appropriate police and support services have been involved, and the patient feels safe in her environment.

Discharge

Many patients may be managed on an outpatient basis with close Ob/Gyn follow-up and strict instructions to return for certain indications. Returning to the ED is a viable alternative if a patient is unable to follow up in the Ob/Gyn clinic. Losing a patient to follow-up is unacceptable. For example, outpatient medical treatment of selected ectopic pregnancies with methotrexate is possible if the patient is hemodynamically stable, has adequate pain control, tolerates fluids by mouth, is reliable, and can follow up appropriately. A patient with an ectopic pregnancy being treated with methotrexate should return to the ED immediately if she develops severe pain, intractable nausea, fever, severe weakness, lightheadedness, or has syncope.

Pearls, pitfalls and myths

- All women of childbearing age should be considered pregnant until proven otherwise.
- Do not rely on the patient's β-HCG to decide whether to order a pelvic US when considering ectopic pregnancy. Ectopic pregnancies produce β-HCG at an abnormal rate. A β-HCG level above the discriminatory zone only helps to determine whether a viable IUP should be visible on transvaginal US. Therefore, use the β-HCG to assist with interpretation of the pelvic US.
- Heterotopic pregnancy has a reported incidence as low as 1:100 in patients undergoing ART. Therefore, the diagnosis must be considered even in patients with confirmed IUPs. The likelihood of heterotopic pregnancy in patients not undergoing ART is much lower. Clinical suspicion should guide the work-up and follow-up.
- Suspected PID and STIs should be treated empirically before laboratory results return. Results often will not be back before the patient's disposition from the ED, and life- or fertility-threatening sequelae may occur without treatment (e.g., abscess, sepsis, infertility). Many patients at greatest risk of developing these conditions have limited financial resources, suffer from substance abuse, or have other problems limiting their access to follow-up care.
- Several diagnoses can be missed without full consideration of all diagnostic possibilities (e.g., ovarian torsion). An ovarian cyst or mass may predispose a patient to ovarian torsion, which may not be appreciated on pelvic examination. In fact, many question the diagnostic value of the pelvic examination, especially in the obese patient.
- Sexual and physical abuse should be considered in all women and children, especially if they present with nonspecific complaints or inconsistent findings. Often, the only way to uncover this history is to directly ask the patient in a private setting. Therefore, it is prudent to ask friends, spouses, boyfriends, girlfriends or family members to leave the room when obtaining this important history.
- A patient's pelvic pain may be caused by several nonpelvic etiologies. Furthermore, pelvic pain may be the result of certain catastrophic conditions, which must be aggressively managed if present.

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35 Rash

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Scope of the problem

The skin is the body's most visible organ system and its main protection against the environment. Skin complaints account for 4–10% of all emergency department (ED) visits annually in the United States. Skin disease can represent a wide array of disease processes, from a local dermatologic disease to the manifestation of an underlying systemic illness. The majority of rashes that present to the ED involve allergies, irritants and infections.

Although most of these rashes are benign and self-limited, cutaneous lesions are often the first clinical sign of serious systemic disease. Dermatologic findings associated with serious infectious diseases include meningococcemia, gonococcemia, cellulitis, toxic shock syndrome (TSS), staphylococcal scalded skin syndrome (SSSS), disseminated herpetic infections, and Rocky Mountain spotted fever (RMSF). Other potentially life-threatening skin diseases can result from medications, such as Stevens–Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), or urticaria with anaphylaxis. Carcinomas and other inflammatory skin diseases (pustular psoriasis, pemphigus, pemphigoid and systemic lupus erythematosus [SLE]) also have the potential to be life-threatening (Table 35.1).

Table 35.1 Life-threatening dermatoses

Behçet's syndrome
Bullous pemphigoid
Cutaneous T-cell lymphoma
Disseminated gonococcemia
Disseminated herpes
Disseminated zoster
Generalized exfoliative erythroderma
Hematologic disorders
Kaposi's sarcoma
Kawasaki disease
Malignant melanoma
Meningococcemia
Pemphigus vulgaris
Pustular psoriasis
Rocky Mountain spotted fever
Staphylococcal scalded skin syndrome
Stevens-Johnson syndrome
Systemic lupus erythematosus
Toxic epidermal necrolysis
Toxic shock syndrome
Urticaria with anaphylaxis

Anatomic essentials

The skin is divided into three layers. The outer layer is the *epidermis*, serving as the most protective barrier against the environment. Underneath the epidermis, the vascularized *dermis* provides support and nutrition for the cells of the epidermis. Other important skin structures are found in the dermal layer, including nerves, sweat glands, hair follicles and sebaceous glands. The inner layer is the subcutaneous layer, which contains adipose tissue.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 35.2).

History

A complete history of the eruption is essential and should include information regarding the duration, rate of onset and location of the current eruption. Symptoms including pruritus, pain and fever should be noted.

When did the rash begin?

Sudden onset of a rash is more concerning than a rash that has been present for days. Sudden onset of rash while eating shellfish most likely represents an allergic reaction, which may signal the beginning of anaphylaxis. A rash that develops after 3 days of fever in a 12-month-old child is likely roseola infantum, a benign condition that resolves spontaneously.

Where on your body did you first notice the rash? Has it spread?

Identifying the location where the rash first appeared helps further differentiate its etiology. Rashes that present on the scalp and then erupt on the elbows may represent psoriasis. RMSF starts on the wrists and ankles, and then spreads to the trunk (known as centrifugal spread).

Have you had this rash before?

Rashes that are recurrent are more likely to represent an underlying dermatitis or noninfectious systemic illness, such as a recurring rash on the face from seborrheic dermatitis.

Have you had a fever associated with the rash?

In children and adults, fever associated with rash often signifies the presence of an infection. For example, a young child with fever and sore throat who presents with a diffuse, red, sandpaper-like rash on the trunk,

Table 35.2 Rash red flags

History	Concerning diagnosis
Onset and progression (rapid progression is most lethal)	Meningococcemia, urticaria, drug eruption
Travel	Dengue, typhus, Lyme disease, RMSF
Fever, systemic complaints	Meningococcemia, dengue, SJS, SSSS, TEN, TSS, Kawasaki disease, RMSF, scarlet fever, EM, syphilis, Lyme disease
Medications, recent new medication	SJS, TEN, anaphylaxis, urticaria, drug eruption, drug–drug interaction
Immune compromised	HIV, Kaposi's sarcoma, disseminated zoster, shingles, meningococcemia, TEN, SJS, syphilis, EM major, TTP, necrotizing fasciitis, disseminated fungal infection
Age (very young or old)	Meningococcemia, Kawasaki disease, pemphigus vulgaris, sepsis, TEN, SJS, TSS
Examination finding	**Concerning diagnosis**
Mucous membrane involvement	SJS, EM major, TEN, pemphigus vulgaris, Kawasaki disease
Fever, tachycardia, hypotension, toxic appearance	Meningococcemia, TSS, TEN, RMSF, SJS
Skin sloughing (Nikolsky sign)	TEN, SSSS, pemphigus vulgaris
Location	***Central:*** Lyme disease, viral exanthem, drug reaction ***Peripheral:*** Meningococcemia, SJS, EM, RMSF, syphilis, Lyme disease, scabies
Petechiae/purpura	Meningococcemia, Purpura fulminans, DIC, necrotizing fasciitis, disseminated gonococcal infection, RMSF, HSP, TTP, endocarditis, vasculitis
Heart murmur, Janeway lesions	Endocarditis
Severe localizing pain, tenderness in extremities	Necrotizing fasciitis, cellulitis
Arthralgias	RMSF, drug reaction, viral illness

DIC: disseminated intravascular coagulation; EM: erythema multiforme; HIV: human immunodeficiency virus; HSP: Henoch-Schönlein purpura; RMSF: Rocky Mountain spotted fever; SJS: Stevens-Johnson syndrome; SSSS: Staphylococcal scalded skin syndrome; TEN: toxic epidermal necrolysis; TSS: toxic shock syndrome; TTP: thrombocytopenic thrombotic purpura.

back and extremities is likely to have scarlet fever. Fever with sudden onset of a petechial rash in a college student or military recruit is meningococcemia until proven otherwise.

Does the rash itch?

Rashes that are pruritic indicate an inflammatory reaction with histamine release. Pruritus represents an intradermal response that can be from a local exposure to an irritant or a systemic reaction, such as an allergic reaction to shellfish. The patient who presents with pruritus and linear eruptions on his hands or legs after a recent hiking trip likely has contact dermatitis from poison ivy exposure. Intense pruritus is often associated with urticaria or scabies.

Is the rash painful?

A history that the rash is associated with pain may be significant. With herpes zoster (shingles), pain often precedes the rash. In patients with necrotizing fasciitis, the pain may be out of proportion to physical examination findings.

Have you used new soaps, perfumes, lotions, or detergents? Have you recently been exposed to plants, animals, or insects?

Many commercial products contain chemicals that can produce a local inflammatory reaction. The patient who presents with a new rash on his trunk and arms after changing laundry detergent brands likely has contact dermatitis. Many lotions for dry skin contain alcohol, which may exacerbate eczema or contact dermatitis. In addition, exposure to animal dander, insect bites and plants may produce allergic contact dermatitis.

Do you have any known environmental allergies or recent excessive sun exposure?

Exposure to chemicals at home or work may cause a contact dermatitis. A history of recent sun exposure may represent sunburn, atopic dermatitis, or an allergic reaction. Photosensitivity reactions are common with photoallergic or phototoxic medications, such as tetracyclines, quinolones, sulfonamides, neuroleptics, or nonsteroidal anti-inflammatory, antimalarial and cardiac drugs.

Have you used any medications to treat the rash?

There are many over-the-counter medications available to the public, including antihistamines and topical low-potency steroids. Use of medications prior to seeking medical attention may indicate a partially- or undertreated medical condition. Furthermore, some of these therapies may exacerbate rather than alleviate the rash, such as topical steroid use for a cutaneous fungal infection.

What medications do you take regularly? Are you taking any new medications?

Rashes associated with medications range from mild allergic eruptions to anaphylaxis or other life-threatening systemic complications. Therefore, a complete history of previous and current medications, dosages, duration of therapy, and prior history of allergic or adverse reactions to medications should be obtained. Recent immunizations may account for an allergic eruption or exanthem. Review patient use of over-the-counter medications, herbals, dietary supplements and vitamins.

Associated symptoms

Respiratory

Ask about nasal discharge, sore throat, shortness of breath and cough. Viral exanthems frequently appear following a viral upper respiratory tract infection. These exanthems are typical of those caused by viruses seen in children, such as coxsackie and varicella. Influenza and adenovirus, more common in adults, are rarely associated with rashes. Bacterial sources may also cause exanthems. The rash of scarlet fever, caused by group A streptococci, appears 1–3 days after the onset of pharyngitis.

Patients with urticarial rash associated with anaphylaxis may exhibit signs of respiratory distress, including shortness of breath, wheezing and cough, if the respiratory tract or mucous membranes are involved.

Gastrointestinal

Ask about abdominal pain, nausea, vomiting and diarrhea. A patient with abdominal pain, vomiting, diarrhea and hypotension may have a systemic life-threatening illness, such as TSS. If the patient is ill-appearing, begin resuscitation and complete a thorough evaluation.

Henoch-Schönlein purpura (HSP) is a small-vessel vasculitis that typically occurs in patients between the ages of 2 and 11 years. Ninety percent of patients presenting with HSP have GI complaints, such as abdominal pain and heme-positive stool.

Neurologic

Ask about altered mental status, headache, seizures and other neurologic symptoms. Meningitis presents with a brief prodrome of headache and fever, followed by rapid clinical decline with altered mental status. Patients with herpes or varicella encephalitis may present with headache or new-onset seizures.

Genitourinary

Ask about pregnancy, sexual history and any prior genitourinary lesions. Taking a sexual history will provide important information. The generalized rash of secondary syphilis is called the "great masquerader" because of its varied presentations. More than 60% of patients with secondary syphilis do not remember having a chancre. Always ask if the patient is pregnant; order a pregnancy test if this is a possibility. Certain rashes produce few side effects in the general population, but may be devastating to an unborn fetus.

Past medical

A history of previous and recurrent eruptions along with other systemic disease (e.g., diabetes, SLE, cancer) is helpful in identifying the cause of the rash. While reviewing the patient's occupational history and hobbies, environmental and chemical exposures, recent travel history and immunization status should be documented as well.

Family

Family history of rashes and certain systemic illnesses, such as SLE, should be considered. Document known first-degree relatives with a family history of environmental and food allergies.

Physical examination

Although the history may help narrow the differential diagnosis, the ability to identify, interpret and describe what is observed is of greater importance. Rashes are classified using specific nomenclature (Tables 35.3 and 35.4).

Table 35.3 Primary skin lesions

Term	Definition
Macule	Flat, non-palpable discoloration <1 cm in size. May be brown, blue, red, or hypopigmented
Patch	Flat, non-palpable discoloration >1 cm in size
Papule	Solid, raised, palpable lesion up to 0.5 cm in diameter, may become confluent (plaque)
Plaque	Circumscribed, elevated, superficial, solid lesion >0.5 cm in diameter
Nodule	Circumscribed, rounded, raised, palpable lesion >0.5 cm in diameter. A large nodule is a tumor
Vesicle	Well-circumscribed, raised, fluid-filled lesion <0.5 cm in size
Bulla	Well-circumscribed, fluid-filled, raised lesion >0.5 cm in diameter
Wheal (hive)	Firm, edematous, transient plaque resulting from infiltration of the dermis with fluid

Table 35.4 Secondary and other skin lesions

Term	Definition
Scales	Excess dead epidermal cells that are produced by abnormal keratinization and shedding
Crust	Collection of dried serum and cellular debris (scab)
Erosion	Focal loss of epidermis that does not penetrate the dermis (no scar)
Ulcer	Focal loss of epidermis and dermis (scar)
Excoriation	Erosion caused by scratching (often linear)
Lichenification	Area of thickened epidermis induced by scratching; surface looks like a washboard
Petechiae	Round, pinpoint, flat purplish spots secondary to intradermal or subdermal hemorrhage <0.5 cm diameter
Purpura	Blue or purple in color; secondary to hemorrhage in the skin >0.5 cm diameter

Additional descriptors for shape include serpiginous, arcuate, annular, discoid, target, dermatomal and confluent.

Physical examination of patients with a rash should be performed from head to toe with the patient completely disrobed in a warm, well-lit examination room. Practitioners should allow plenty of time for a thorough physical examination of the skin.

General appearance

The general appearance of the patient will often suggest the severity of underlying disease causing the rash in question. Patients may be well or ill-appearing depending on the severity of rash. However, most patients presenting with rash appear nontoxic, although uncomfortable.

Vital signs

Fever is often used as a marker for infection. Conversely, an afebrile patient may still have a rash secondary to a viral or bacterial source. Patients with petechial or purpural lesions and signs of sepsis, tachycardia or hypotension are presumed to have a bacterial infection and should be aggressively resuscitated.

Other vital signs may be helpful in diagnosing an infectious systemic process or an acute allergic reaction with anaphylaxis. However, most infectious causes of rash do not affect the heart rate, blood pressure or respiratory rate unless accompanied by severe dehydration, sepsis, or airway compromise.

Head and neck

Inspect the head and neck to identify signs of infection. Examine the scalp and mucosa for rash or lesions. Bluish-white lesions of the buccal mucosa with surrounding erythema are called Koplik's spots. They are pathognomonic for measles infection. Examine the soft palate for petechiae, which may indicate an underlying streptococcal infection. When an allergic reaction is suspected, look for edema of the soft palate and uvula. Examine the neck for signs of infection, including reactive lymph nodes, swollen glands, or nuchal rigidity. Auscultate the neck for stridor if an allergic reaction is suspected.

Genital

Rashes in the groin and on the genitalia necessitate a thorough examination. A pelvic examination is indicated if there is suspicion of disseminated gonococcal infection or TSS secondary to a retained foreign body (i.e., tampon) in the vaginal canal. Tests for sexually transmitted infections (STIs) like syphilis (Figure 35.1) and gonorrhea should also be performed.

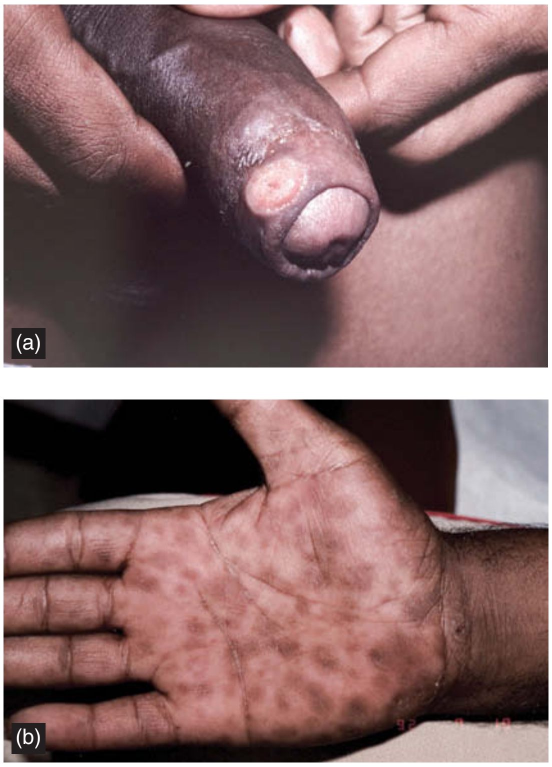

Figure 35.1
(a) Chancre of primary syphilis: round to oval indurated plaque, eroded not ulcerated. (b) Secondary syphilis: nonconfluent, macular erythematous-to-brown lesions. Courtesy: Steven Shpall, MD.

Skin

Examine the skin in a systematic and orderly process, noting the distribution, pattern, arrangement and morphology of the rash. Many rashes have a predilection for certain areas of the body, so patients should be completely disrobed.

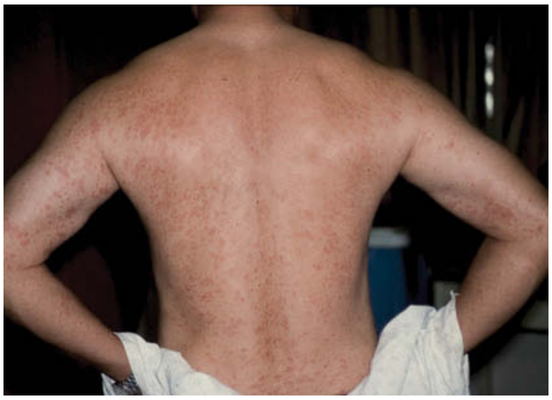

Figure 35.2
Pityriasis rosea. Round to oval spots with an inner collarette of scale (scale inside the lesion, not at its edge) distributed along skin lines. Courtesy: Steven Shpall, MD.

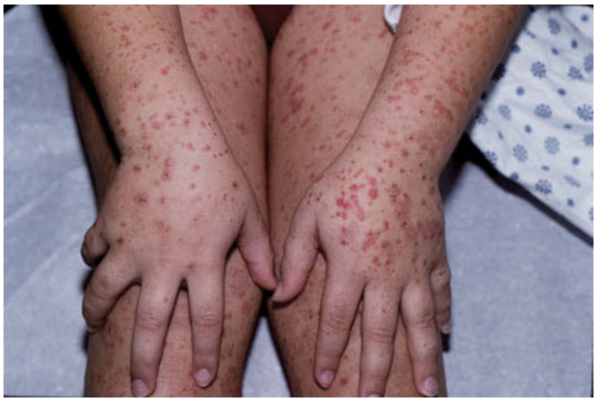

Figure 35.3
Henoch-Schönlein purpura. Palpable purpura with small hemorrhagic (purple) macules and papules, usually on the extensor surface of the extremities. Courtesy: Steven Shpall, MD.

Document the pattern of the rash. A rash located only on skin exposed to the environment or a particular object points to reactions associated with sun exposure, jewelry (nickel), or lotions. Pityriasis rosea is typically localized to the trunk and proximal extremities (Figure 35.2). The lesions of hand–foot–mouth disease are located where the name implies. Erythema nodosum and HSP (Figure 35.3) have a predilection for the lower extremities.

Lesion arrangement (symmetry and configuration) should be noted. Rashes that are bilaterally symmetric often signify systemic disease or uniform external exposure. Configuration refers to the relationship between multiple lesions, such as the linear pattern of poison ivy exposure or the Christmas-tree distribution of pityriasis rosea (Figure 35.2).

Recognition of the primary lesion is vital in establishing the diagnosis. The primary lesion can be altered by secondary issues, including excoriation, healing, previous medications, or complications of infection. Once the primary lesion is noted and its morphology determined, identify the likely cause from the differential diagnosis (Table 35.5).

Table 35.5 Differential diagnosis of primary and secondary lesions

Morphology	Differential considerations
Bullae	Bullous impetigo, bullous pemphigoid, pemphigus vulgaris, toxic epidermal necrolysis, thermal burn, toxicodendron dermatitis
Crusts	Eczema, tineas, impetigo, contact dermatitis, insect bite
Erosions	Candidiasis, tineas, eczema, toxic epidermal necrolysis, toxic-infectious erythemas, erythema multiforme, primary blistering diseases (bullous pemphigoid, pemphigus vulgaris), brown recluse spider bite
Macules	Drug eruption, rheumatic fever, erythema multiforme, cellulitis, lice infestation, secondary syphilis, viral exanthems, early meningococcemia
Nodules	Basal cell carcinoma, melanoma, lipoma, warts
Papules	Atopic dermatitis, acne, folliculitis, psoriasis, eczema, urticaria, toxicodendron dermatitis (poison ivy, oak or sumac), insect bites
Petechiae	Gonococcemia, leukocytoclastic vasculitis, meningococcemia
Plaques	Eczema, pityriasis rosea, tinea corporis and versicolor, psoriasis, urticaria, erythema multiforme
Purpura	Platelet abnormalities, Rocky Mountain spotted fever, scurvy, senile purpura
Pustules	Folliculitis, acne, gonococcemia, herpetic infections, impetigo, psoriasis
Scales	Psoriasis, pityriasis rosea, toxic-infectious erythemas, secondary syphilis, tineas
Ulcers	Aphthous lesions, chancroid, decubitus, thermal injury, subacute/chronic ischemia, malignancy, chancre, primary blistering disorders, pyoderma gangrenosum, stasis
Vesicles	Herpetic infections, toxic epidermal necrolysis, toxicodendron dermatitis, thermal burn, bullous pemphigoid, pemphigus vulgaris
Wheals	Angioedema, hives, urticaria, erythema multiforme

Differential diagnosis

Tables 35.6–35.10 describe the differential diagnosis of various causes of rash.

Table 35.6 Differential diagnosis of viral etiologies of rash

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Erythema infectiosum (Fifth disease)	Childhood rash, primarily age 2–14 years 50% of adults have serologic evidence of past infection Caused by parvovirus B19 Increased prevalence in winter and spring	Characterized by erythematous plaques on cheeks; slapped cheek appearance Low-grade fever, headache, sore throat, nausea/vomiting 8–10 days before rash Asymptomatic infection is common, but severe complications can be seen in pregnant, anemic, or immunocompromised patients Women (not men) can have acute polyarthropathy that can last 2 weeks to 4 years	Diagnosis is made clinically Laboratories not indicated Treatment usually only supportive care
Hand-foot-mouth-disease	Largely a disease of childhood Age 2–4 years More common summer and autumn	Characterized by ulcerative oral lesions, primarily on soft palate, and tender pustular rash on palms and soles of feet Treatment usually only supportive care	Diagnosis is made clinically Laboratories not indicated
Herpes simplex virus eruption	Two most common serotypes: 1 and 2 Most common in children and young adults	Grouped vesicles on erythematous base on keratinized skin and mucous membranes Usually on cheeks, lips, mouth, fingers and genitalia Symptoms 1–2 weeks	Tzanck smear if diagnosis in question Viral cultures HSV antibody serologies Acyclovir or other antivirals used for both treatment and prevention of eruptions Prednisone may decrease acute pain, but increases complications
Herpes zoster (Shingles) (Figure 35.4)	Nearly 100% of adults in the United States are seropositive for anti-VZV antibodies by third decade of life Two-thirds of cases occur in patients > 50 years old	Rash erupts as papules and transforms to vesicles or bullae in 24 hours Vesicles become pustules in 48 hours and crusts by day 7 Erupts in dermatomal pattern (pathognomonic) Typically does not cross midline unless patient is immunocompromised	Diagnosis made by history and physical examination Tzanck smear if diagnosis in question Laboratories only indicated if severe secondary infection suspected Treatment with acyclovir or equivalent anti-viral, pain medication and prednisone Consult ophthalmology if ocular involvement
Measles	Highly contagious disease of childhood Rarely seen in children in the United States because of immunization Outbreaks may be seen in third decade of life or in unvaccinated children	Characterized by fever, cough and coryza Rash on face, neck and shoulders *Koplik's spots* in mouth are bluish-white papules with erythema on the buccal mucosa (pathognomonic)	Diagnosis is made clinically Laboratories not indicated Treatment usually only supportive care
Roseola infantum (exanthem subitum or Sixth disease)	Affects infants between 6 and 36 months of age Caused by HHV-6 and HHV-7	Characterized by sudden appearance of rash after defervescence of a high fever (3–4 days later) Infant usually appears well despite fever Rash is small pink macules and papules that become confluent and fade	Diagnosis is made clinically Laboratories not indicated Treatment usually only supportive care
Rubella (German measles)	Benign childhood infection of young adults Rarely seen in the United States due to immunization	Pink maculopapules that start on forehead and spread to face, trunk and extremities Characteristic 3-day course, after which the rash fades completely Infection during pregnancy can result in congenital defects	Diagnosis is made clinically Laboratories not indicated Treatment usually only supportive care Infection during pregnancy – consider therapeutic abortion or passive immunization
Varicella (chicken pox)	Nearly 95% occurs before the age of 10 years 3–4 million cases annually in the United States Decreasing prevalence due to vaccine Transmitted by both direct contact and airborne droplets	Highly pruritic, general viral symptoms usually precede rash by one day Begins as macules 10–21 days post-exposure, then papular eruption that evolves into vesicles Vesicles become pustules and crust over a 12-hour period Continual eruptions over 4–5 days; infectious while vesicles are present *"Dewdrop-on-a-rose-petal"* Beware of severe complications, especially in immune compromised (pneumonia, meningitis, encephalitis)	Diagnosis made by a history of viral prodrome and recent exposure Laboratories not indicated Oral acyclovir may decrease severity of outbreak if started ≥24 hours of first eruptions Avoid aspirin and NSAIDs in children

HHV: human herpesvirus; HSV: herpes simplex virus; NSAIDs: nonsteroidal antiinflammatory drugs; VZV: varicella zoster virus.

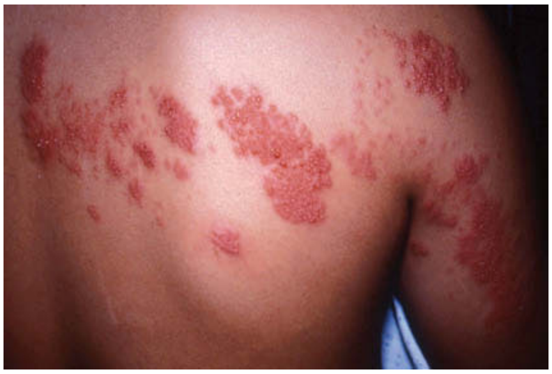

Figure 35.4
Herpes Zoster (Shingles). Erythematous macules and papules developing into vesicles on an erythematous plaque, and finally into crusts, distributed over one or two dermatomes. Courtesy: Steven Shpall, MD.

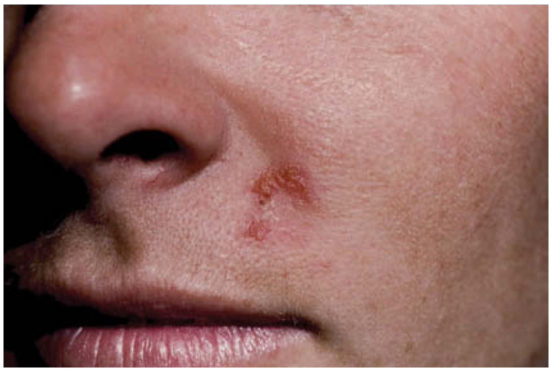

Figure 35.5
Impetigo. Honey-colored crusts often on erythematous base. Courtesy: Steven Shpall, MD.

Table 35.7 Differential diagnosis of bacterial etiologies of rash

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Erysipelas	Occurs at any age Most frequently in children <3 years old and older individuals Commonly caused by group A streptococci	Characterized by red, hot and tender area of skin High fever and chills associated with group A streptococci Pus or clear discharge at site of entry into the skin	Gram stain of discharge if present Blood cultures are very low yield Treatment with penicillin G, dicloxacillin, or equivalent Diagnosis is made clinically
Impetigo (Figure 35.5)	Primary infections most common in children Secondary infections in patients with underlying dermatoses Caused by *S. aureus* and group A streptococci	Superficial bacterial infection of the epidermal skin Rash appears as a golden yellow-crusted erosion Can be pruritic Contagious for first 2–3 days of treatment Commonly seen on face around mouth and cheeks	Laboratories not indicated Treatment with oral and/or topical antibiotics, such as 2% mupirocin ointment, dicloxacillin, first-generation cephalosporins or azithromycin
Meningococcemia	Occurs at any age More common in winter and spring Spread from nose and mouth Most common in teenage and college-age individuals	Rash is maculopapular with petechiae High fever, tachycardia, tachypnea and hypotension	Immediate antibiotic treatment that penetrates the CSF blood–brain barrier (penicillin G, ceftriaxone, cefotaxime, ampicillin or chloramphenicol) CBC, chemistries, clotting studies Blood and CSF cultures Isolation and admission to the hospital
Scarlet fever (Figure 35.6)	Seen primarily in children with pharyngitis, usually 3–12 years Usually caused by group A streptococci Rarely caused by *S. aureus*	Rash appears 1–3 days after onset of strep pharyngitis symptoms Scarlatiniform rash is a finely punctate erythema on the upper trunk with a sandpaper-like feel Progresses to neck, back, groin and axilla Spares palms and soles Pharynx is beefy red with *strawberry tongue*	Rapid direct antigen test to screen for group A streptococci Oral swab for bacterial culture ASO titer if diagnosis in question Treatment with penicillin G or equivalent May return to school 24 hrs after fever subsides
Staphylococcal scalded skin syndrome (Figure 35.7)	Most common in neonates but also seen in infants and small children Caused by toxin-producing *S. aureus* Infections of umbilical stump or nasal infection	Widespread detachment of the superficial layers of the epidermis Ranges from localized bullous impetigo to extensive epidermolysis Desquamation of the affected area is common	CBC and blood cultures Bacterial cultures of wound not indicated Oral or intravenous antibiotics based on severity (erythromycins, penicillinase-resistant penicillins or cephalosporins) Hospital admission for severe cases

(*continued*)

Table 35.7 Differential diagnosis of bacterial etiologies of rash (*cont.*)

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Toxic shock syndrome	Most common in women 20–30 years of age Primarily caused by toxin-producing *S. aureus*	Generalized scarlatiniform erythroderma most intense around infected area Edema of face, hands and feet is common; looks like mild sunburn	Blood cultures and wound cultures (often negative) CBC, chemistries and LFTs Intravenous antibiotics with staphylococcal coverage, such as oxacillin, cefoxitin, vancomycin or clindamycin Hospitalization and fluid resuscitation
Syphilis (Figure 35.1)	*Treponema pallidum* Called the "great masquerader" because of varied rash presentations	Generalized, painless and non-pruritic Distributed on skin and mucous membranes Follows skin cleavage lines Discrete, scaly, red-brown papules and plaques Associated with headache, sore throat, malaise and generalized arthralgias	Laboratory testing with VDRL or RPR necessary to make diagnosis Dark-field examination of scrapings may be beneficial

ASO: anti-streptolysin; CBC: complete blood count; CSF: cerebrospinal fluid; LFTs: liver function tests; *S. aureus*: *Staphylococcus aureus*; RPR: rapid plasma reagin; VDRL: Venereal Disease Research Laboratory.

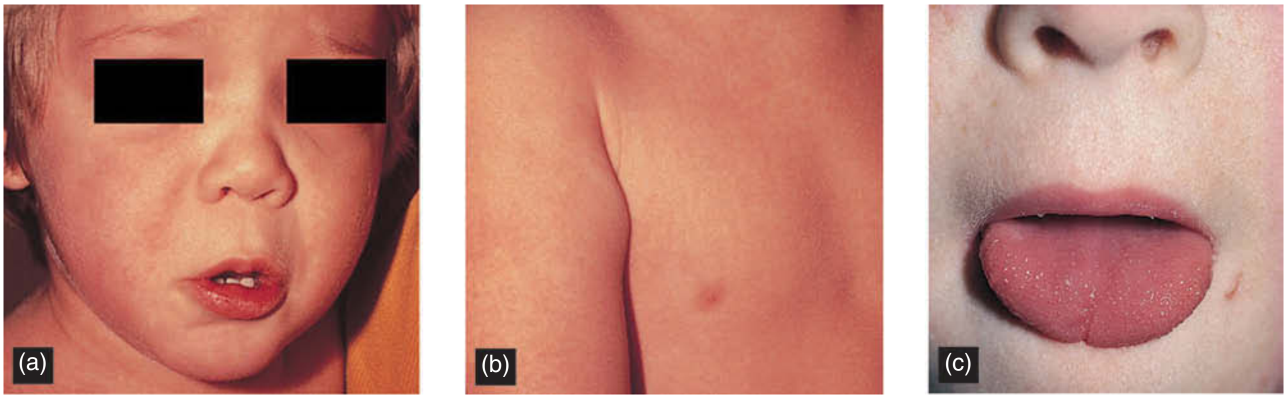
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Figure 35.6
Streptococcal scarlet fever. (a) The patient has a flushed face and perioral pallor; (b) diffuse, blanching, erythematous rash that has a sandpapery consistency on palpation; (c) the characteristic red strawberry tongue with glistening surface and prominent papillae. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed. Copyright 2002, with permission from Elsevier.

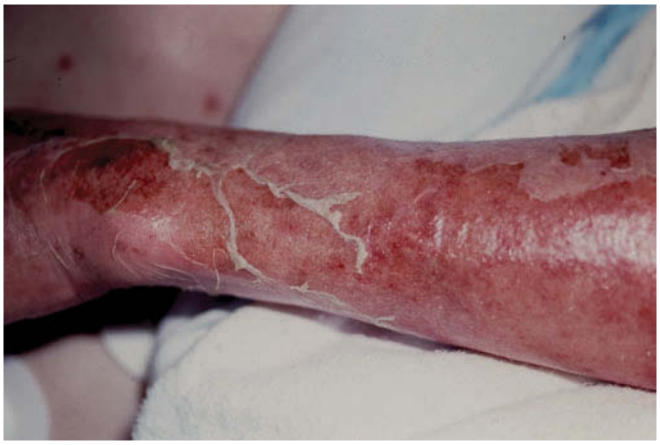

Figure 35.7
Staphylococcal scalded skin syndrome. Erythema in which a superficial split in the epidermis develops, leading to widespread exfoliation of large sheets of the upper epidermis. Courtesy: Steven Shpall, MD.

Table 35.8 Differential diagnosis of fungal etiologies of rash

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Tinea capitis	Mostly children 6–10 years old Increased rural prevalence Blacks > whites Risk factors include debilitation, malnutrition and chronic disease	Inflammatory type associated with pain, tenderness and/or alopecia	Wood's lamp Cultures Topical antifungals are not effective Systemic treatment with griseofulvin, terbinafine, fluconazole or ketoconazole; adjunctive selenium sulfide shampoo
Tinea pedis	20–50 years old Males > females	May be dry and scaly or macerated, peeling; associated with fissures between 4th and 5th toes	Scrapings to detect hyphae Wood's lamp Fungal culture Treatment with topical or oral antifungals, such as terbinafine, naftifine or fluconazole
Tinea corporis	Occurs in all age groups Higher incidence in animal workers and individuals with pets	Characterized by small to large scaling, sharply demarcated plaques Lesions have peripheral enlargement and central clearing	Diagnosis by KOH slide preparation Wood's lamp Treatment with topical azole cream is usually effective Systemic antifungal treatment for large infections or if refractory to topical creams
Tinea cruris	Predisposing factor is a warm, moist environment Males > females	Lesions are often bilateral and begin in skin folds Half moon-shaped plaque with well-defined scaly border	Clinical diagnosis Antifungal topical agents

KOH: potassium hydroxide.

Table 35.9 Differential diagnosis of infestations and bites

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Lyme disease	Tick-borne illness Vaccine now available for high-risk individuals Caused by spirochete *Borrelia burgdorferi*	Initial erythematous macule or papule that expands with distinct red border with central clearing (erythema migrans), must be ≥3.5 cm	Skin biopsy of erythema migrans lesion (spirochetes ≤40%) Serology studies Borrelia culture from skin biopsy Oral antibiotics (penicillin G, doxycycline, amoxicillin or azithromycin) and close outpatient follow-up
Rocky Mountain spotted fever (Figure 35.8)	Incidence highest 5–9 years old, approximately 600 cases/year Fatality highest in males *Rickettsia rickettsii* Transmitted by ticks Occurs mainly in northern climates in the spring, later in southern climates	Prodrome of anorexia, irritability, malaise, fever and chills 2–14 days after tick bite Followed by abrupt fever, severe headache, generalized myalgias, rigors, photophobia and prostration Rash can start on the 1st day (14%) up to the 6th day (20%), or does not appear at all (13%) Rash begins on wrists, forearms and ankles Early lesions are 2–6 mm pink, blanching macules that evolve to deep red papules and then become hemorrhagic over 1–4 days	Diagnosis depends on clinical symptoms and history of potential or confirmed tick exposure, because laboratory confirmation cannot occur before 10–14 days Treatment with tetracycline, doxycycline or chloramphenicol Associated with hyponatremia, thrombocytopenia and hypoalbuminemia with or without increased WBC Most patients are hospitalized
Scabies	Microscopic mite *Sarcoptes scabiei* White, transparent creature <0.5 mm long Transmitted by close personal contact Incubation life span 30 days	Pruritic, worse at night Usually starts 2 weeks post-exposure Papulovesicular dermatitis Distribution predominately volar wrists, medial palms, interdigital web spaces and axillary folds Usually spares face and scalp, except in infants Skin burrows seen	Skin scrapings may demonstrate mites, eggs, or feces Treatment includes washing all clothes and bed linens

WBC: white blood cell.

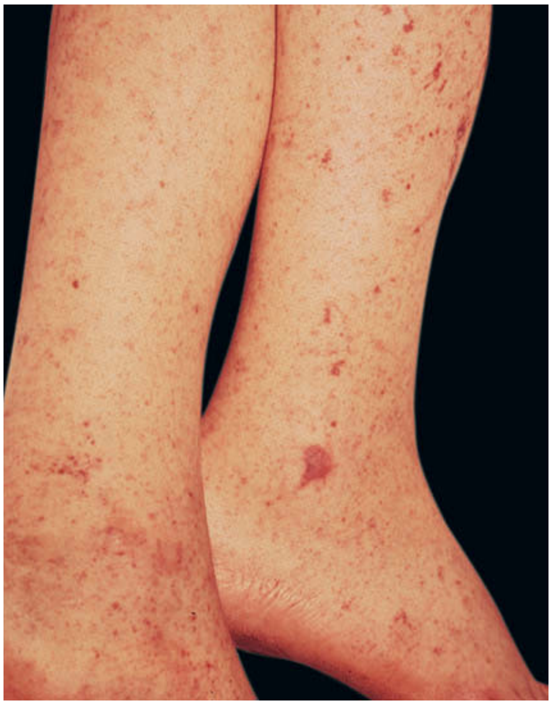

Figure 35.8
Rocky Mountain spotted fever. A generalized petechial eruption that involves the entire cutaneous surface, including the palms and soles. Reprinted with permission from Habif TP, *Clinical Dermatology*, 4th ed., page 525, Copyright 2004, with permission from Elsevier.

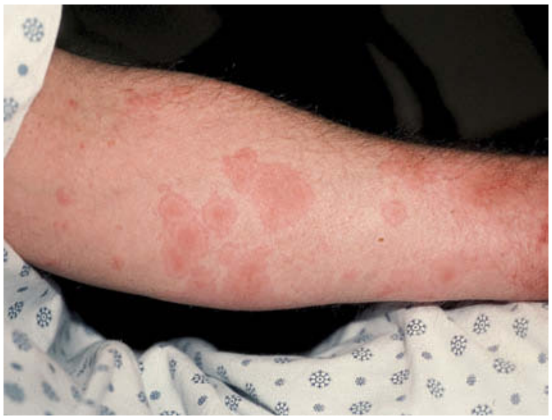

Figure 35.9
Erythema multiforme. Target lesions, erythematous patches or plaques with dusky central areas that can develop into bullae. Courtesy: Steven Shpall, MD.

Table 35.10 Differential diagnosis of dermatitis and inflammatory disorders

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Allergic contact dermatitis	Often associated with plants (allergic phytodermatitis) Poison ivy/oak are the most common causes	Primarily on hands and exposed extremities Begins as erythematous areas that evolve into edematous papules, nodules and plaques Often in a linear arrangement	Topical corticosteroids are effective for small areas with non-bullous lesions
Atopic dermatitis (eczema)	Begins in the first year of life in 60% of patients Possible association with aeroallergens (dust mites) and foods (peanuts, milk, eggs) Exacerbated by skin dehydration from frequent showers and hand washing Chronic, remitting and relapsing disease seen with a personal or family history of atopy (asthma or allergic rhinitis)	Patients have dry skin Pruritus is the hallmark of atopic dermatitis Scratching leads to lichenification of the skin, which causes the skin to become more dried out Predilection for flexure surfaces, sides of the neck, face, wrists and dorsum of the feet	Bacterial cultures for possible secondary infection with *S. aureus* Viral culture to rule out HSV in crusted lesions Check serum IgE levels Treatment: oral antihistamines, topical corticosteroids and skin hydration with emollient creams
Bullous pemphigoid	Autoimmune disorder Occurs in 6th to 8th decade of life Complement activation leading to inflammatory cascade response Lesions caused by autoantibodies directed at basement membrane, resulting in subepidermal blister formation	Erythematous, papular, or urticarial-type lesions followed by bullae formation with predilection for flexural surfaces Bullae contain serous or hemorrhagic fluid Bleeding is sometimes a problem Found predominantly on axillae, groin, abdomen and lower legs	Neutrophils at dermal-epidermal junction on light microscopy Serum tests for circulating auto-antibodies IV fluid replacement Strong topical steroids and oral tetracycline preferred with azathioprine or dapsone

(*continued*)

Table 35.10 Differential diagnosis of dermatitis and inflammatory disorders (*cont.*)

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Erythema multiforme (Figure 35.9)	Occurs at any age, but 50% ≤20 years old Associated primarily with HSV infection 50% of cases from unknown origin More frequent in men than women	Vesicle and bullae in the center of a papule Peripheral clearing produces distinct target lesion	Clinical diagnosis but confirmed by biopsy showing perivascular mononuclear infiltrate Treatment supportive with pain medication, but frequent recurrent episodes may be treated with oral acyclovir
Henoch–Schönlein purpura (Figure 35.3)	Children 2–11 years of age Peaks in winter No clear etiology Some association with group A beta-hemolytic strep and viruses Acute vasculitis with IgA deposits Male/female 2:1	Palpable purpura on buttocks and lower extremities May begin as urticaria Hemorrhagic vesicles in severe cases Association with arthritis in 2/3 of cases May be associated with intussusception	Increased WBC, mild anemia and thrombocytopenia can be seen 40% will have proteinuria and hematuria Blood cultures may be necessary to rule out sepsis Supportive care Hydration Prednisone, NSAIDs
Irritant contact dermatitis	Reactions resulting from direct damage to epithelium with subsequent cytokine release Hands are the most commonly affected area Most cases are caused by chronic exposure	Symptoms of itching, burning and stinging are usually the only manifestations Dry skin with erythema and chapping common Severe cases produce a caustic burn with vesiculation	Patch testing can be done by a dermatologist for possible allergic etiology Avoidance of caustic agents is treatment of choice Topical corticosteroid and barrier creams are effective
Kawasaki disease	Peak onset at 1 year of age; mean age 2.5 years Slight male and Asian predominance Acute febrile illness of infants and children Unknown etiology Systemic vasculitis of microvessels	Cutaneous and mucosal erythema and edema with subsequent desquamation Lesions appear 1–2 days after onset of fever (of at least 5 days) Rash first noted on palms and soles, then spreads to trunk and extremities Edema of hands and feet develop after rash Oropharynx becomes erythematous	Clinical diagnosis includes fever for ≥5 days and skin changes Laboratories: WBC >18,000 common LFTs abnormal Thrombocytosis and elevated ESR after 10th day of illness Urine may show sterile pyuria Treatment with high-dose aspirin and IVIG Admission to hospital required
Pemphigus vulgaris	Autoimmune disorder Occurs in 4th to 6th decade of life Loss of normal cell-to-cell adhesion in the epidermis High morbidity and mortality from secondary infection of lesions	Starts with painful oral lesions Skin lesions are round or vesicles and bullae with serous content *Nikolsky's sign* positive (slight thumb pressure causes the skin to wrinkle, slide laterally and separate from the dermis) Predominantly on scalp, face, chest, axilla and groin	Immunofluorescence of skin biopsy reveals IgG deposits IV fluid replacement necessary
Pityriasis rosea (Figure 35.2)	Occurs between first and fourth decades of life More common in the spring and fall months Possibly caused by HHV-7	Begins with a single truncal lesion or "*herald patch*" (2–5 cm diameter salmon-colored, single oval scaly patch) Secondary eruption 1–2 weeks later usually on the trunk and proximal aspects of extremities Lesions are erythematous macules or papules "Christmas tree" pattern, following Langer's lines	Diagnosis made by skin biopsy and light microscopy Self-limited with spontaneous remission in 6–12 weeks Treatment with oral antihistamines and topical steroids
Psoriasis	Peak incidence 20–30 years; second peak 55–60 years	May be precipitated by trauma (Koebner's phenomenon) or infection Pruritus common Silver, scaly rash on an erythematous base Located on scalp, extensor surfaces and groin area May produce arthritis in distal joints	Should be managed by a dermatologist due to need for "shifting" therapies

(*continued*)

Table 35.10 Differential diagnosis of dermatitis and inflammatory disorders (*cont.*)

Diagnosis	Epidemiology	Symptoms/signs	Work-up/treatment
Stevens-Johnson syndrome (Figure 35.10)	Occurs at any age but most common in adults >40 years 50% are associated with drug exposure Drugs most frequently implicated: sulfa, aminopenicillins, carbamazepine, phenytoin and allopurinol	Prodrome with fever and flu-like symptoms 1–3 days later develop mucocutaneous lesions Skin rash is EM, which is brightly erythematous with bullae Fever is common Secondary infection may occur, making diagnosis more difficult Anemia, lymphocytopenia and neutropenia may occur	Diagnosis is confirmed by biopsy IV fluids critical to replace fluids lost from wounds Treatment is similar to that of burns, mostly supportive Early diagnosis and withdrawal of suspected drugs critical
Toxic epidermal necrolysis (Figure 35.11)	Occurs at any age but most common in adults >40 years 80% are associated with drug exposure	Skin rash is EM, which is brightly erythematous with bullae >30% epidermal detachment Fever is common and typically higher than in SJS Secondary infection may occur, making diagnosis more difficult *Nikolsky's sign* positive	Diagnosis confirmed by biopsy IV fluids critical to replace fluids lost from skin Treatment is similar to that of burns; consider transfer to a burn center Early diagnosis and withdrawal of suspected drugs critical Systemic steroids have not been proven helpful
Urticaria	Most common skin rash for which acute care is sought (15–20% of population affected) Mast cell degranulation and histamine release causes hives Majority of etiologies unknown	Benign and self-limited (usually) Raised erythematous borders with serpiginous edges and blanched centers Diameter ranges from a few mm to 30 cm Pruritic Lasts for a few minutes to several hours	Laboratory tests not usually indicated Consider Strep screen or culture Symptomatic treatment with antihistamines Prednisone short course preferred over taper Epinephrine if severe associated reaction

EM: erythema multiforme; ESR: erythrocyte sedimentation rate; HHV: human herpesvirus; HSV: herpes simplex virus; IgE: immunoglobulin E; IgG: immunoglobulin G; IV: intravenous; IVIG: intravenous immunoglobulin; LFTs: liver function tests; NSAIDs: nonsteroidal antiinflammatory drugs; SJS: Stevens-Johnson syndrome; WBC: white blood cell.

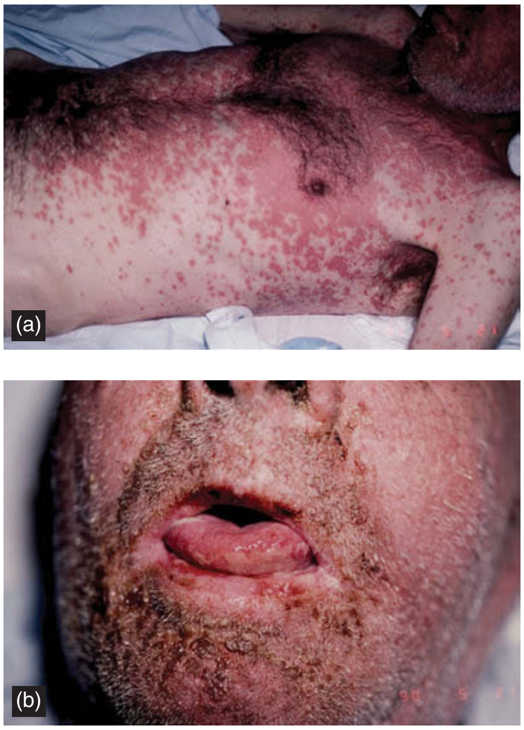

Figure 35.10
Stevens–Johnson syndrome. (a) Atypical or incomplete target lesions that coalesce and can develop into bullae. (b) The mucous membranes usually are involved with erosions and crusting. Courtesy: Steven Shpall, MD.

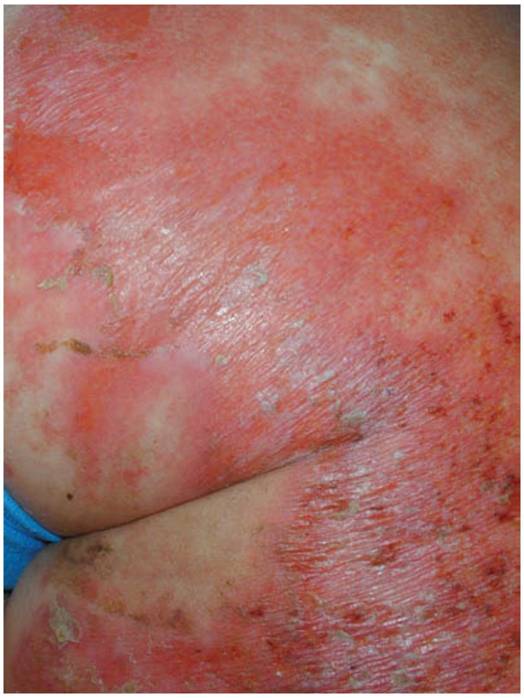

Figure 35.11
Toxic epidermal necrolysis. Erythema in which a full-thickness split below the epidermis develops, leading to widespread exfoliation of large sheets of epidermis. Courtesy: Steven Shpall, MD.

Diagnostic testing

Traditional laboratory tests for the evaluation of most rashes are generally non-diagnostic and of little use in the ED. Specific tests for viral infections, such as the Tzanck smear for herpetic eruptions, can be performed when the diagnosis is in question. Patients presenting with severe bacterial infections or signs of cardiovascular collapse, as may occur in septic shock, require the following tests: complete blood count (CBC) with differential to look for signs of hemolysis, leukocytosis and demargination; prothrombin time/partial thromboplastin time (PT/PTT) to look for etiologies of petechiae or purpura; Gram stain to identify organisms; blood cultures for identification of bacteremia; and chemistries to look for signs of renal failure or electrolyte abnormalities. Lyme serologies, VDRL testing for syphilis, an erythrocyte sedimentation rate (ESR) for vasculitis should be considered.

For noninfectious rashes, laboratories should be directed towards identification of an immunologic or hematologic etiology (Table 35.11). Specific tests for scabies may be useful in the appropriate situation. Results for such testing may not be immediately available and should be done in conjunction with the appropriate follow-up physician.

Table 35.11 Methods of diagnostic testing

Diascopy	A glass slide pressed firmly against a red lesion will determine whether the rash is due to capillary dilation (blanchable) or to extravasation of blood (non-blanchable).
KOH preparation	Scrape scales from the skin, hair, or nails. Add 10% KOH solution to dissolve tissue material. Identification of septated hyphae indicates fungal infection; pseudohyphae and budding spores indicate yeast infection.
Tzanck preparation	Scrape the base of a vesicle and smear cells on a glass slide. Multinucleated giant cells are associated with herpes simplex, zoster and varicella infections.
Scabies preparation	Scrape skin of a burrow and place on a slide. Mites, eggs, or feces seen in scabies infections.
Wood's lamp	Examination under a long-wave ultraviolet light ("black" lamp). Tinea capitis will fluoresce green or yellow on the hair shaft.

KOH: potassium hydroxide.

General treatment principles

The treatment of rashes varies greatly based on the cause of the eruption and the severity of the illness (Tables 35.6–35.10). The main goal of treatment is largely supportive and symptomatic, aimed at relieving pain and pruritus. Only those patients with severe systemic illness from overwhelming infection, fluid losses and severe pain require inpatient care.

Volume repletion

Patients with signs of sepsis, as may occur with toxic shock syndrome, or with severe fluid losses, as in toxic epidermal necrolysis, should be aggressively resuscitated with intravenous crystalloid fluid. The rate of repletion is based on the patient's degree of hypovolemia, vital sign derangement and physical examination.

Pruritus and pain control

Patients often complain that their rash is pruritic and painful. Use of antihistamines (oral and topical) for symptomatic relief with topical steroid agents or systemic corticosteroids should provide the patient significant relief. Nonsteroidal antiinflammatory drugs (NSAIDs) and narcotics should be used judiciously for pain control. For example, adults with zoster should be given high doses of NSAIDs with or without narcotics for pain and inflammation. Aspirin should be avoided in children with varicella or chickenpox due to the risk of Reye syndrome.

Emollients

Emollient creams and lotions restore water and lipids to the epidermis. Preparations that contain urea or lactic acid have special lubricating properties and may be the most effective. Creams are thicker than lotions, and many lotions contain alcohol to ease application (which can cause irritation). As petroleum jelly and mineral oil contain no water, water should be added to the skin prior to application.

Topical corticosteroids

Topical steroids are very powerful drugs for treating dermatologic diseases. Steroids are divided by potency: mild (hydrocortisone), medium (triamcinolone), high (fluocinonide), and ultra-high (betamethasone, clobetasol). Their effects result in part from their ability to induce vasoconstriction of small vessels in the dermis. It is this degree of vasoconstriction that determines a steroid's potency classification. Lowering the concentration of the drug would not necessarily decrease the amount of vasoconstriction; many have the same vasoconstrictive properties despite different concentrations (0.25%, 0.5%, 1%). Generic substitutes are not necessarily equivalent, and adequate potency and treatment length are important considerations when prescribing a steroid cream. High- and ultra-high–strength topical steroids should be reserved for resistant lesions, lichenified areas and hand lesions. Medium-strength steroid creams are useful for most skin areas. Therefore, triamcinolone 0.1% cream is often prescribed in the ED. Only low-potency creams should be used on the face, and steroids should never be placed on the eyelids.

The base determines the rate at which the active ingredient is absorbed through the skin. Creams can be used in most areas of the body, and are excellent for intertriginous areas. Ointments are translucent, greasy, more lubricating, have greater penetration than creams, and are too occlusive for use in acute eczematous inflammation or intertriginous areas. Gels are effective for acute exudative inflammation, such as poison ivy. Since they are clear, gels are cosmetically acceptable on hair-covered areas. Lotions are clear or milky, most useful for the scalp, and may result in stinging and drying when applied to intertriginous areas. The amount of cream dispensed is important; 1 g of cream covers 10 cm × 10 cm of skin, 20–30 g covers the entire skin of most adults. For ointments, the amount that fits on your fingertip typically covers the equivalent of the front and back of the hand.

Antivirals and antibiotics

Viral exanthems do not require antibiotics. However, the use of antivirals (such as acyclovir or valacyclovir) for varicella, zoster and herpetic eruptions may reduce the duration of symptoms, decrease the incidence of post-herpetic neuralgia, or decrease future outbreaks. For bacterial infections, topical antibiotic ointments for superficial cutaneous infections and intravenous antibiotics for patients with systemic infections are necessary. Antibiotics should target the likely bacterial pathogens. Be aware of developing antibiotic resistance in the community; the Centers for Disease Control and Prevention and the hospital pharmacist or infectious disease specialist should be consulted for recommendations.

Antifungals

The newest class of antifungal agents, the allylamines (e.g., terbinafine), have been shown to produce higher cure rates and more rapid responses in dermatophyte infections than older agents. Some of the oral medications (fluconazole) are effective in weekly dosing patterns, which may increase compliance. Because most fungal infections require long treatment courses (≥ 2–12 weeks), lack of compliance is frequently associated with treatment failure. Other side effects, such as hepatic injury, have been associated with ketoconazole and less frequently with griseofulvin, so monitoring of liver enzymes is advised.

Immunosuppressants

Autoimmune-mediated disorders may require the use of systemic steroids or other medications to produce immunosuppression. Immunosuppressant medications should be given only after consultation with the appropriate specialist. Such medications carry severe adverse effects, and need close monitoring to prevent iatrogenic disease.

Special patients

Elderly

Geriatric patients often take more medications than the general population. Therefore, a careful review of all medications, including recent changes, should be documented. Assess for allergic reactions as well as drug–drug interactions. The geriatric population may not tolerate certain medications, such as antihistamines or narcotic analgesics.

Pediatric

Many viral exanthems occur only in childhood and are self-limited. Caution should be exercised when recommending or using antihistamines in infants and young children. Exanthems were previously numbered according to their historical appearance and description:

- First disease: measles
- Second disease: scarlet fever
- Third disease: rubella
- Fourth disease: "Dukes" disease (probably coxsackie virus or echovirus)
- Fifth disease: erythema infectiosum
- Sixth disease: roseola infantum

Pregnancy

Pregnant women exposed to a patient with rubella, varicella or Fifth disease are at risk for fetal complications, and therefore need expeditious follow-up with appropriate health care professionals. Drugs used to treat rashes in a pregnant woman should be prescribed only after discussion with an obstetrician, dermatologist, or reference on drugs in pregnancy. Pruritic urticarial papules and plaques of pregnancy syndrome (PUPPPS) affects about 1% of all pregnant patients.

Immune compromised

Patients who are immunocompromised are more susceptible to infection and secondary infection when their skin integrity has been compromised. Suspected bacterial infections in this group should be treated aggressively, with appropriate parenteral antibiotics and observation in the hospital if systemic illness is suspected.

Disposition

Dermatologic consultation

The majority of patients who present to the ED with a rash do not require an emergent consultation with a dermatologist. Emergency physicians and primary care providers can adequately diagnose and care for most non–life-threatening rashes. In patients with severe dermatologic disease or in cases in which the diagnosis remains in question, dermatologic consult may be required for evaluation and possible biopsy.

Admission

Most patients with a rash do not require inpatient care. Such care is reserved for those with systemic bacterial infection, signs of sepsis, life-threatening dermatoses, severe dehydration and intractable pain. Patients who require admission should first be adequately resuscitated in the ED. A dermatology or infectious disease consultation may be needed for patient's requiring admission.

Discharge

After a thorough evaluation in the ED, most patients with rash are discharged home with supportive care instructions. Follow-up with a patient's primary health care provider should be scheduled within 1–2 days for reevaluation and assessment of outpatient treatment. Referral to a dermatologist should be made if patients have severe disease or a chronic dermatologic process.

Pearls, pitfalls and myths

- The majority of rashes are benign and self-limited, requiring only supportive care. However, a few rashes are life-threatening and should not be overlooked (Table 35.1).
- History and physical examination are vital to diagnose a rash or identify its cause.
- Toxic epidermal necrolysis and staphylococcal scalded skin syndrome look similar; however, the first requires removal of the offending agent (often an antibiotic) and the second requires treatment with an antibiotic.
- The rash of toxic shock syndrome can be indistinct and should be considered in patients with fever and volume depletion associated with a rash.
- The early rash of meningococcemia may be macular, maculopapular, or petechial; early identification is important and potentially lifesaving.
- Extensive work-ups with laboratory studies are often unnecessary in the ED when evaluating most rashes.
- Urticarial skin lesions may be the first sign of infection, infestation, or systemic disease requiring further investigation.
- Although the disease causing a rash may be benign, patients who are immune compromised or pregnant may have higher risks of complications.

Acknowledgment

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36 Scrotal pain

Jonathan E. Davis, MD

Scope of the problem

Among the most challenging conditions in emergency medicine is a male who presents with acute scrotal pain. An "acute scrotum" is painful swelling of the scrotum or its contents, accompanied by local signs and symptoms. The highly sensitive nature of such male genital complaints can lead to anxiety and embarrassment for the patient and the caregiver.

Precise diagnosis of acute scrotal problems is not always straightforward; the goal is to identify true genitourinary (GU) emergencies requiring prompt evaluation and treatment. Identification of testicular torsion is of paramount importance, as it may threaten testicular viability and future fertility if not managed swiftly and appropriately. Similarly, early identification and aggressive management of necrotizing fasciitis of the perineum (Fournier's disease) is critical.

Distinguishing between etiologies can be particularly challenging in children, who (like adults) are most likely to present with an "undifferentiated" acute scrotum. In the vast majority of pediatric cases, the acute scrotum can be attributed to one of three diagnostic entities: testicular torsion, epididymitis, or appendage torsion. The most common diagnostic considerations in adults presenting with an acute scrotum are testicular torsion, epididymitis and epididymo-orchitis.

Anatomic essentials

The male genitalia includes the *penis* (containing the paired erectile bodies and penile urethra) and *scrotum* (encasing paired testes and epididymis) (Figure 36.1). There are several fascial planes that collectively comprise the *perineum,* providing protection and stability to the enclosed structures. However, these anatomic layers also provide a conduit for rapid spread of infection.

The scrotal wall contains several layers deep to the scrotal skin, many of which are contiguous with the penis, perirectal region and anterior abdominal wall. Each testis is encapsulated within a dense connective tissue layer known as the *tunica albuginea*. A break in the integrity of this thick connective tissue represents a "ruptured" testicle, which may occur following direct trauma. External to the testicular parenchyma and tunica albuginea is the *tunica vaginalis*, which envelops each testicle and fastens it to the posterior scrotal wall. The *scrotal ligament*

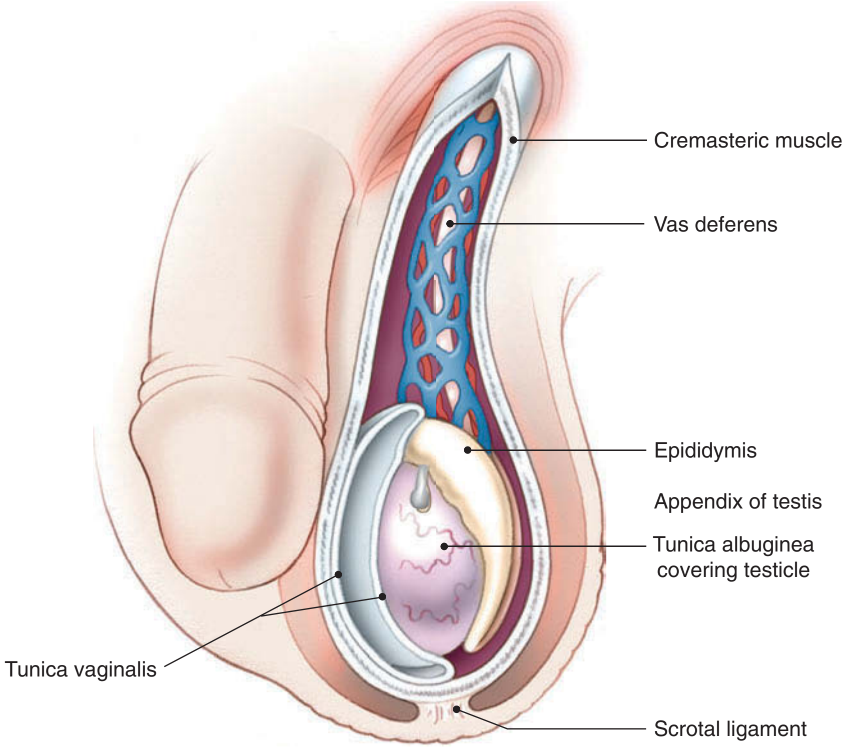

Figure 36.1
Anatomy of the scrotum. © Chris Gralapp.

(gubernaculum) provides additional stability by anchoring each testicle inferiorly. The tunica vaginalis consists of both visceral (contiguous with the tunica albuginea) and parietal portions, with potential space interposed. The significance of this potential space is that the lack of firm attachment between the testicle and the posterior scrotal wall makes each testis prone to rotate about the spermatic cord within the tunica vaginalis, resulting in *testicular torsion.* The spermatic cord contains both the blood supply to each testicle via the gonadal vessels and the vas deferens. Interruption of blood flow to the testes by twisting the spermatic cord (which occurs in testicular torsion) can lead to rapid ischemia and subsequent infarction of the affected testicle. The *appendix testes* are embryologic remnants without physiologic function, located at the uppermost pole of the testes. These appendages are prone to torsion as well, leading to self-limited localized necrosis of the appendage. This results in pain that may be confused with discomfort due to torsion of the testicle.

The *epididymis* is a fine tubular structure that adheres closely to the posterolateral aspect of each testicle. It is involved in promoting sperm maturation and motility. Similar to the appendix testes, the *appendix epididymi* are embryologic remnants attached to the head of each epididymis with no known function. These are also prone to torsion, leading to localized necrosis of the affected appendage. The *vas deferens* is a tubular structure involved in sperm transit, extending from the epididymis to the prostatic portion of the urethra.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 36.1).

History

A diligent and focused history in a patient presenting with acute scrotal pain is essential to formulating an appropriate differential diagnosis and management plan. Acute scrotal complaints often include a component of patient embarrassment and apprehension; this is especially true in adolescents. In any situation where a patient presents with a family member or friend, it is important to offer to speak with the patient alone to ensure patient confidentiality and, in some cases, facilitate an accurate history.

How did the pain begin (sudden versus gradual onset)?

Pain that begins abruptly and severely is concerning for testicular torsion. The sudden twisting of the spermatic cord (characteristic of testicular torsion) rapidly

Table 36.1 Scrotal pain red flags

History	**Concerning diagnoses**
Sudden onset, intermittent/colicky pain, sharp/stabbing pain, minor trauma preceding onset of pain, vomiting	Testicular torsion
Fever	Fournier's disease or epididymo-orchitis
Hypertension (in an adult)	AAA
Hypotension	Ruptured AAA or Fournier's disease
Constipation/obstipation	Incarcerated/strangulated inguinal hernia
Neonate with inconsolable crying	Testicular torsion or incarcerated/strangulated inguinal hernia
Immune compromise (diabetes, HIV, other)	Fournier's disease
GU trauma	Occult GU injury until proven otherwise
Examination finding	**Concerning diagnoses**
Documented tachycardia or hypotension	Leaking/ruptured AAA or Fournier's disease
Writhing in pain, elevated testis with transverse lie	Testicular torsion
Perineal/scrotal erythema, ecchymoses or crepitus	Fournier's disease
Normal GU exam (with or without a pulsatile abdominal mass)	AAA
Testicular nodularity/firmness	Testicular carcinoma
Exquisite prostate tenderness	Prostatitis
RLQ abdominal tenderness	Acute appendicitis
Persistent bulge in inguinal region despite manipulation	Incarcerated/strangulated inguinal hernia
Subtle evidence of GU trauma (abrasions, contusions, ecchymoses)	Occult GU injury until proven otherwise
Intrascrotal hematoma (hematocele)	Ruptured testicle (traumatic)

AAA: abdominal aortic aneurysm; GU: genitourinary; HIV: human immunodeficiency virus; RLQ: right lower quadrant.

decreases the blood supply to the affected testicle, causing ischemic pain. This is in contrast to the more indolent and smoldering pain of epididymitis, a gradually progressive inflammatory process. Chronic inguinal hernias often present with isolated scrotal pain of prolonged duration. However, an incarcerated (unable to reduce) or strangulated (ischemia and infarction of herniated bowel) hernia typically presents in a more dramatic fashion.

Is the pain "constant and progressive" or "intermittent and colicky?"

As a general rule, constant and progressive pain likely represents a progressive inflammatory process (acute appendicitis is a classic example). Intermittent and colicky pain is more consistent with rapid onset and offset conditions, such as irritation and spasm of hollow structures (renal or biliary colic are classic examples) or a twisting mechanism (as in testicular torsion). Epididymitis is a progressive inflammatory condition, so pain generally develops gradually and worsens over time. Patients may exhibit pain with ambulation and movement as a result of the inflammation. Pain of testicular torsion, on the other hand, may be intermittent and colicky, as the spermatic cord may spontaneously torse and detorse.

What were you doing when the pain began?

It is critical to elicit a history of blunt or penetrating trauma to the scrotum, penis or surrounding structures, as traumatic injury must be included in the differential under these circumstances. Testicular torsion is often accompanied by an inciting history of minor trauma, leading to rotation of the testicle (and twisting of the spermatic cord) within the tunica vaginalis. However, testicular torsion may occur in the absence of such events, even during sleep.

How long has the pain been present?

The pain of acute testicular torsion often develops over minutes, whereas pain associated with more indolent inflammatory conditions such as epididymitis develops over several hours to days. Testicular masses, such as testicular cancer, usually progress over several weeks to months. However, a patient with a testicular tumor may develop acute pain secondary to hemorrhage within the tumor, given its rich vascular supply.

How would you characterize the pain (dull, aching, sharp, stabbing, throbbing)?

The patient's description of the pain may help differentiate between potential etiologies. Pain associated with epididymitis is often described as dull and aching. Early in its course, pain may be mild, but increases in severity commensurate with worsening inflammation. Sudden pain associated with testicular torsion is often described as sharp, stabbing or throbbing.

How would you rate the severity of your pain on a scale of 0–10?

Patients with testicular torsion often complain of severe, ischemic pain. The pain associated with epididymitis or appendage torsion is often less severe than pain from testicular torsion. Unfortunately, the patient's quantification of pain is often inconsistent and generally unreliable in narrowing the differential diagnosis. Indeed, a majority of queried patients may answer "10 out of 10" due to the highly sensitive nature of the genitalia.

Have you ever had similar episodes of pain?

Patients with testicular torsion may have had prior episodes of similar pain that resolved spontaneously. Furthermore, pain of chronic conditions (e.g., hernias, hydroceles, varicoceles, tumors) may present with subacute or chronic pain with intermittent exacerbations.

Are alleviating or exacerbating factors present?

Pain resulting from an inflammatory process (epididymitis) may be temporarily relieved with rest and scrotal elevation with supportive undergarments, such as a jockstrap. Movement or activity often exacerbates pain due to inflammatory conditions. In contrast, patients with testicular torsion often writhe in pain and have difficulty finding a position of comfort.

Can you show me with one finger exactly where the pain is located?

It is essential to ascertain the precise anatomic region(s) responsible for the pain. Pain may be due to structures within or adjoining this particular region, or may be referred from other areas. The majority of patients with the complaint of acute testicular pain will have a problem isolated to the genitalia. However, several conditions may lead to referred testicular or scrotal pain, including abdominal aortic aneurysm (AAA), renal colic, or pyelonephritis.

Does the pain move anywhere?

It is common for patients with acute scrotal pain from a variety of causes to complain of lower abdominal, proximal lower extremity (i.e., inner thigh, groin), back, or flank pain. Likewise, it is important to consider acute GU pathology in any male patient presenting with seemingly isolated pain to the aforementioned anatomic regions. For instance, GU pathology should be considered in any male presenting with lower abdominal pain.

Associated symptoms

Systemic

It is critical to ask about systemic findings in any patient presenting with an acute scrotum. As a general rule, males with testicular torsion are more ill-appearing, with associated systemic signs and symptoms including nausea and vomiting. In contrast, patients presenting with appendage torsion or uncomplicated epididymitis exhibit fewer systemic symptoms. However, patients with epididymitis may present with low-grade fever, nausea or malaise. In addition, patients with more advanced degrees of infection (e.g., progressive infection that envelops the ipsilateral testis, known as epididymo-orchitis) often present with higher fever and greater systemic involvement.

Urinary

Inquire about changes in urination, including urgency, frequency, dysuria, hesitancy and hematuria. Urinary symptoms may accompany many causes of acute scrotal pain. Classically, epididymitis may be accompanied by urinary complaints. An inability to void is important, as this may indicate urethral obstruction, prostatitis, or severe intravascular volume depletion.

Genital

Ask about reproductive tract symptoms, such as erectile function, penile discharge and ejaculatory changes. A yellow-green penile discharge suggests the diagnosis of urethritis or epididymitis, often caused by *Gonorrhea* and *Chlamydia* species in sexually active males. Hematospermia may be present in cases of epididymitis, as the inflammatory process leads to spilling of blood into the seminal fluid. Pneumaturia may be the result of a colovesical fistula, often caused by an eroding malignancy.

Gastrointestinal

Ask specifically about abdominal or flank pain, nausea, vomiting, distention and bowel changes. One important consideration in the patient with GU complaints and abdominal findings (such as pain, distention, and constipation or obstipation) is an incarcerated or strangulated inguinal hernia. Patients with a retroperitoneal abdominal process, such as renal colic, pyelonephritis, or ruptured AAA, may present with pain referred to the ipsilateral testicle with or without associated flank or abdominal pain.

Physical examination

General appearance

The general appearance of a patient presenting with an acute scrotum provides important diagnostic clues. Most often, patients with testicular torsion are more ill-appearing than patients with other etiologies of acute scrotal pain. In addition, patients with testicular torsion or renal colic tend to writhe in pain and cannot find a position of comfort. In contrast, patients with epididymitis or epididymo-orchitis tend to minimize activity, as movement generally exacerbates their discomfort.

Vital signs

Inflammatory and infectious conditions such as epididymitis, scrotal abscess, or Fournier's disease tend to cause fever. However, fever is a nonspecific finding that may be present in a variety of conditions. Abnormalities of other vitals signs may help uncover more advanced stages of disease progression. Hypotension and tachycardia may result from dehydration, sepsis, or acute blood loss (as with a leaking or ruptured AAA). Tachycardia and tachypnea may occur as a consequence of substantial pain, or may signify more ominous underlying derangements.

Abdomen

A complete abdominal examination is crucial in any patient presenting with acute scrotal pain. It is important to remember that many intra-abdominal processes may present with a component of (or even isolated) scrotal or testicular pain. Always assess for costovertebral angle tenderness (CVAT), often present in retroperitoneal processes including pyelonephritis, renal colic, and expanding or ruptured AAAs. These conditions may present with GU pain. In addition, it is important to assess for lower abdominal tenderness or masses, which may be present in acute appendicitis, inguinal hernias, GU malignancies, traumatic injuries, or progressive infection (i.e., Fournier's disease).

Genitalia

It is important to examine the male genitalia while the patient is standing and supine. Caution should be exercised when examining a standing patient, however, as some males may experience a strong vagal response to scrotal or prostate stimulation leading to pre-syncope or syncope. Examination of the scrotum may cause significant discomfort, even in the absence of pathology. Because many patients will have unilateral GU symptoms, it is best to examine the unaffected side first.

Inspection

Visual examination of the genitalia may reveal cutaneous rashes or lesions, abnormal testicular symmetry or position, edema (evident by loss of scrotal skin folds), or masses. Key visual features of testicular torsion include a high-riding testicle and a transverse lie of the affected testicle, both resulting from twisting of the spermatic cord (Figure 36.2). It is important to look for evidence of scrotal or perineal erythema or ecchymosis in older male patients with scrotal pain. These may be clues to Fournier's disease (Figure 36.3a and 36.3b). Fournier's disease most often affects diabetic or other immune-compromised patients. An early feature of necrotizing fasciitis may be significant pain in the absence of pronounced physical findings.

Palpation

Differentiating between etiologies of acute scrotal pain is challenging, as various scrotal conditions may present in

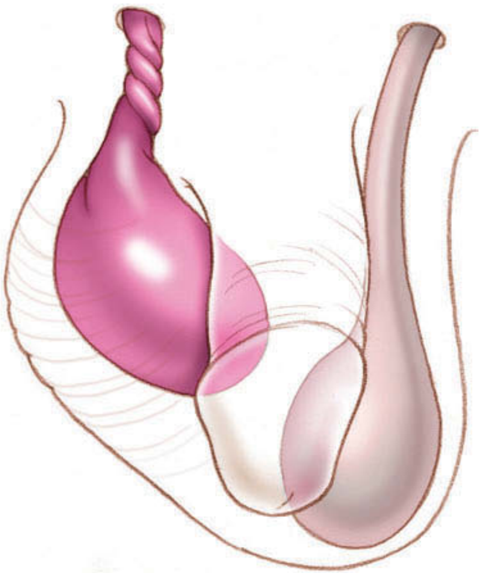

Figure 36.2
Testicular torsion. © Chris Gralapp.

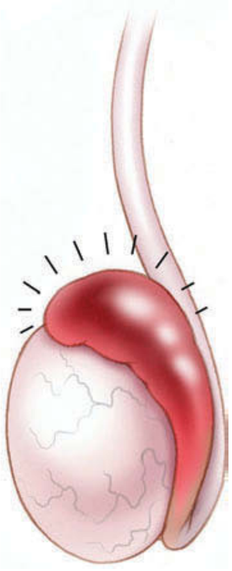

Figure 36.4
Epididymitis. © Chris Gralapp.

a similar fashion: unilateral (or bilateral) scrotal swelling and testicular enlargement with blurring of the distinction between the testicle and epididymis. Often confounding the problem is the exquisite pain and discomfort elicited by the examination itself. However, some findings (if present) are commonly associated with specific diagnoses:

- Isolated swelling and tenderness of the epididymis: epididymitis is likely (Figure 36.4). The natural progression of this infection is first to affect only the epididymis, with progression to the ipsilateral testicle (epididymo-orchitis).
- Isolated nodularity at the superior pole of either the testicle or epididymis: appendage torsion, given the anatomic location of these structures.
- Isolated testicular swelling: testicular torsion, orchitis or vasculitis (i.e., Henoch-Schönlein purpura).
- Swelling in the inguinal region: inguinal hernia.
- Swelling surrounding the testicle: hydrocele or hematocele. Hydroceles result from fluid accumulation (blood in the case of hematocele) in the tunica vaginalis. A varicocele is an abnormal engorgement of the gonadal venous plexus, classically described as a "bag of worms" and appreciated on palpation of the spermatic cord superior to the testicle.
- Testicular nodularity or firmness: carcinoma until proven otherwise.

Rectal

A digital rectal examination provides information about the prostate and prostatic portion of the urethra. Exquisite

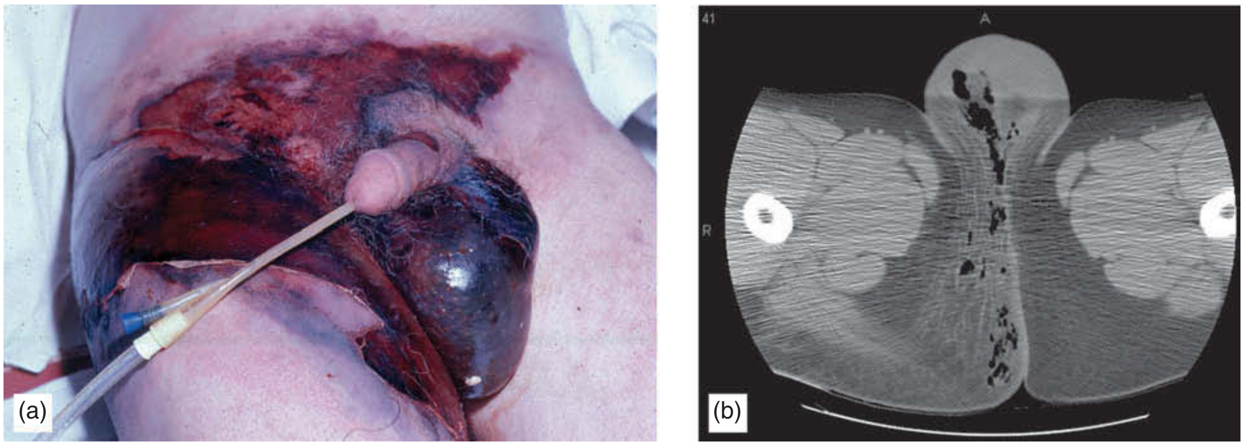

Figure 36.3
(a) Advanced Fournier's disease. Courtesy: Knowledge and Skills Website RCS, Edinburgh, Scotland. (b) Axial CT section through the groin, showing gas within the scrotum and right perineal region, consistent with necrotizing fasciitis (Fournier's gangrene). Courtesy: Gus M. Garmel, MD.

prostate tenderness may indicate acute infection (i.e., prostatitis). Prostate firmness and enlargement is a finding in benign prostatic hypertrophy; nodularity is concerning for carcinoma. These conditions may present with varying GU symptoms.

Special signs/techniques

There are several adjuncts to the traditional examination of the male GU tract. Certain signs, if present, may aid in the proper identification of genital pathology.

Although imperfect, the presence of an intact ipsilateral *cremasteric reflex* helps exclude the diagnosis of testicular torsion (Figure 36.5). This reflex is elicited by stroking the inner thigh, which should result in contraction of the cremaster muscle and reflex elevation of the ipsilateral testicle greater than 0.5 cm. It is important to note that although the presence of an intact reflex may be useful to exclude torsion, its absence is nonspecific. Other scrotal conditions may cause a blunted reflex (e.g., epididymitis), and some individuals lack this reflex altogether (particularly males during the first 3 years of life). In addition, there have been several published reports of testicular torsion in the setting of an intact cremasteric reflex, so findings must be cautiously interpreted in the context of the overall clinical picture.

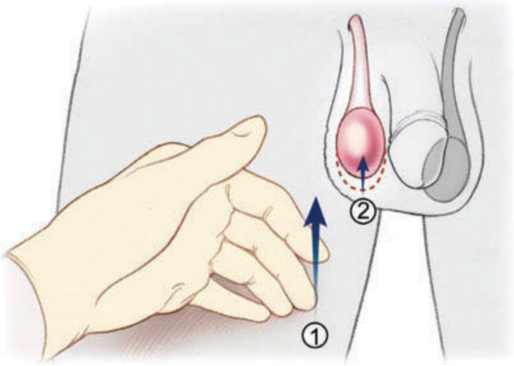

Figure 36.5
Cremasteric reflex. © Chris Gralapp.

Prehn's sign, relief of pain with scrotal elevation, was previously thought to help differentiate epididymitis (relieved with scrotal elevation) from testicular torsion (no change in symptoms). However, this sign has been found unreliable in distinguishing between these disorders, and its use for this purpose is not recommended.

In cases of suspected appendage torsion, a pathognomonic *blue dot sign* (Figure 36.6) represents visualization of the infarcted appendage ("blue dot") through thin, non-hormonally stimulated prepubertal skin. *Scrotal transillumination* (by shining a light through the scrotum) can determine whether the scrotum is filled with light-transmitting fluid, as is the case of a hydrocele. However, practitioners who seldom perform this technique may find that *every* hemiscrotum tends to transilluminate, so the results should be interpreted with caution.

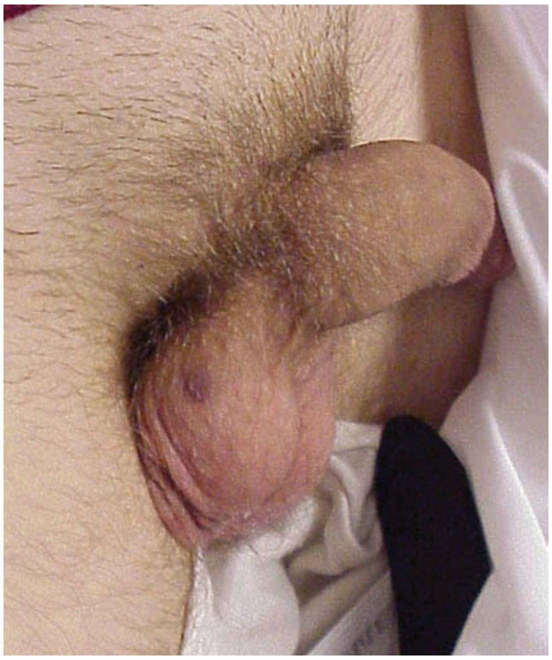

Figure 36.6
Blue dot sign. Courtesy: Selim Suner, MD.

Differential diagnosis

Table 36.2 describes causes of scrotal pain. Table 36.3 illustrates the characteristics of testicular torsion, epididymitis and appendage torsion.

Diagnostic testing

The key to managing acute scrotal pain is the timely recognition of life-, fertility-, or testicular viability–threatening conditions. Of the potential diagnoses, recognition of true testicular or scrotal emergencies takes precedence. Because testicular torsion produces end-organ ischemia, rapid detorsion must occur in order to prevent subsequent infarction and necrosis. This is typically accomplished in the operating room. Most routine diagnostic aids (such as blood or urine testing) add little to distinguish between the etiologies of acute scrotal pain. Rather, they may lead to diagnostic delays and worse patient outcomes. If the history and physical examination suggest the diagnosis of testicular torsion, urology consultation and (likely) surgical exploration should occur without delay. A patient of appropriate age with classic historical and physical examination findings of testicular torsion does not require diagnostic or confirmatory tests. In less certain presentations, a confirmatory radiologic study (Doppler ultrasound or radionuclide imaging) is indicated. In cases of Fournier's disease, delays in recognition and definitive surgical debridement increase mortality; early consultation and administration of broad-spectrum antibiotics is indicated in all suspected cases.

Table 36.2 Differential diagnosis of scrotal pain

Diagnosis	Symptoms	Signs	Work-up
Abdominal aortic aneurysm	Constant or intermittent flank, abdomen, or GU pain	Tachycardia, hypertension (prior to rupture) or hypotension (following rupture); pulsatile abdominal mass	US or CT; immediate surgical consultation
Acute appendicitis	Fever, nausea/vomiting, anorexia, RLQ pain	Fever, RLQ tenderness classic; may have associated abdominal rebound/guarding	CBC, urinalysis; CT or US; surgical consultation
Appendage torsion	More indolent onset of symptoms compared with testicular torsion; rarely "systemic" symptoms such as nausea and vomiting	Tender nodule at head of testicle or epididymis; "blue dot sign" pathognomonic	Imaging to ensure normal intratesticular blood flow (and therefore exclude testicular torsion)
Epididymitis	More indolent onset of symptoms compared with testicular torsion	*Early:* firmness and nodularity isolated to epididymis. *Late:* with progression, inflammation becomes contiguous with testicle (epididymo-orchitis)	Testicular US (or radionuclide imaging) reveals preserved (or increased) blood flow to affected testicle
Epididymo-orchitis	Often more "systemic" findings compared with isolated epididymitis	Large, swollen scrotal mass. Indistinct border between testicle and epididymis	Same as epididymitis
Fournier's disease	Perineal pain, swelling, redness, bruising, fever, vomiting, lethargy/weakness ("systemic" signs of illness)	Fever, paucity of local findings in early stages (pain "out of proportion" to physical findings); may rapidly progress to fulminant sepsis and shock	Emergent surgical consultation for debridement; broad-spectrum antibiotics (covering Gram-positive, Gram-negative and anaerobic species)
Hematocele	Large, painful scrotal mass; often antecedent history of trauma	Ecchymoses of scrotal skin; testicular tenderness	US
Hernia	Unilateral inguinal/scrotal swelling and pain	Reducible, incarcerated and strangulated forms; latter two often more tender on examination	Surgical consultation if incarcerated or strangulated; surgical referral if reducible
Hydrocele	Gradual onset of swelling	Transillumination may be helpful	US examination identifies fluid-filled cavity
Idiopathic scrotal edema	Typically unilateral scrotal swelling and edema; primarily seen in children <10 years old	Scrotal, perineal, inguinal erythema and edema	US in case of undifferentiated acute scrotum; exclude infectious etiology necessitating antimicrobial treatment
Orchitis	Gradual onset of unilateral (or bilateral) testicular swelling and pain	Swelling and tenderness isolated to testicle (or testes); no epididymal involvement	Often seen in conjunction with other systemic diseases; therefore, treatment is disease-specific
Scrotal skin infection	Variable depending on cause	Must distinguish between lesions localized to scrotal wall versus those contiguous with deeper structures	US (or CT) may be helpful in determining extent of deeper structure involvement
Testicular torsion	Sudden and severe onset of pain; often associated with nausea and emesis ("systemic" symptoms)	High-riding testicle and transverse lie of testicle; intact ipsilateral cremasteric reflex makes diagnosis of testicular torsion less likely	Emergent urology consultation in high-probability cases; consider US (or radionuclide) imaging if diagnosis uncertain
Trauma	History of blunt or penetrating mechanism of injury	Highly variable depending on mechanism	Urology consultation
Tumor	Gradually progressive testicular mass; often painless	May appreciate mass, firmness, or induration	US
Varicocele	Gradual onset of unilateral swelling; often painless	Abnormally enlarged spermatic cord venous plexus (described as a "bag of worms" on palpation)	US
Vasculitis (i.e., HSP)	Testicular swelling and pain	Associated vasculitis findings (such as buttock/lower extremity purpura and renal involvement in HSP)	Laboratory evaluation, including renal function tests; may necessitate admission

CBC: complete blood count; CT: computed tomography; GU: genitourinary; HSP: Henoch-Schönlein purpura; RLQ: right lower quadrant; US: ultrasound.

Table 36.3 Differentiating characteristics of testicular torsion, epididymitis and appendage torsion

Historical features and physical findings	Testicular torsion	Epididymitis	Appendage torsion
Age	Incidence peaks in neonatal and adolescent groups, but may occur at any age	Primarily adolescents and adults	In years prior to puberty
Risk factors	Undescended testicle (neonate), rapid increase in testicular size (adolescent), failure of prior orchiopexy	Sexual activity/promiscuity, GU anomalies, GU instrumentation	Predisposing anatomy
Pain onset	Sudden	Gradual	Sudden or gradual
Prior episodes of similar pain	Possible (spontaneous detorsion)	Unlikely unless prior history of the same	Occasional
History of trauma	Possible	Possible	Possible
Nausea/vomiting	Common	Rare	Rare
Dysuria	Rare	Common	Rare
Fever	Rare	Common in advanced disease (epididymo-orchitis)	Rare
Location of swelling/tenderness	Testicle, progressing to diffuse hemiscrotal involvement	Epididymis, progressing to diffuse hemiscrotal involvement	Localized to head of affected testicle or epididymis
Cremasteric reflex	Testicular torsion unlikely if present	Typically present	Typically present
Testicle position	High riding testicle, transverse alignment	Normal position, vertical alignment	Normal position, vertical alignment
Pyuria	Less likely	More likely	Less likely

Laboratory studies

Urinalysis

Findings suggestive of a urinary tract infection (i.e., pyuria, bacteruria, nitrites, leukocyte esterase) may be present in cases of epididymitis.

Complete blood count (CBC)

An elevated white blood cell (WBC) count may be present in the setting of infection or inflammation, making this finding nonspecific and adding little to narrow the differential. Moreover, the time needed to obtain this test and interpret results often delay diagnosis and definitive management. Patients with advanced infections (e.g., scrotal abscess, epididymo-orchitis, or Fournier's disease) may have a markedly elevated WBC count or granulocyte predominance.

Radiologic studies

Ultrasound

A color-flow duplex Doppler ultrasound may help establish a diagnosis (or exclude other possibilities) in indeterminate cases of acute scrotal pain. The classic finding suggestive of testicular torsion is diminished testicular blood flow (Figure 36.7). In epididymitis, perfusion will be normal (or increased due to the vasodilatory action of inflammatory mediators on local vascular beds). An infarcted appendage may be visualized on ultrasound as well, if appendage torsion is responsible for the painful presentation. Ultrasound may also identify hydroceles, hematoceles, varicoceles, hernias, tumors, abscesses and gonadal vasculitis.

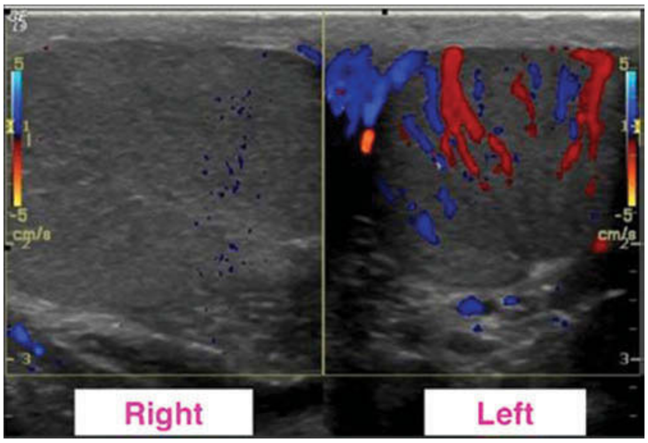

Figure 36.7
Color Doppler sonogram of right testicular torsion. A transverse color Doppler scan of both testes demonstrates normal flow to left testis and complete absence of flow to the right testis due to torsion.

Radionuclide imaging

Given the widespread availability of and expertise with ultrasound technology, the inherent risks associated with radiation exposure, and the lack of availability and time required to obtain the study, radionuclide imaging of the acute scrotum has fallen out of favor at most centers. Findings suggestive of testicular torsion include diminished radionuclide uptake due to compromised blood flow. Likewise, appendage torsion and epididymitis result in preserved (or increased) uptake compared with the unaffected testicle.

Computed tomography

Computed tomography (CT) may assist in the evaluation of GU abscess, Fournier's disease, or acute abdominal pathology as the etiology for an acute scrotum, but is not helpful in diagnosing testicular torsion.

General treatment principles

As with any patient presenting to the ED, priorities begin with the ABCs (airway, breathing, circulation). The primary goals of treatment are physiologic stabilization, symptom relief, administration of timely antibiotics when indicated and, in some cases, preparation for surgical intervention.

Pain relief

Patients with acute scrotal problems typically present with a distressing component of pain. The greatest priority is identifying GU pathology that necessitates rapid surgical intervention. Therefore, initial pain relief should be administered parenterally in order to keep the patient ready for possible surgical intervention. Under no circumstances should analgesia be withheld pending consultation. If the likelihood of surgical intervention is low, and the pain is mild to moderate on presentation, a trial of oral medications can be offered. Agents used most frequently are narcotic analgesics, nonsteroidal antiinflammatory drugs (NSAIDs), and acetaminophen. In addition, the pain of testicular torsion may be relieved by *manual detorsion* of the affected testicle. As the testes most frequently torse towards the midline (in a lateral to medial fashion), detorsion is typically accomplished by rotation of the affected testicle from medial to lateral (like "opening a book"). The end point for an attempt at manual detorsion is either pain relief or pain that is unrelenting (indicating exacerbation of ischemia). Scrotal elevation may help relieve pain in patients with epididymitis. This may be accomplished with a towel roll or supportive undergarment (jock strap). In addition, ice may reduce edema and provide some degree of analgesia.

Antibiotics

Antimicrobial agents are indicated if an infectious process is identified or suspected. Early broad-spectrum antibiotic therapy is imperative in all cases of suspected Fournier's disease. Suggested regimens include extended-spectrum penicillin/beta-lactamase inhibitors (e.g., ampicillin/sulbactam, piperacillin/tazobactam), or third- or fourth-generation cephalosporin plus clindamycin, with the addition of vancomycin to either regimen. Antibiotics are the cornerstone of therapy for epididymitis (Table 36.4). Antimicrobial selection is guided by patient demographics: younger, sexually active males are treated with agents to cover *Neisseria gonorrhoeae* and *Chlamydia trachomatis* (e.g., ceftriaxone IM plus oral doxycycline). Antibiotic therapy in older males is directed at covering common "urinary" pathogens most frequently encountered in this demographic group (e.g., *Escherichia coli* and *Klebsiella*). Caution must be observed when prescribing oral fluoroquinolones due to a slight increase in the likelihood of tendon rupture. Epididymitis may occur in prepubescent males due to reflux of sterile urine into the epididymis (often resulting from minor congenital GU anomalies). Treatment of the resulting "chemical" (non-infectious) inflammation is with agents that cover common urinary pathogens.

Table 36.4 Recommended antibiotic regimens for epididymitis

For acute epididymitis most likely acquired sexually:
Ceftriaxone 250 mg IM in a single dose *plus* Doxycycline 100 mg orally BID for 10 days
For acute epididymitis most likely caused by enteric organisms:
Levofloxacin 500 mg orally QD for 10 days *or* Ofloxacin 300 mg orally BID for 10 days

BID: twice a day; IM: intramuscularly; QD: once a day.

Adapted from Centers for Disease Control and Prevention. Sexually transmitted diseases treatment guidelines, 2010. *MMWR* 2010;59(No. RR-12). Available at: http://www.cdc.gov/std/treatment/2010/STD-Treatment-2010-RR5912.pdf (accessed January 28, 2011).

Special patients

Elderly

The evaluation and management of elderly males with acute scrotal pain follows the same general guidelines as for other patients, with a few notable exceptions. First, accurate historical information may be elusive in patients with age-related cognitive impairment. Also, family members and caregivers may be unaware of complaints or findings associated with the GU region. In addition, geriatric patients have relatively limited physiologic reserve. This creates management challenges (i.e., effects of administered narcotics, antiemetics, IV fluids), challenges in surgical preparation (i.e., need for preoperative medical evaluation), and diminished resistance to rapid progression of various disease states. Institutionalized or sedentary patients may present with acute symptoms of chronic GU pathology, such as strangulation of a chronically incarcerated hernia or coagulopathy-induced bleeding into a hydrocele cavity. An important consideration in any older male patient with testicular discomfort is pain referred from a retroperitoneal process, such as an expanding or ruptured AAA.

Immune compromised

Diabetic and other immune-compromised patients are at increased risk for infections of the scrotum. Fournier's disease is more likely in this group, and may present with significant perineal pain in the absence of notable scrotal pathology (pain "out of proportion" to physical findings). GU examination revealing scrotal erythema, ecchymosis, or subcutaneous emphysema (resulting from gas-forming organisms) may indicate advanced disease. Fournier's disease requires aggressive hydration, broad-spectrum antibiotics, and emergent surgical debridement.

Pediatric

Evaluation and management of pediatric patients present unique challenges. Preverbal children may be unable to express the location and nature of their pain. Completely undress all patients with an unclear presentation or uncertain diagnosis. In infants, acute scrotal pain from testicular torsion or incarcerated inguinal hernia may cause inconsolable crying. Also, consider sexual abuse in prepubescent males presenting with sexually transmitted infections or GU trauma, such as human bites or contusions to the scrotum or perineum. In addition, care must be taken to respect and address privacy issues, especially in the adolescent age group. In some cases, a caregiver may be uncomfortable discussing their child's problem. A useful approach to facilitating a more comprehensive history and examination is to first interview, examine, and discuss with the peri- or post-pubescent male alone, then offer to speak with all parties.

Disposition

Several urologic emergencies require immediate evaluation by a urologist in the ED. Emergent conditions discussed in this chapter include known or suspected cases of testicular torsion, Fournier's disease, incarcerated or strangulated hernia, and AAA (vascular surgery should be consulted if AAA is suspected). In addition, a low threshold for urology consultation in any case of GU trauma (blunt or penetrating) is prudent.

Patients with acute scrotal pain who don't have conditions requiring emergent consultation can be referred for urgent primary care or specialty follow-up. When in doubt, err on the side of caution and obtain appropriate consultation in the ED. Another approach is to obtain urgent follow-up in an appropriate clinic by discussing the case with an on-call specialist once emergent conditions have been reliably excluded.

Address pain issues by providing options for adequate analgesia, including over-the-counter analgesics or prescription narcotics. Also, be certain to provide outpatient antibiotics when indicated.

Patients with unclear diagnoses, intractable pain or vomiting, unreliable follow-up, or an unstable social situation may require inpatient management by either a primary care provider or appropriate specialist.

Pearls, pitfalls and myths

- In cases of suspected torsion, emergent specialist consultation is imperative. Remember that "time is testicle." Be careful to avoid "castration by procrastination."
- Maintain a high index of suspicion for testicular torsion in all age groups, even though its peak incidence occurs in adolescents and neonates.
- Any asymptomatic testicular mass, firmness, or induration is a malignancy until proven otherwise.
- Ultrasound examination (especially with Doppler) is extremely useful to differentiate etiologies of acute scrotal pain.
- The appearance of overt physical findings in Fournier's disease may be a harbinger of terminal disease progression. The hallmark of this disease is pain "out of proportion" to physical findings in any high-risk (i.e., diabetic or other immune compromised) patient. A high index of suspicion must be maintained at all times and emergent consultation obtained.
- A urologist should be consulted in all but the most trivial cases of GU trauma and unclear emergencies of the scrotum to help guide treatment and disposition.

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37 Seizures

Mary Beth Johnson, MD and Stephen R. Hayden, MD

Scope of the problem

Seizures are common presenting complaints to the emergency department (ED). One in 20 persons will have a seizure during his or her lifetime. This increases to one in 11 in a person who has reached the age of 80 years. Adults presenting to the ED with a first-time seizure account for nearly 1% of annual ED visits. Seizures have a bimodal distribution, declining from older childhood until age 60 years, when the incidence increases again. Febrile seizures occur in approximately 3% of children and account for approximately 30% of all childhood seizures. Noncompliance with anticonvulsant medications is the most common cause for seizures in adults less than 60 years of age. The most common cause of seizures in adults over 60 years is stroke, followed by malignancy. The mortality rate from seizures is between 1% and 10%, with the highest mortality due to status epilepticus (SE). Seizures can cause permanent neurologic sequelae.

Anatomic essentials

A seizure occurs when there is excessive, abnormal cortical activity. The clinical manifestations depend on the specific area of the brain cortex involved. A distinction is often made between primary and secondary seizures. *Primary seizures* are seizures recurring without consistent provocation or cause. *Secondary seizures* occur as a response to certain toxic, metabolic, or environmental events (Table 37.1). *Generalized seizures* occur from an electrical event that simultaneously involves both cerebral hemispheres and is accompanied by loss of consciousness. These can be convulsive (grand mal seizures) or nonconvulsive (absence seizures). *Partial seizures* involve one cerebral hemisphere, and can be divided further into those in which consciousness is maintained (simple partial) and those in which consciousness is abnormal (complex partial).

Proposed mechanisms for a seizure include the disruption of normal anatomical cortical structure or disruption of local metabolic or biochemical function of neuronal cells. Either of these mechanisms can produce sustained depolarization of neuronal cells, creating an ictogenic focus, followed by recruitment of adjacent cells. When the electrical discharge extends below the cortex to the reticular activating system, consciousness may become impaired. Most commonly, seizures are self-limited, with the sustained electrical activity resolving spontaneously. *Status epilepticus* is defined when the abnormal electrical activity is sustained for more than 5 minutes, and may occur with any type of seizure. This recent definition more accurately reflects the relatively short time before neuronal injury is likely to occur. Furthermore, seizure activity lasting more than 5 minutes is unlikely to terminate spontaneously even if the type of seizure would generally resolve on its own prior to this.

Table 37.1 Etiology of secondary seizures

Metabolic causes
• Hypoglycemia • Hyponatremia/hypernatremia • Hypocalcemia • Hypomagnesemia • Renal failure/dialysis complications • High anion gap acidosis • Thyroid disease
Infectious causes
• Meningitis • Encephalitis • CNS abscess • HIV disease • Syphilis • Toxoplasmosis • Neurocysticercosis
Degenerative CNS disease
• Alzheimer's disease • Neurofibromatosis • Tuberous sclerosis • Multiple sclerosis
Drugs and toxins
• Noncompliance with anticonvulsants • Withdrawal of EtOH/sedative/hypnotic • Cocaine • Methamphetamines • PCP • Anticholinergic agents (including tricyclic antidepressants) • Salicylates • Lithium • Isoniazid • Tramadol • Bupropion
Anatomic causes
• Post-traumatic • CNS neoplasms • CNS vasculitis (SLE, polyarteritis) • Arteriovenous malformation • Stroke (CVA)
Miscellaneous causes
• Febrile seizures • Eclampsia • Cerebral arterial gas embolism • Pseudoseizures

CNS: central nervous system; CVA: cerebrovascular accident; EtOH: ethanol; HIV: human immunodeficiency virus; PCP: phencyclidine; SLE: systemic lupus erythematosus.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history

Table 37.2 Seizures red flags

History	Concerning diagnosis
Fever, headache, neck stiffness	Meningitis, abscess, febrile seizure
History of trauma	Hemorrhage, traumatic brain injury
Vomiting	Mass lesion, tumor, aspiration pneumonia
Overdose, ingestion	Hypoglycemia, tricyclic antidepressant, isoniazid
Medications	Adverse drug effects, hypoglycemia, reduced seizure threshold
Alcohol, benzodiazepine or drug abuse	Withdrawal seizures
Altered mental status	Brain injury, postictal period, hypoglycemia, hypoxia
Multiple seizures, prolonged	Status epilepticus
Neurologic complaints	Mass lesion, tumor, Todd's postictal paralysis
New onset	Epilepsy, seizure disorder
Coagulopathy or anticoagulation	Hemorrhage
Examination finding	**Concerning diagnosis**
Signs of trauma	Traumatic brain injury, hemorrhage
Petechial rash, coagulopathy	Meningitis, meningococcemia
Vesicular rash	Herpes encephalitis
Rash (esp. palms and soles), bubo	Neurosyphilis
Abnormal neurologic or mental status examination	Tumor, mass effect, focal lesion, ingestion, hypoglycemia, stroke, hemorrhage
Hypertension, tachycardia, piloerection	Alcohol, benzodiazepine or drug withdrawal

and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 37.2).

History

Do you have a prior history of seizures?

The work-up for a patient with an established seizure disorder differs significantly from that of a patient who presents with a first-time seizure. New-onset seizures require a more extensive evaluation for the conditions in Table 37.1.

How long did the seizure last? How many seizures did you have?

This information may need to come from witnesses. It may be helpful to ask the patient "What is the last thing you remember clearly before the event?" and "What is the very next thing you remember clearly after the event?" These answers may give an estimate of the elapsed time. The duration of the event and whether the seizures were recurrent provide important information regarding the severity of the seizure. Historically, seizure activity lasting more than 30 minutes, or recurrent seizures without clearing of mental status between successive seizures, defined SE. This definition has been revised by many experts to only 5 minutes of continuous seizure activity, or recurrent seizures without a return to consciousness between them.

Has there been a recent change in your seizure pattern?

A significant change in seizure pattern would require a work-up similar to that for first-time seizures.

Did you hit your head prior to having a seizure?

It is important to distinguish between a patient who sustains head trauma followed by a seizure and a patient who has a seizure, then falls and sustains head trauma. Even in patients with known seizure disorders, those who hit their head immediately prior to a seizure have a higher incidence of significant abnormalities on head computed tomography (CT).

What medications are you taking?

Many medications are associated with seizures at toxic doses. Some medications lower the seizure threshold at non-toxic doses. It is important to know if the patient is taking anti-seizure medications in order to measure medication levels. Noncompliance with anticonvulsants is the most common cause of recurrent seizures in known epileptics. Use of anticoagulants such as warfarin should increase suspicion for spontaneous or traumatic intracranial bleeding.

Did you have an aura or premonition prior to the event?

Many patients will describe an aura prior to having a seizure. Symptoms may include paresthesias, abnormal odors or sounds, flashing lights, or scotoma.

Do you remember what you were doing immediately preceding your loss of consciousness?

Retrograde amnesia of activities preceding the event is more indicative of a seizure.

Did you bite your tongue? Were you incontinent of urine?

These are important questions to ask, as these occurrences are more likely with a seizure.

Did anyone witness the seizure? How long did you feel confused after the event?

If possible, obtain history from the paramedics or other witnesses of the event. Try to determine if tonic or clonic motor activity occurred, if the patient maintained consciousness during the event, or if there was focality to the neurologic symptoms. A postictal period of 30 minutes is not unusual after a seizure.

Do you drink alcohol daily, regularly, or heavily?

If the answer to any of these questions is yes, then ask *"When was your last drink?"* Seizures are a common manifestation of alcohol withdrawal. Furthermore, patients who drink regularly are at greater risk for falls (resulting in subdural hematomas) or hyponatremia.

Do you use recreational (street) drugs?

Common drugs of abuse such as cocaine, crystal methamphetamine, phencyclidine (PCP) and ecstasy may induce seizures.

Have you had any prolonged stays in another country?

Any recent travel to or from another country is important to determine. Neurocysticercosis is the leading cause of adult-onset epilepsy in areas of the world where it is endemic, particularly Latin America, Asia and Africa.

Associated symptoms

Hypertension, tachycardia, muscle soreness and headache are commonly associated with seizures. Elicit symptoms of pain or soreness after the seizure. A posterior shoulder dislocation, often not discovered until several days after the event, is commonly missed following a prolonged tonic–clonic seizure. Symptoms such as headache occurring prior to seizure activity could prompt investigation of hemorrhagic brain pathology.

Past medical

In addition to determining any prior history of seizures, it is important to ascertain a prior history of renal or liver disease, malignancy, diabetes, cardiovascular disease, hypertension, human immunodeficiency virus (HIV) risk factors, drug or alcohol abuse, recent trauma, degenerative central nervous system (CNS) diseases, prior neurosurgery, psychiatric history, family history of seizures, and pregnancy, as each of these is associated with secondary seizures. A previous diagnosis of neurocysticercosis is important to elicit as well.

Physical examination

The primary purpose of the physical examination is to evaluate for focal neurologic deficits or other findings that might suggest a secondary cause for seizures. Careful physical examination will also reveal if the patient (assumed to be postictal) is actually still seizing. Thorough examination should also identify sequelae of the seizure activity.

General appearance

The physical examination is often normal in the seizure patient. Of primary importance in the immediate postictal period is whether the patient is adequately protecting the airway. Level of consciousness will often be depressed; therefore, airway patency should be evaluated and maintained. On occasion, patients may bleed profusely from oral trauma, requiring that the airway be secured. Urinary incontinence may support the diagnosis of a seizure.

The postictal state is often characterized by decreased responsiveness, disorientation, amnesia and headache. The mental status examination should gradually improve over time, ranging from a few moments to an hour or more. This may not be consistent between patients or even between seizures in the same patient. If mental status does not improve appropriately, further investigation should occur. Some patients in nonconvulsive SE are mistakenly assumed to be postictal instead of actively seizing.

Vital signs

Following a seizure, patients are frequently hypertensive, tachycardic, tachypneic, and may have low oxygen saturation. These abnormalities should improve as the patient recovers. Mild hyperthermia may occur, but a significantly elevated temperature or persistently abnormal vital signs should prompt further investigation for a secondary cause of the seizure.

Head and neck

Head and neck examination should focus on identifying signs of head trauma, such as cephalohematoma, depressed skull fracture, lacerations, abrasions and hemotympanum. Pupils should be evaluated for reactivity and symmetry, and cranial nerve abnormalities should be sought. Anisocoria may suggest impending herniation through the foramen magnum. Nystagmus, especially rotary, is associated with certain drugs such as PCP. Persistent eye deviation may indicate ongoing seizure activity. The oropharynx must be examined for signs of oral trauma. The presence of bites or contusion to the lateral tongue strongly suggests that a seizure occurred. The finding of oral thrush may suggest that the patient is immunocompromised. *Automatisms* (repetitive actions such as lip smacking, swallowing, or chewing) are frequent in complex partial seizures and may be the only indication of ongoing seizure activity. The cervical spine should be palpated for midline tenderness or deformity. If evidence of neck trauma is not present, the neck should be evaluated for meningismus.

Pulmonary

Pulmonary examination should assess symmetry and character of breath sounds, looking for aspiration that may have occurred during the seizure. Neurogenic pulmonary edema can occur following a seizure, but is usually mild and self-limited. It is characterized by diffuse crackles, hypoxia, and the absence of signs of congestive heart failure.

Cardiovascular

Cardiac examination may reveal a heart murmur, possibly indicating subacute bacterial endocarditis (SBE) with resultant embolization as the cause of the seizure. An irregular heart rate or carotid bruits may accompany a stroke, which is a common cause of new-onset seizures in the elderly. A transient episode of ventricular irritability or decreased heart rate may cause an episode of diminished cerebral hypoperfusion and syncope, which may mimic or result in seizure activity.

Neurologic

A detailed neurologic examination must be performed in all seizure patients. Carefully assess for focal motor or sensory deficits and abnormalities of cerebellar function. Postictal paralysis (Todd's paralysis) may follow generalized or partial seizures. This is typically characterized by weakness of one extremity or complete hemiparesis. Symptoms may persist for up to 24 hours. Todd's paralysis indicates a higher likelihood of an underlying structural cause for the seizure and should therefore be investigated. Tremulousness of the extremities and tongue, in conjunction with other stigmata of ethanol abuse, suggest an alcohol withdrawal seizure.

Extremities

Examination of the extremities may demonstrate evidence of trauma. Particular attention should be paid to identifying shoulder dislocations (seizures are a common cause of posterior shoulder dislocations).

Skin

The skin should be examined for lesions consistent with infections, such as meningococcemia or SBE (splinter hemorrhages, Janeway lesions, Osler's nodes). Evidence of underlying liver disease or coagulopathy should be sought. Similarly, skin lesions such as axillary freckling, café au lait spots, port wine nevi, or subcutaneous nodules may indicate a neurocutaneous disorder. Needle tracks indicate IV drug use, which may suggest a toxicologic or infectious etiology for seizures.

Differential diagnosis

Even when witnessed, other abnormal states of consciousness can produce twitching or jerking movements that can be difficult to distinguish from a seizure. In 40–50% of cases of vasovagal syncope (fainting), brief twitching movements occur. In general, the tonic or clonic movements associated with a seizure are much more forceful and prolonged, and are often associated with a postictal state, urinary incontinence, or tongue biting.

Table 37.3 Differential diagnosis of seizure

Diagnosis	Classic symptoms	Signs	Work-up
Breath-holding spells in infants	Usually brought on by anger, fear, frustration Pallor or cyanotic spells Apnea or bradycardia	Normal neurologic examination	ECG to check QT interval EEG during spells
Extrapyramidal reactions	Tongue deviation Torticollis Jerking, random limb movements History of taking phenothiazines/butyrophenones	Level of consciousness usually maintained	Clinical diagnosis in setting of taking specific medications
Hyperventilation syndrome	Shortness of breath followed by paresthesias, carpopedal spasm and loss of consciousness	Increased respiratory rate Normal neurologic examination	Typically clinical diagnosis, ABG may be useful
Psychogenic seizures	May closely mimic symptoms of true seizure, often in the setting of emotional distress	Usually quick return to normal mental status post-event	Continuous EEG monitoring during events
TIA/CVA	Sudden onset of weakness, difficulty speaking, or walking	Abnormal neurologic findings	Head CT, ECG, laboratories
Vasovagal syncope	Brief twitching movements Classic prodrome includes lightheadedness, tunnel vision	No postictal state Lack of tongue biting	ECG, glucose testing, pregnancy test (if appropriate)

ABG: arterial blood gas; CT: computed tomography; CVA: cerebrovascular accident; ECG: electrocardiogram; EEG: electroencephalogram; TIA: transient ischemic attack.

Other causes of sudden loss of consciousness that resemble a seizure include hyperventilation syndrome, prolonged breath-holding spells in infants, numerous toxic and metabolic states, transient ischemic attack (TIA), cerebrovascular accident (CVA), narcolepsy and causes of syncope (Table 37.3). Extrapyramidal reactions, especially in children, may be confused with seizure activity. Psychogenic states, in particular pseudoseizures, may be difficult to distinguish from true seizure activity. Continuous electroencephalography (EEG) monitoring during such episodes may be necessary to establish a correct diagnosis. Witnessed cardiac arrest is often initially mistaken as seizure activity.

Diagnostic testing

The diagnostic work-up for patients presenting to the ED with seizures depends on whether it was a first-time seizure, what underlying conditions the patient has, and whether a clear seizure precipitant (hypoglycemia, hypoxia, intoxication, withdrawal state) is identified (Figure 37.1).

Laboratory studies

Complete blood count

The complete blood count (CBC) is rarely useful in a seizure patient. The white blood cell (WBC) count is often elevated from demargination during the seizure. However, a CBC is often ordered in patients presenting with a seizure when the mental status is not returning to normal, the patient has HIV or immunosuppression (to determine the absolute neutrophil count), or the patient has a platelet disorder or coagulation abnormality. In febrile patients, including young children with a febrile seizure, a CBC may be ordered as part of the evaluation for the underlying source of infection.

Electrolytes

Electrolytes, especially sodium, calcium and magnesium, are indicated for patients with a first-time seizure or alcoholics who likely have nutritional deficiencies. Additionally, any patient in whom the mental status is not returning to normal following seizure activity should have electrolytes checked as part of the work-up. Children less than 1 year of age who have had a seizure in the absence of fever should also have electrolytes checked.

Blood glucose

All patients presenting with a seizure should have a rapid bedside glucose performed and confirmed in the lab.

Cerebrospinal fluid analysis

A lumbar puncture (LP) is indicated in a seizure patient when the mental status fails to return to normal, if a subarachnoid hemorrhage is suspected and neuroimaging is negative (e.g., sudden onset of the worst headache in conjunction with a seizure), or when a CNS infection is suspected (patients with fever, headache or stiff neck). In addition, cerebrospinal fluid (CSF) analysis may be helpful for HIV or immunocompromised patients who are at risk for opportunistic CNS infections (diagnosed only from CSF analysis). CSF analysis may also be helpful in the evaluation of a febrile infant who has had a seizure. Traditionally, an LP was recommended following a febrile seizure in the age group less than 12 months. However, newer evidence-based guidelines suggest that for infants with a febrile seizure who otherwise appear well and with no history of recent antibiotic use, routine LP is not indicated. If the child appears ill, the mental status does not return to normal, or the neurologic examination has focal deficits, an LP should be performed.

Anticonvulsant levels

Anticonvulsant levels are indicated in seizure patients who report taking medications for which a therapeutic level can be determined. These include valproic acid, carbamazepine, phenytoin and phenobarbital.

Toxicology screening

Toxicology screening should be performed when the mental status is not returning to normal after a seizure. In the setting of specific toxic exposures for which toxin levels can be determined (e.g., carbon monoxide), toxicologic screening may be useful. For first-time seizures, if the patient can provide an appropriate history, routine toxicology screening is of little value.

Creatine phosphokinase

Creatine phosphokinase (CPK) levels may be important in patients with SE, seizures resulting from toxic ingestions (especially sympathomimetics), or a prolonged downtime before coming to the ED.

Pregnancy testing

Pregnancy testing should be performed in any woman of childbearing age in whom the mental status does not return to baseline after the seizure (consider eclampsia in late pregnancy or in the immediate postpartum period, especially in obese patients). Additionally, pregnancy testing should be considered prior to initiating new medications if the possibility of pregnancy exists.

Coagulation

Coagulation studies should be performed if the patient with a seizure has a known coagulation disorder, is on anticoagulant medication, or has any condition that might be expected to alter coagulation parameters (i.e., liver disease or alcoholism).

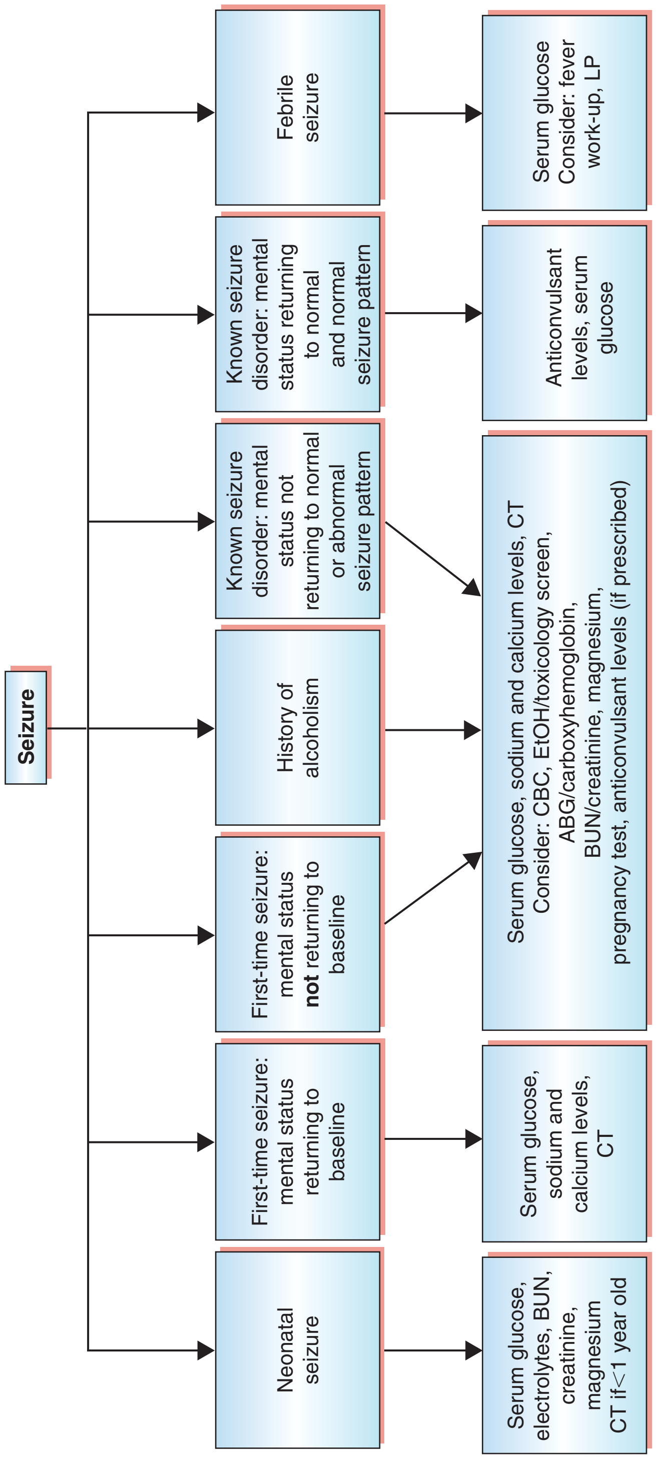
Seizure
Neonatal seizure
First-time seizure: mental status returning to baseline
First-time seizure: mental status **not** returning to baseline
History of alcoholism
Known seizure disorder: mental status not returning to normal or abnormal seizure pattern
Known seizure disorder: mental status returning to normal and normal seizure pattern
Febrile seizure
Serum glucose, electrolytes, BUN, creatinine, magnesium CT if<1 year old
Serum glucose, sodium and calcium levels, CT
Serum glucose, sodium and calcium levels, CT Consider: CBC, EtOH/toxicology screen, ABG/carboxyhemoglobin, BUN/creatinine, magnesium, pregnancy test, anticonvulsant levels (if prescribed)
Anticonvulsant levels, serum glucose
Serum glucose Consider: fever work-up, LP

Figure 37.1
Algorithm for the evaluation of the patient with seizures. ABG: arterial blood gas; BUN: blood urea nitrogen; CBC: complete blood count; CT: computed tomography; EtOH: ethanol; LP: lumbar puncture. Adapted from Bradford JC, Kyriakedes CG. Evaluation of the patient with seizures: An evidence based approach. *Emerg Med Clin North Am* 1999;17(1):203–20.

Radiologic studies

Chest radiograph

A portable (AP) chest radiograph should be obtained if there is concern for pulmonary infection or lung mass. Aspiration is a risk in seizure patients, although a chest radiograph may be normal initially after a small episode. However, in patients with recurrent seizures, aspiration does occur and may show up on chest film.

Computed tomography

CT should be performed in any patient with a first-time seizure, a patient whose mental status fails to return to normal, the setting of neonatal seizures without fever, seizures following head trauma, patients with coagulation disorders or on anticoagulants, patients with known or suspected malignancy, or patients with an abnormal neurologic examination. CT is also indicated in a patient at risk for neurocysticercosis.

Magnetic resonance imaging

If available, many neurologists prefer MRI over CT for new-onset seizures if mental status has returned to normal.

Additional plain films

Plain films should be obtained if physical examination suggests a fracture or dislocation from trauma prior to or during seizure activity.

Other studies

An electrocardiogram (ECG) should be performed if the seizure is believed to be due to drug or toxin exposure (especially tricyclic antidepressants), or if the patient has underlying comorbid conditions that increase the risk of coronary artery disease or dysrhythmias.

An EEG can be performed as part of the work-up of a seizure as an outpatient for first-time seizures, or more emergently at the bedside when the mental status does not return to baseline (to evaluate for nonconvulsive SE). EEG is necessary in the setting of SE requiring general anesthesia and paralysis or barbiturate coma.

Diagnostic considerations

For *first-time seizures* without an obvious cause, with mental status returning to normal, current evidence-based guidelines suggest that serum glucose, sodium and calcium levels be obtained, and a head CT performed. An LP is indicated only if an adult has a fever without an obvious source, or to evaluate for possible subarachnoid hemorrhage. The question arises whether the head CT needs to be performed during the ED visit on all seizure patients. The decision must be based on the patient's current condition, access to medical care, reliability of follow-up, and their ability to understand the nature of their medical condition and follow-up instructions. It is often expeditious to perform a head CT (or MRI) in the ED. If the mental status does not return to normal, then the work-up should also include a CBC, alcohol/toxicology screening, blood urea nitrogen (BUN), creatinine, magnesium level, ECG, pregnancy test, and arterial blood gas (ABG) with carboxyhemoglobin level if clinically warranted. In this case, a head CT should be performed urgently in the ED. Although unlikely, an EEG may be performed if available.

For patients with a *known seizure disorder,* typical seizure pattern and mental status returning to normal, rapid glucose testing and appropriate serum anticonvulsant levels are recommended. For a known seizure patient who has an abnormal seizure pattern or mental status that does not return to normal, the work-up is identical to that of a first-time seizure patient whose mental status is not returning to normal, with anticonvulsant levels tested.

For a known *alcoholic* in whom an alcohol withdrawal seizure is suspected and mental status is returning to baseline, rapid (and repeated) glucose determination and anticonvulsant levels may be performed if the patient takes anticonvulsants. Routine laboratory testing is usually unnecessary unless performed for a more generalized health status evaluation or if the patient has significant comorbidities. If there is concomitant evidence of head trauma, a head CT is indicated. If the mental status does not return to normal, the work-up should proceed as for a first-time seizure patient whose mental status does not return to normal.

Febrile seizures in the pediatric population are relatively common occurrences, especially if there is a history of febrile seizures in a first-degree relative. For an otherwise healthy, well-appearing child with a febrile seizure, serum glucose testing should be obtained. Other diagnostic tests to evaluate an occult source of fever (CBC, blood cultures, urinalysis [UA] and culture, chest radiograph, and stool studies) should be considered. Traditionally, some authorities have recommended LP for children less than 12 months old, and as an option in children less than 18 months of age. Current evidence-based guidelines, however, do not recommend routine LP for febrile seizures unless the patient has a history of recent antibiotic use. If a child appears ill or their mental status does not improve after the febrile seizure, a comprehensive fever work-up including LP is warranted in addition to considering the tests outlined for a first-time seizure patient whose mental status is not returning to normal.

Neonatal seizures without fever need to have a variety of diagnostic tests performed, including serum glucose, electrolytes, BUN, creatinine, calcium and magnesium levels, and head CT if the child is less than 1 year old.

A number of conditions deserve special consideration in the setting of a seizure. If the patient has a history or findings of coagulopathy, platelet disorders, or is taking anticoagulant medication, the work-up should include a CBC, coagulation studies and head CT. In patients with history of renal failure, BUN, creatinine and electrolytes should be tested. Patients who are known or suspected HIV-positive or immunosuppressed should have a CBC and head CT preceding an LP. For toxic exposures or ingestions, consider an ECG; specific drug, alcohol, acetaminophen and salicylate levels; and toxicology screens. Patients with a history of malignancy or recent head trauma should have a head CT performed.

General treatment principles

Status epilepticus

SE is a life-threatening emergency with potential for airway compromise, severe hypoxia and neurologic injury. Whether the seizure activity is continuous or recurrent without clearing of the mental state, assume the airway is compromised. Active measures should be taken to secure the airway. Placing the patient in the left lateral decubitus position, removing dentures, and carefully suctioning vomit and saliva are appropriate initial steps. If the airway cannot be adequately protected by these simple measures, endotracheal intubation may be necessary. A rapid acting benzodiazepine (e.g., midazolam) is a good choice as the induction agent, as it may terminate seizure activity. If tonic activity or trismus prevents adequate opening of the mouth, paralytics may be necessary. Bear in mind that once the patient is paralyzed, tonic–clonic activity will cease, but the abnormal cortical neuronal activity may continue (i.e., the patient continues to seize without demonstrating muscular activity). Second or third-line anticonvulsants may be necessary, and EEG monitoring will be required as soon as practical.

SE is often the result of a secondary cause for seizures (Table 37.1). These conditions may be rapidly reversible and must be diagnosed early and corrected immediately (i.e., hypoglycemia, hyponatremia and hypoxia). Structural abnormalities (i.e., subarachnoid or intracranial hemorrhage) and toxic states may be present; these conditions should be considered early and appropriate management instituted (e.g., isoniazid [INH] overdose would be treated with the antidote pyridoxine). Concomitant with the work-up for these diseases, abortive therapy may be instituted.

Pharmacologic abortive seizure therapy

In general, the first-line medication for terminating seizure activity in the ED is intravenous (IV) or intramuscular (IM) benzodiazepines (Table 37.4). IV access must be established if not already accomplished, which can be extremely difficult. Diazepam can be administered via an endotracheal tube, rectally, or through an intraosseous line. Benzodiazepines enhance gamma amino butyric acid (GABA)-mediated inhibition, and are effective in terminating seizures in 80–90% of cases. Diazepam, lorazepam and midazolam are commonly used in the ED to abort seizures because of their rapid onset and efficacy.

Common second-line pharmacologic agents used in the ED for seizures include phenytoin (or the pro-phenytoin drug fosphenytoin) and phenobarbital. Phenytoin suppresses neuronal recruitment around the seizure focus. The propylene glycol diluent with phenytoin may cause hypotension and cardiac dysrhythmias if administered too rapidly IV. Fosphenytoin can be administered quickly IV without deleterious effect, and has the additional advantage of IM administration if IV access is a problem. Phenobarbital is a CNS depressant that decreases neuronal electrical activity at the seizure focus as well as terminates recruitment. Most experience with phenobarbital as a second-line agent to terminate seizures is in the pediatric population. Barbiturates have the adverse effects of significant sedation, hypotension and respiratory depression; patients must therefore be monitored closely.

If seizure activity is not suppressed by the above measures, additional therapeutic options in the ED include using valproate, barbiturate coma, or general inhalational anesthesia. Valproate may be administered rectally, and a parenteral form exists (Depacon). A recent meta-analysis, however, found that there are certain risks associated with IV Depacon, including neurotoxicity; it must be administered over 60 minutes. This review concluded that the evidence is too limited to routinely recommend IV valproate unless it is not desirable to induce a barbiturate coma or begin inhalational anesthesia. Levetiracetam (Keppra) was approved for IV use in 2006. Although no randomized controlled trials have been performed to date,

Table 37.4 Pharmacologic treatment of seizures

Drug	Adult dose	Pediatric dose
Diazepam	5–10 mg IV every 5–10 min	0.2–0.5 mg/kg IV/PR/ETT/IO
Lorazepam	2–4 mg IV every 5–10 min	0.05–0.1 mg/kg IV (maximum 4 mg)
Midazolam	2.5–15 mg IV or IM (unpredictable absorption IM)	0.15 mg/kg IV then drip 2–10 mcg/kg/min *or* 0.15 mg/kg IM (unpredictable absorption)
Phenytoin	1 g IV load at 50 mg/min	20 mg/kg IV at 1 mg/kg/min
Fosphenytoin	15–20 mg PE/kg IV/IM single loading dose	10–20 mg PE/kg IV/IM single loading dose
Phenobarbital	10–20 mg/kg IV at 60–100 mg/min	10–20 mg/kg IV × 1, then 5–10 mg/kg IV every 15–30 min
Pentobarbital	10–15 mg/kg IV first 1–2 hrs then 1 mg/kg/hr IV	10–15 mg/kg IV first 1–2 hrs then 1 mg/kg/hr IV
Valproate	20 mg/kg PR or 15 mg/kg IV (maximum 250 mg)	20 mg/kg PR or 15 mg/kg IV (maximum 250 mg)
Levetiracetam	1500 mg IV	20 mg/kg/day initial dose

ETT: endotracheal tube; IM: intramuscular; IO: intraosseous; IV: intravenous; PE: phenytoin equivalents; PR: per rectum.

Table 37.5 Pediatric seizures in children

Seizure type	Clinical findings	Work-up and management
Febrile seizure	Age <5 years Well appearing Lack of signs of serious bacterial illness Normal neurologic examination	CBC, blood cultures, UA, CXR if indicated to evaluate for underlying infection LP if recent antibiotic course (some recommend LP routinely for age <1 year)
Morning after seizure	Ambulatory child drinks leftover alcohol from a party, becomes hypoglycemic and seizes	History is key, parental education
Ketotic hypoglycemia	Children 6–18 months Periods of caloric deprivation lead to profound hypoglycemia	Rapid glucose testing Urine dipstick for ketones Consider chemistry panel
Absence seizures	Staring episodes Automatisms Appear awake but are not responsive No postictal period	Electrolytes, glucose Head CT (or MRI) EEG
Infantile spasms	History of congenital abnormality or perinatal brain injury Myoclonic seizures in clusters Development delay Age <1 year	Head CT (or MRI) Electrolytes, glucose EEG (high voltage chaotic slowing, hypsarrhythmia)
Status epilepticus	Seizure activity for >5 minutes Series of seizures; mental status does not return to normal between events	Head CT, electrolytes, glucose, CBC, toxicology screening, ECG and EEG

CBC: complete blood count; CT: computed tomography; CXR: chest X-ray; ECG: electrocardiogram; EEG: electroencephalogram; LP: lumbar puncture; MRI: magnetic resonance imaging; UA: urinalysis.

several case-control studies have demonstrated efficacy of levetiracetam in SE when first-line therapies have failed.

The typical agent used to induce barbiturate coma is pentobarbital. Pentobarbital suppresses all cortical neuronal activity, decreases intracranial pressure (ICP), and increases cerebral perfusion. It also induces respiratory depression or arrest and causes hypotension; therefore, patients must be intubated and monitored closely.

Isoflurane inhalational anesthesia is another option for seizure activity that does not respond to first- or second-line agents or treatment of the underlying cause. Isoflurane suppresses electrical seizure foci and is titratable. It is preferred that patients are intubated for airway protection before instituting isoflurane anesthesia to terminate seizures. Inhalational anesthesia is usually reserved for patients admitted to the intensive care unit (ICU) or operating room, unless there is a significant delay in transferring the patient to these settings.

Special patients

Pediatric

Febrile seizures (by definition) in children are associated more with the rapidity of temperature rise than the absolute temperature. In determining the work-up for a child with a febrile seizure, clinicians should be guided by the general appearance of the child and consider the underlying cause of the fever. Generally, anticonvulsant therapy is not instituted for simple febrile seizures. It is important to educate parents on appropriate temperature control measures, including alternating doses of acetaminophen (15–20 mg/kg/dose orally [PO] or per rectum [PR] every 4–6 hours) and ibuprofen (5–10 mg/kg/dose PO every 6–8 hours). Additionally, the child can have frequent sponge baths with tepid water, as this is a very effective cooling measure.

In children with afebrile seizures, many of the same diagnostic and therapeutic considerations are present as with adults (Table 37.5). Two special causes of childhood seizures are worth mentioning. The first is the “morning after” seizure caused by drinking alcohol left within reach of children at a party, leading to hypoglycemia and seizures. Another cause of hypoglycemia-induced seizures in children is *ketotic hypoglycemia.* Periods of caloric deprivation cause abnormally low blood glucose levels and seizures. This occurs in young children between 6 and 18 months of age when the interval between feedings is increased. The diagnosis is suggested in the ED when typical symptoms occur, and hypoglycemia and ketonuria are present.

Alcoholic

Alcohol withdrawal seizures may occur 6 hours or more after abrupt cessation of drinking. They are typically generalized and recurrent. With each episode of alcohol cessation, the seizure threshold may decrease, increasing the risk and severity of seizures. Other symptoms include those associated with autonomic instability, such as tachycardia, hypertension, mild hyperthermia and CNS activation, including tremor and jerking movements. If the patient’s mental status is significantly altered or confused, *delirium tremens* ensues. This carries a mortality rate as high as 20–30% in some series.

Alcoholics are at risk for other secondary causes of seizures, such as hypoglycemia, intracranial bleeding, other drugs/toxins, and electrolyte disturbances (especially hypomagnesemia or hypocalcemia). Treatment of alcohol withdrawal seizures includes benzodiazepines (often in high doses), which substitute for the GABA-enhancing effect of ethanol. Most patients with alcohol withdrawal seizures do not need second-line treatments such as phenytoin.

Pregnant

Seizures in pregnancy are generally of two categories: *gestational,* in which underlying seizure disorder or anticonvulsant levels are adversely impacted by hormonal and metabolic changes of pregnancy; and *eclamptic,* associated with hypertensive encephalopathy, proteinuria, edema and seizures. Eclampsia generally occurs after the 20th week of pregnancy. The likelihood decreases after delivery, though seizures may occur days to weeks following delivery. It may not be obvious that a woman is more than 20 weeks pregnant, so all women of childbearing age presenting in SE should be evaluated for pregnancy. The treatment of seizures in the setting of eclampsia is controversial; obstetrics or perinatology specialists should be consulted early. In addition to the same management considerations for patients in SE previously outlined, high-dose parenteral magnesium sulfate therapy (4 g IV bolus followed by 2 g/hour infusion) has traditionally been used to help control seizure symptoms. Careful monitoring of respiratory status and reflexes is essential when giving magnesium.

Cerebral arterial gas embolism (CAGE)

When the cerebral circulation is showered with air bubbles, a common manifestation is abrupt seizure activity. In self-contained underwater breathing apparatus (SCUBA) divers, rapid ascent from depth while holding their breath may lead to pulmonary overinflation, tear of the bronchoalveolar sheath and air entering the pulmonary venous system, return of air to the left side of the heart, and systemic embolization of air bubbles. The hallmark of CAGE is the abrupt loss of consciousness with the onset of seizures or other neurologic symptoms upon surfacing. On the other hand, decompression sickness typically has a latent period before the development of neurologic symptoms and does not usually present with seizures. Other causes of CAGE include pulmonary overinflation from positive pressure ventilation, air entry during catheterization or other vascular procedures, brain surgery, cardiac surgery and cardiopulmonary bypass. There have even been reports of CAGE from inhaling directly from a helium tank and from vaginal insufflation during pregnancy. The abrupt appearance of any new neurologic symptoms, including seizures, during any of the above activities or procedures should prompt investigation for pulmonary overinflation injury and CAGE. The definitive treatment for CAGE is hyperbaric oxygen therapy (HBO). The patient should be placed on 100% oxygen until arrangements can be made with a hyperbaric consulting service.

Pseudoseizures

Pseudoseizures can be difficult to differentiate from true seizures. Pseudoseizures often occur in the setting of emotional distress. They are characterized by palpitations, choking sensations, dizziness, malaise, sensory disturbances, crying and alterations in consciousness with or without motor manifestations. Unlike true seizures, motor activity generally consists of side-to-side head movements, opisthotonus, pelvic thrusting, trembling and random asynchronous movements. Urinary incontinence and postictal somnolence may occur, although these are less likely. The postictal period following pseudoseizures is often brief. Pseudoseizures tend to be more gradual in evolution and longer in duration. Some patients are skilled at mimicking the tonic–clonic activity of a seizure. In a few cases, patients can be startled out of pseudoseizure activity or may respond to noxious stimuli such as ammonia capsules. Ultimately, pseudoseizures are diagnosed when normal EEG activity is documented during apparent seizure activity.

Disposition

Patients with SE, mental status examinations that do not return to normal, or seizures associated with serious underlying medical conditions should be admitted to an ICU or monitored setting and given first- and often second-line medications to suppress seizure activity. Most other seizure patients can be managed in the outpatient setting, with close follow-up and support from family or friends.

Patients with a first-time seizure in whom the work-up does not identify a serious underlying medical condition can be discharged with close follow-up. The question arises whether to institute anticonvulsant therapy after a first-time seizure. Factors that increase the risk of recurrent seizures include EEG abnormalities (though this diagnostic test is infrequently obtained in the ED), partial versus generalized seizures, recurrent seizures in the ED, history of intracranial surgery or head trauma, and persistent neurologic abnormality such as Todd's paralysis. These patients are more likely to be candidates for initiating seizure medications after a first episode, done in consultation with a neurologist or the patient's primary care provider. Many neurologists recommend levetiracetam as a first-line oral agent due to excellent bioavailability and favorable adverse event profile.

The side effects of traditional anticonvulsants are numerous; some patients cannot tolerate them, and many drug interactions may occur. In the absence of factors that are likely to predict recurrent seizures, and if provoking factors are easily managed (i.e., hypoglycemia), anticonvulsant therapy after a first-time seizure is not indicated. In patients with a known seizure disorder, anticonvulsant levels should be checked and managed appropriately in consultation with the physician responsible for those medications, if possible.

Many states have mandatory reporting laws for any episode of sudden lapse of consciousness, including seizures. Physicians should follow state laws regarding this, and advise patients not to drive or engage in activities that might be hazardous if a seizure were to recur. It is reasonable to document that this occurred.

Pearls, pitfalls and myths

- The most common cause of recurrent primary seizures is subtherapeutic anticonvulsant levels (often due to noncompliance); check appropriate anticonvulsant levels in a patient on these medications.
- Because the most common cause of secondary seizures is hypoglycemia, rapid glucose determination is important for all seizure patients.
- Benzodiazepines are the first-line treatment for seizures or status epilepticus in the ED. They can be administered via multiple routes.
- Urgent neuroimaging is recommended for patients with first-time seizures, suspicion of head trauma or increased ICP, intracranial mass or history of cancer, persistently abnormal mental status, focal neurologic deficit, underlying HIV or other immunocompromising diseases, or in patients taking anticoagulant medications.
- For otherwise healthy, well-appearing children with a febrile seizure, current evidence-based guidelines suggest that an LP is necessary only if there is clinical suspicion of a CNS infection or if the child has a history of recent antibiotic use. Some authorities recommend LP for all children less than 12 months, however.
- It is not always easy to distinguish seizure activity from syncope or pseudoseizures. Seizure activity is suggested by retrograde amnesia, preceding aura, bowel or bladder incontinence, tongue biting, and a prolonged postictal state.
- Alcoholics are at higher risk of falls, electrolyte abnormalities and hypoglycemia. Patients with alcohol withdrawal and a seizure must be carefully evaluated for these secondary conditions before concluding that the seizure is simply the result of the withdrawal state.
- Patients who require intubation and neuromuscular blockade may still be seizing at the neuronal level. Further investigation, such as a bedside EEG, must be performed to determine whether seizure activity is ongoing.
- The appearance of new neurologic symptoms or seizure activity in a SCUBA diver, a patient who is intubated on positive-pressure ventilation, or following vascular procedures should prompt investigation for CAGE.
- For patients with prolonged stays in endemic areas, neurocysticercosis must be considered as an etiology of seizure.
- Follow mandatory reporting laws for all patients experiencing any episode of loss of consciousness, including a seizure.

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38 Shortness of breath in adults

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Scope of the problem

Dyspnea is the perception of uncomfortable breathing and is synonymous with the terms "shortness of breath" or "breathlessness." Other terms patients use to describe dyspnea include "labored," "heavy," or "difficult" breathing. Dyspneic patients often say they feel like they are being smothered, cannot catch their breath, or are unable to get enough air.

Dyspnea is a symptom associated with many disorders, from non-urgent to life-threatening. Like the perception of pain, dyspnea is subjective. Therefore, its severity does not always correlate with the severity of the underlying pathology.

The chief complaint of shortness of breath is encountered in about 3% of all emergency department (ED) patients. Approximately two-thirds of these patients have an underlying cardiac or pulmonary disorder. Specific data on the general prevalence of dyspnea are not available, although 6–27% of the adult population in the Framingham study reported experiencing dyspnea. In some patient populations (e.g., oncology patients), the prevalence of dyspnea is much higher.

Anatomic essentials

In general, breathing is a well-synchronized, unconscious, effortless process. Dyspnea results when ventilatory demand exceeds respiratory function. Although the exact mechanism responsible for dyspnea is unknown, abnormalities or alterations of gas exchange, pulmonary circulation, cardiovascular function, respiratory mechanics, or oxygen (O_2)-carrying capacity of blood may result in dyspnea.

Respirations are generally "automatic" and under the control of the respiratory centers in the central nervous system (CNS) (e.g., medulla and pons). Respirations are regulated by various afferent input from *mechanoreceptors* in the lungs, airways and respiratory muscles, as well as *chemoreceptors* in the blood (e.g., aortic and carotid bodies) and brain. These chemoreceptors sense changes in the blood pH, and partial pressures of O_2 (PO_2) and carbon dioxide (PCO_2), and then transmit signals back to the central respiratory center. Here, the rate of ventilation is adjusted to maintain blood gas and acid–base homeostasis. Feedback from mechanoreceptors regarding the mechanical status and function of the ventilatory pump and respiratory muscles leads to adjustment of the level and pattern of breathing. The efferent nerve pathway to the muscles of respiration starts in the brainstem, crosses over and then travels down the contralateral spinal cord to reach the spinal motor neurons.

Respirations are also subject to voluntary control through input from the cerebral cortex to the respiratory centers in the medulla and pons. Of all the vital signs, respiratory rate is the only one that can be influenced by voluntary control, although to a limited extent.

During "normal" respiration, the main work of breathing is performed by the diaphragm and intercostal muscles. When a patient experiences respiratory distress, contraction of the intercostal muscles becomes more forceful and visible to the observer. This condition is referred to as "intercostal retractions" (or simply "retractions"), and the patient is said to be "retracting." When there is increased work of breathing or respiratory distress, other "accessory" muscles may be recruited in an effort to maintain air movement within the lungs. This may manifest as retractions in the supraclavicular, subcostal and substernal areas. Similarly, "abdominal breathing" (referred to as "see-saw" respirations) occurs when the abdominal muscles are recruited. Marked movement of the abdomen and paradoxical chest wall motion may also occur.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 38.1).

History

If dyspnea is not due to an immediate life-threat, the history and physical examination can proceed in an orderly fashion. Immediate resuscitation takes priority if the shortness of breath is due to respiratory failure, shock, or hypoxia. The history is crucial to determining the etiology of dyspnea and differentiating life-threatening from benign causes.

How did the shortness of breath begin (sudden or gradual onset)?

The acute onset of dyspnea raises concern for disorders such as spontaneous pneumothorax, pulmonary embolism (PE), acute coronary syndrome (ACS), acute pulmonary edema, or anaphylaxis. Gradual onset of shortness of breath may represent pneumonia, congestive heart failure (CHF), reactive airway disease (RAD), or malignancy.

Table 38.1 Shortness of breath in adults red flags

History	Concerning diagnosis
Altered mental status (e.g., confusion, agitation, or restlessness)	Hypoxia (decreased pO_2)
Somnolence	Elevated pCO_2
Chest pain	ACS or PE
Sudden onset SOB	Pneumothorax, PE, ACS, CHF or anaphylaxis
Syncope	ACS, dysrhythmia, valvular heart disease, cardiomyopathy or PE
Orthopnea, PND	CHF
Age >50 years	Cardiac or pulmonary disease
Worse with exertion or unable to do prior activities due to SOB	Cardiac or pulmonary disease
Hemoptysis	Malignancy, tuberculosis or serious infection
History of malignancy	Pneumonia or other significant infection
History of CHF, COPD, DVT or PE	CHF, COPD or PE
Fever, chills or night sweats	Tuberculosis, pneumonia or malignancy
IVDA, alcoholism or other drug abuse	Pneumonia
HIV+	Pneumonia, PCP, tuberculosis or malignancy
Examination finding	**Concerning diagnosis**
Fever	Malignancy or pulmonary infection
Low oxygen saturation	Hypoxemia from cardiac or pulmonary disease
Drooling, stridor or “tripod” position	Airway obstruction
Accessory muscle use, retractions or fatigue	Respiratory failure
Central cyanosis	Hypoxemia, VQ mismatch, right-to-left shunt or hemoglobin abnormality
Inability to speak in full sentences	Severe asthma, COPD or CHF
Jugular venous distention	CHF or pericardial tamponade
Rapid respirations	Significant cardiac or pulmonary disease
Distant heart sounds	Pericardial effusion or tamponade
Poor air movement	Severe asthma or COPD
Peripheral edema	CHF
Unilateral peripheral edema	DVT resulting in pulmonary embolism
Clubbing	Chronic pulmonary or heart disease
Diaphoresis	Autonomic stimulation/sympathetic overdrive (body’s reaction to hypoxemia or infection, poor cardiac output)

ACS: acute coronary syndrome; CHF: congestive heart failure; COPD: chronic obstructive pulmonary disease; DVT: deep venous thrombosis; HIV: human immunodeficiency virus; IVDA: intravenous drug abuse; PCP: *Pneumocystis carinii* (jiroveci) pneumonia; PE: pulmonary embolism; PND: paroxysmal nocturnal dyspnea; SOB: shortness of breath; VQ: ventilation-perfusion.

However, variation commonly occurs with these classic presentations.

Have you had shortness of breath before?

Previous episodes of dyspnea suggest a chronic cardiac or pulmonary disorder such as asthma, chronic obstructive pulmonary disease (COPD), or CHF. Asthma is the most likely diagnosis in a child or non-smoking adult who experiences chronic symptoms of shortness of breath that wax and wane, especially if wheezing is present. COPD should be considered in an adult smoker with recurrent episodes of dyspnea. In an adult with dyspnea and bilateral lower extremity edema, CHF is a possible etiology.

A patient with chronic shortness of breath (for months or years) may present with an acute exacerbation of dyspnea. This is often the case with COPD or CHF. The physician needs to determine what caused the sudden worsening of symptoms, such as medication non-compliance, pulmonary infection, change in diet (e.g., increased salt intake in a CHF patient), or cardiac ischemia or myocardial infarction (MI).

Does anything make the shortness of breath better or worse?

Orthopnea is dyspnea that is worse with lying down and better with sitting or standing. Typically, CHF and pulmonary edema are associated with orthopnea. Orthopnea is also one of the earliest findings in patients with diaphragmatic weakness from neuromuscular disorders. *Paroxysmal nocturnal dyspnea* (PND) refers to the patient waking up suddenly at night with acute shortness of breath. In patients with CHF, this is due to the accumulation of fluid in the lungs while the patient is supine during sleep. As a result of PND or orthopnea, patients may sleep sitting in a chair or with multiple pillows under their head in order to avoid becoming dyspneic. *Trepopnea* is dyspnea associated with a unilateral recumbent position, and may occur in patients with unilateral lung disease, ball-valve airway obstruction, diaphragmatic paralysis, or COPD.

Do you have chest pain?

Chest pain associated with dyspnea suggests a serious underlying disorder, such as ACS or PE. Sharp chest pain worsened by breathing but not by movement suggests a PE, spontaneous pneumothorax or pleurisy.

Do you have palpitations or feel an abnormal heartbeat?

Palpitations or an irregular heartbeat felt by the patient generally signifies a cardiac etiology for the dyspnea, particularly if new. When a dyspneic patient complains “my heart was beating fast (or slow),” “skipped beats,” or “was beating funny,” it suggests a dysrhythmia.

Do you have swelling of the lower extremities?

Is it difficult for the patient to put on their shoes because of edematous feet? Bilateral lower extremity edema may occur with right-sided CHF, whereas unilateral lower extremity swelling with calf pain is suspicious for a deep vein thrombosis (DVT). Dyspnea in a patient with a known DVT or unilateral leg swelling is worrisome for PE.

Do you feel faint or did you pass out?

A dyspneic patient who passed out (syncope) or almost passed out (near-syncope) may have significant cardiac (ACS, MI, dysrhythmias, valvular disease, or cardiomyopathy) or pulmonary disease, such as PE. However, syncope/near-syncope may result from other causes, such as gastrointestinal (GI) hemorrhage or volume depletion from dehydration (due to excessive fluid loss or inadequate oral fluid intake).

Do you have any upper respiratory symptoms?

Patients with pulmonary infections (pneumonia or acute bronchitis) resulting in shortness of breath may also have cough, purulent sputum, fever and/or chills, and possibly prodromal upper respiratory tract symptoms. Acute upper respiratory infections (URIs) or “cold” symptoms include rhinorrhea and generalized myalgias. Dyspnea with symptoms such as drooling, hoarseness, aphonia and “muffled voice” suggest upper airway disorders, specifically airway obstruction (such as epiglottitis or foreign body).

Are you coughing up blood?

Hemoptysis may occur with pulmonary or upper airway tumors/malignancies, pulmonary infections, tuberculosis and vasculitis. Frothy blood-tinged sputum is a classic sign of acute pulmonary edema.

Do you have tarry or bloody stools?

Patients with severe anemia (whether from GI bleeding or other causes) may experience dyspnea because of decreased O_2 delivery to the tissues or secondary to limited O_2 carrying capacity from lack of red blood cells (RBCs).

Have you been vomiting?

Vomiting can lead to electrolyte abnormalities, resulting in dyspnea, or may be due to diabetic ketoacidosis, with dyspnea secondary to a compensatory respiratory alkalosis from metabolic abnormalities.

Have you had recent unintentional weight loss?

Weight loss could be due to malignancy, especially lung cancer in a smoker.

Past medical

A history of previous PE, DVT, or clotting disorder in a dyspneic patient suggests PE. Recent surgery (especially abdominal or pelvic surgery), atrial fibrillation, pregnancy, malignancy, and prolonged immobility are predisposing factors (Table 38.2).

Table 38.2 Predisposing conditions for pulmonary embolism

Birth control pills and/or estrogens
Cardiac disease: atrial fibrillation, congestive heart failure
Clotting disorders
Family history
Immobility
Malignancy
Pregnancy
Previous pulmonary emboli or deep vein thrombosis
Recent surgery, especially abdominal or pelvic

A previous history of neurologic or muscular disorders may provide a clue to the etiology of dyspnea, as patients with such conditions may experience respiratory muscle weakness and develop respiratory failure. CNS disorders that affect the respiratory center in the medulla could lead to respiratory failure from loss of the central respiratory drive. Traumatic injury or disease that affects cervical nerve roots 3, 4 and 5 (C3, C4, C5) or either phrenic nerve can cause paralysis of the diaphragm.

Oncology patients, patients taking immunosuppressive agents, or patients with autoimmune disorders are especially prone to dyspnea. This may be due to infection or severe anemia secondary to bone marrow suppression.

Medicines can have dangerous side effects and may provide clues to underlying diseases. Female patients on birth control pills or estrogens are at risk for PE.

Social

Tobacco, alcohol and illicit drugs

A significant smoking history raises concern for COPD or lung cancer. An intoxicated patient with shortness of breath, cough and fever may have aspiration pneumonia. Patients using illegal drugs may develop a pneumonitis from the adulterants used to "cut" the drugs. Intravenous drug abusers can contract human immunodeficiency virus (HIV) infection from an infected needle, and may present with *Pneumocystis carinii* (jiroveci) pneumonia (PCP) as a manifestation of acquired immune deficiency syndrome (AIDS).

Review the patient's occupational history or any acute or chronic exposures to toxins

An acute hypersensitivity reaction or toxic lung injury may occur in workers who inhale various particulates. A dyspneic patient exposed to fire may have smoke inhalation or a hypersensitivity reaction from burning chemicals or toxins. A dyspneic patient with gradual onset of symptoms and exposure to toxins at work over years may have asbestosis, silicosis, berylliosis, or "coal worker's" pneumoconiosis.

Physical examination

Physical examination may be instrumental in diagnosing the etiology of dyspnea, as well as in determining which patients are critically ill and need immediate therapy (even resuscitation) (Table 38.3).

Table 38.3 Clinical signs of respiratory distress

Abnormal vital signs
– Heart rate: tachycardia or bradycardia
– Respiratory rate: tachypnea or bradypnea
– Abnormal respiratory pattern: periodic, Biot's, Cheyne–Stokes, apnea
Cyanosis
Pursed lips
Grunting
Nasal flaring
Intercostal retractions
Use of accessory muscles
Abdominal breathing
Paradoxical motion of chest

General appearance

The patient's general appearance is a critical part of the physical examination. Patients sitting upright and leaning forward on their hands (the "tripod" position) are urgently attempting to maintain an open airway and improve ventilation. Pursed lips, intercostal retractions, or accessory muscle use are other methods to facilitate air entry into the lungs. A patient who can speak in full sentences does *not* have significant respiratory distress; a patient who can speak only a few words has moderate respiratory distress. A patient who is too short of breath to even answer with a few words is experiencing severe respiratory distress. An anxious appearance or agitation suggests hypoxia; somnolence suggests hypercarbia.

Vital signs

Fever is nonspecific and may occur with respiratory infections or other etiologies, such as PE. Although a fever is typically found in patients with respiratory infections, they may have a normal temperature or hypothermia. Patients with PE rarely have temperatures >102°F.

Hypotension can occur secondary to dehydration, sepsis, shock, or hemorrhage. The combination of hypotension and dyspnea should raise concern for PE, cardiac disease and tension pneumothorax. Increased respiratory effort and rate, especially if prolonged, may contribute to dehydration due to increased insensible losses from the airway and lungs.

Tachycardia usually accompanies respiratory distress, although exceptions may occur in patients taking beta-blockers or calcium channel blockers. These medications can blunt the heart rate (HR) response to respiratory distress.

The respiratory rate (RR) can be a clue to an underlying respiratory problem. Typically, there is a compensatory increase in RR to improve oxygenation and ventilation. An abnormally low RR (*bradypnea*) is an ominous sign, usually signifying impending respiratory failure. Although a normal RR generally implies normal respiration, this finding can be tragically misleading and must be interpreted in the context of the patient's presentation. For example, an asthmatic in respiratory distress will initially increase his RR in order to improve his oxygenation and ventilation. However, this compensation can only be maintained for a finite period of time. When he begins to tire from the additional work of breathing, his RR starts to drop. It may fall into the normal range during this period of decompensation before the patient becomes bradypneic and has a respiratory arrest.

All patients complaining of shortness of breath should have pulse oximetry measured. Although pulse oximetry has a few technical limitations, a pulse oximetry reading <90% indicates significant hypoxemia and requires immediate evaluation.

Head, eyes, ears, nose and throat

Signs of respiratory distress include nasal flaring, pursed lips, grunting and perioral cyanosis. Any facial or neck abnormalities or asymmetry secondary to masses, tumors, infections or edema that might result in an obstructed airway should be identified. Drooling or inability to handle secretions is worrisome for upper airway obstruction. Stridor may accompany upper airway obstruction.

If signs of airway obstruction are present, allow the patient to remain sitting upright or in the position they find maximally comfortable, since this may keep the upper airway open. Do not force the patient to lie flat or tilt their head backwards. Do not insert a tongue blade into the mouth, since this may worsen airway obstruction and convert a partial airway obstruction into a complete obstruction. Most patients in respiratory distress prefer the upright position, as gravity can assist the diaphragm by pulling the abdominal contents down.

If the patient has no signs of airway obstruction and is stable, examine the oropharynx for masses, edema, infections and bleeding. The presence of oral thrush suggests immune compromise, such as HIV, and raises concern for opportunistic infections like pneumocystis pneumonia (PCP).

Neck

Examine the neck for asymmetry, masses, swelling and jugular venous distention. Listen for bruits over the carotid arteries consistent with vascular disease. Jugular venous distention on inspiration (*Kussmaul's sign*) may occur in pericardial tamponade, PE, or pneumothorax.

Pulmonary

Examination of the lungs is mandatory in the evaluation of any patient with dyspnea, as this often reveals the etiology of a patient's dyspnea.

Inspect the chest for symmetric rise, deformities, or paradoxical movement. Paradoxical chest movement refers to chest contraction during inspiration and the chest fluttering out during expiration; this is the opposite of normal chest wall motion. Intercostal, supraclavicular, subcostal and substernal retractions indicate respiratory distress.

Palpate the chest for any areas of tenderness, masses, or crepitus (subcutaneous air suggests a pneumothorax).

Auscultate all lung fields for wheezing, rales and rhonchi. Unilaterally decreased breath sounds suggest pneumothorax, atelectasis, pleural effusion, or pneumonia. Wheezing is usually due to bronchospasm, which most often occurs in pulmonary diseases such as asthma, COPD, bronchiolitis and acute bronchitis. Wheezing may also occur with CHF ("cardiac asthma"), foreign bodies and PE. Rales may be present due to underlying cardiac disease (pulmonary edema or CHF) or pulmonary disease (pneumonia). Rhonchi may be present with pneumonia or bronchitis.

Hyperresonance on percussion occurs with a pneumothorax; dullness on percussion suggests a pleural effusion, infiltrate, hemothorax or chylothorax.

Cardiac

Feel for the precordial impulse or thrills. Auscultate for abnormal heart sounds such as an S3 or S4, rubs, clicks, or murmurs. An S3 gallop or S4 may occur with CHF. A pericardial friction rub is sometimes heard with a pericardial effusion; a click suggests valvular disease. A murmur may be present with valvular disease or other cardiac disorders, or may be physiologic.

Palpate the pulses for strength, equality and regularity. A weak or thready pulse occurs in shock.

Abdomen

Inspect for abdominal distention, ascites, or pregnancy, which can limit movement of the diaphragm and interfere with respiration. Identify the "see-saw" respirations of abdominal breathing. Hepatosplenomegaly can occur with right-sided CHF. The presence of hepatojugular reflux (distention of the neck veins with firm palpation of the liver) indicates heart failure.

Neurologic

A generalized muscular disorder with peripheral muscle weakness may also have respiratory muscle weakness, leading to respiratory distress or failure.

Extremity

Inspect for cyanosis, edema, clubbing, cords, venous distention, changes of peripheral vascular disease, infection, or nicotine stains. Clubbing indicates longstanding hypoxia. The presence of unilateral leg swelling with dyspnea suggests PE.

Skin

Examine the skin for color (pallor, cyanosis), temperature (cold, cool, warm, or hot) and moisture (clammy or dry). The skin should also be examined to assess capillary refill, identify rashes (suggesting infection) or petechiae (indicating a hematologic disorder, vasculitis, or infection such as meningococcemia), or track marks from intravenous drug abuse (IVDA).

Differential diagnosis

Although the differential diagnosis of dyspnea is extensive (Table 38.4), the majority of dyspneic patients have a cardiopulmonary etiology. Most clinicians approach

Table 38.4 The etiology and differential diagnosis of dyspnea

- **I. Respiratory system**
  1. Airway obstruction
     - Foreign body
     - Mass: tumor/malignancy
     - Angioedema
     - Infections: epiglottitis, retropharyngeal abscess, parapharyngeal abscess, croup, bacterial tracheitis, bronchitis
     - Tracheomalacia, tracheal stenosis (congenital or acquired – often post-intubation)
  2. Lungs (pulmonary parenchyma)
     - Asthma
     - Chronic obstructive pulmonary disease
     - Infections: pneumonia, lung abscess
     - Trauma: pulmonary contusion, pulmonary hemorrhage
     - Pulmonary edema (non-cardiogenic)
     - Atelectasis
     - Bronchiectasis
     - Pulmonary fibrosis
     - Environmental/occupational lung disease: coal worker's pneumoconiosis, asbestosis, silicosis, berylliosis
     - Adult respiratory distress syndrome
     - Rheumatologic/autoimmune disorders: sarcoidosis, systemic lupus erythematosus
     - Hemorrhagic: Goodpasture's syndrome
     - Mass: tumor, malignancy (primary or metastatic)
  3. Pleura
     - Trauma: hemothorax, pneumothorax (tension, simple)
     - Atraumatic: spontaneous pneumothorax
     - Infections: empyema, pyothorax
     - Chylothorax
     - Pleural effusion
     - Pleural adhesions
     - Mass: pleural tumor, malignancy
  4. Chest wall
     - Trauma: flail chest, fractured ribs, other chest wall injury
     - Bony abnormalities: pectus excavatum, kyphoscoliosis
  5. Decreased lung volume due to interference with chest expansion
     - Abdominal distention
     - Abdominal mass
     - Diaphragm injury
     - Ruptured diaphragm
     - Paralysis of diaphragm
- **II. Cardiac**
  1. Myocardium
     - Coronary artery disease: ischemia, infarction
     - Myocarditis
     - Cardiomyopathy
     - Rheumatologic/immunologic disorders: systemic lupus erythematosus, sarcoidosis, any disease that infiltrates or destroys myocardium
     - Congestive heart failure
  2. Pericardium
     - Pericarditis
     - Pericardial tamponade
  3. Valvular
     - Aortic, mitral, tricuspid, pulmonic regurgitation or stenosis
  4. Cardiac shunts
     - Atrial septal defect
     - Ventricular septal defect
     - Patent ductus arteriosus
  5. Outflow obstruction
     - Left ventricular outflow tract obstruction: hypertrophic obstructive cardiomyopathy, critical aortic stenosis
     - Myxoma
  6. Congenital heart disease
     - Cyanotic congenital heart disease: tetralogy of Fallot, hypoplastic heart, Eisenmenger's syndrome
     - Shunts: atrial septal disease, ventricular septal disease, ostium primum, patent foramen ovale, patent ductus arteriosus
     - Coarctation of aorta
  7. Dysrhythmias
  8. Decreased cardiac output
     - Shock, myocarditis, dysrhythmias
  9. Heart failure
- **III. Vascular**
  1. Emboli
     - Pulmonary, air, fat, amniotic fluid
  2. Pulmonary hypertension

(*continued*)

Table 38.4 The etiology and differential diagnosis of dyspnea (*cont.*)

3. Veno-occlusive disease
4. Sickle cell disease
5. Vasculitis (rheumatologic/collagen, vascular diseases)
6. Arteriovenous fistula

IV. Neurologic/muscular

1. Neurologic
    - Central nervous system
        - Cerebrovascular accident, traumatic injuries, infections, multiple sclerosis, amyotrophic lateral sclerosis, botulism, organophosphate poisoning
    - Spinal cord
        - Trauma (e.g., spinal cord injury above C3 affects nerves C3, C4, C5 to the diaphragm, resulting in paralysis)
        - Spinal cord diseases: poliomyelitis, amyotrophic lateral sclerosis, spinal muscular atrophies
    - Peripheral nerves:
        - Peripheral neuropathies: Guillain–Barré syndrome, tetanus, tick paralysis
2. Muscle
    - Myopathies: myasthenia gravis, polymyositis, muscular dystrophy, some glycogen storage diseases, periodic paralysis (some forms)

V. Cardiac output

1. Shock
2. Low cardiac output

VI. Metabolic/renal

1. Diabetic ketoacidosis
2. Metabolic acidosis
3. Renal disease: renal tubular acidosis, renal failure

VII. Endocrine

- Thyroid disease: hyperthyroidism, hypothyroidism
- Cushing's disease

VIII. Hematologic

1. Anemia
2. Methemoglobinemia

IX. Gastrointestinal

1. Gastrointestinal reflux
2. Abdominal loading: ascites, obesity, pregnancy

X. Toxins/poisons/drugs

1. Carbon monoxide poisoning
2. Drugs: beta-blockers in patients with asthma or chronic obstructive pulmonary disease

XI. Psychologic

1. Hyperventilation syndrome
2. Anxiety
3. Panic disorder

XII. Deconditioning

Note: Some disorders may affect multiple areas of the nervous system and thus could be listed under several areas. For example, amyotrophic lateral sclerosis affects motor neurons in the spinal cord, brainstem and corticospinal tracts.

the differential diagnosis in terms of organ system, especially respiratory (both pulmonary and extrapulmonary), cardiac, and other causes. However, some physicians consider the etiology in terms of acute versus chronic processes. Table 38.5 lists the presentation, work-up and treatment for causes of dyspnea.

Diagnostic testing

Because the majority of patients with dyspnea have cardiopulmonary disease, the two most common diagnostic tests are the chest radiograph and electrocardiogram (ECG). These two tests are easy to perform, noninvasive, inexpensive, safe, and can quickly confirm or exclude many common diagnoses.

Radiologic studies

Chest X-ray

Because pulmonary disorders are the most common etiology of dyspnea (about half in one study of adults in a clinic setting), the chest X-ray may be the most useful diagnostic test in dyspneic patients of all ages. Posteroanterior (PA) and lateral chest films provide the most information. However, in very ill patients, a portable anteroposterior (AP) film can be performed at the bedside and interpreted immediately. Additionally, this prevents a potentially unstable patient from leaving the ED. The chest film can reveal abnormalities such as an infiltrate, may distinguish CHF (Figure 38.1) from COPD, and can exclude a clinically significant pneumothorax (Figure 38.2). A normal chest X-ray in the presence of unexplained hypoxemia is worrisome for PE. Patients with mild exacerbations of

Table 38.5 Symptoms, signs, evaluation and specific treatments for common and life-threatening causes of dyspnea

Diagnosis	Classic symptoms[a]	Classic signs[a]	Work-up[b]	Specific treatments
Airway obstruction	Drooling Stridor	Respiratory distress Sitting up/leaning forward to open airway ("tripod" position)	Secure airway first if critical or potentially unstable. Laryngoscopy, CT scan of neck, soft tissue X-rays of neck; ENT consultation	O_2; secure airway: intubate, cricothyrotomy, tracheostomy if potentially unstable. Treat underlying cause: aerosolized epinephrine for edema, remove foreign body
Asthma	Wheezing Shortness of breath Chest pain	Respiratory distress Prolonged I/E ratio Wheezing (if severe, there may not be wheezing – the "quiet" chest from no air flow)	Peak expiratory flow (often pre/post treatment to compare), CXR (hyperinflation ± infiltrate), ABG (possibly)	O_2, beta-agonists, corticosteroids, anti-cholinergics, antimicrobials if infection present. Consider magnesium, epinephrine and heliox. If severe, NIPPV or intubation
Congestive heart failure	Shortness of breath (chest pain if CAD also present)	Respiratory distress *L-sided CHF:* rales, JVD *R-sided CHF:* hepatomegaly, splenomegaly, lower extremity edema	CXR, ABG, BNP, cardiac enzymes if precipitated by CAD	O_2, diuretics, nitroglycerin, ACE inhibitors. If severe, NIPPV or intubation
COPD exacerbation	Wheezing Shortness of breath Chest pain	Respiratory distress Decreased breath sounds Wheezing Prolonged I/E ratio Barrel chest Clubbing	Peak expiratory flow (often pre/post treatment to compare), CXR: r/o pneumonia or PTX as exacerbating factors	O_2, (monitor for ↑ PCO_2 but do not withhold O_2 if needed; start low FiO_2 if possible). Beta-agonists, anticholinergics, corticosteroids. If severe, NIPPV or intubation
Pneumonia	Cough Shortness of breath Chest pain Sputum Hemoptysis	Respiratory distress Rales Egophony Fever	ABG (possibly) CBC: ↑ WBC, ± left shift. CXR: infiltrate (may not be visible if dehydrated or early)	O_2, antimicrobials if bacterial etiology suspected. If severe, NIPPV or intubation
Pneumothorax	Abrupt onset Shortness of breath Pleuritic chest pain	Decreased breath sounds on affected side Hyperresonance to percussion Subcutaneous emphysema	CXR	100% O_2, Heimlich valve, Thoravent, tube thoracostomy, needle thoracostomy if tension
Pulmonary embolism	Abrupt onset Shortness of breath Chest pain (usually sharp, pleuritic) Syncope Cough	Respiratory distress (if large PE); ↑ RR, ↑ HR, loud P_2 (if small, VS may be normal) Leg swelling (if DVT, clots may be from pelvis or lower extremity) Low-grade fever	ABG, D-dimer, V/Q scan, helical CT scan, pulmonary angiogram, lower extremity ultrasound	O_2, anticoagulation

ACE: angiotensin converting enzyme; ABG: arterial blood gas; BNP: B-type natriuretic peptide; CAD: coronary artery disease; CHF: congestive heart failure; COPD: chronic obstructive pulmonary disease; CT: computed tomography; CXR: chest X-ray; DVT: deep vein thrombosis; ENT: ear, nose and throat; HR: heart rate; I/E: inspiratory/expiratory; JVD: jugular venous distention; NIPPV: noninvasive positive pressure ventilation; PE: pulmonary embolism; PTX: pneumothorax; V/Q: ventilation/perfusion; VS: vital signs; WBC: white blood cell.

[a] Classic symptoms and signs may or may not be present in any given patient.

[b] Work-up varies depending on patient, test availability and conditions.

asthma or COPD who respond to emergent therapy do not need routine chest X-rays.

Electrocardiogram

An ECG is warranted in any dyspneic patient with known, suspected, or possible coronary artery disease (CAD), and in patients with both dyspnea and chest pain. Dyspnea may represent an "anginal equivalent," especially in elderly patients or patients with diabetes. "Silent" MIs account for approximately 20% of all MIs. The ECG may also detect cardiopulmonary diseases other than ACS that present with dyspnea, including CHF, pericarditis, cardiomyopathy and dysrhythmias. The ECG is rarely helpful in the diagnosis of PE. The "classic" finding of an S1Q3T3 is neither sensitive nor specific; sinus tachycardia and right ventricular strain are more common with PE.

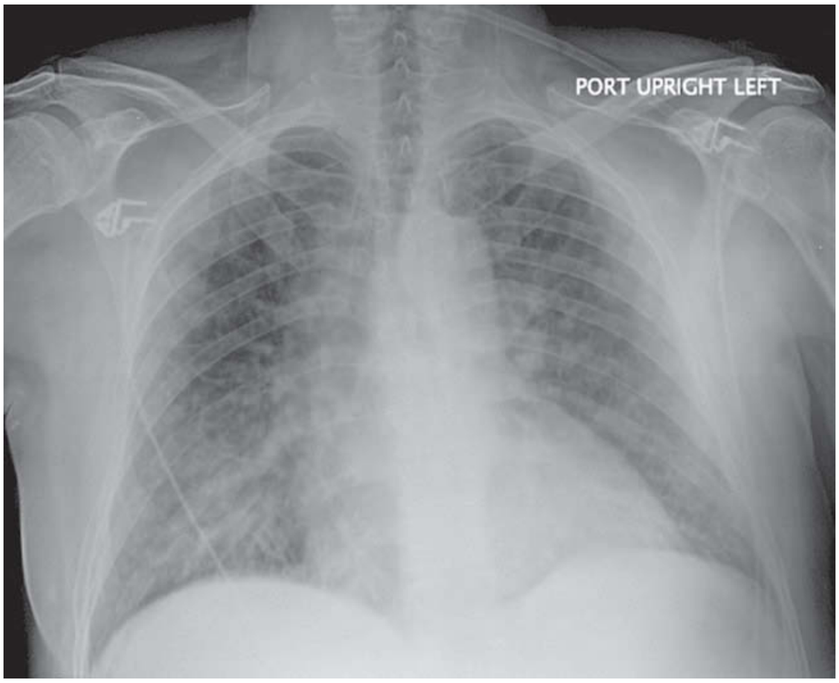

Figure 38.1
Congestive heart failure. AP chest radiograph demonstrating cardiomegaly, peribronchial cuffing and early airspace disease consistent with pulmonary edema. Courtesy: Kathryn Stevens, MD.

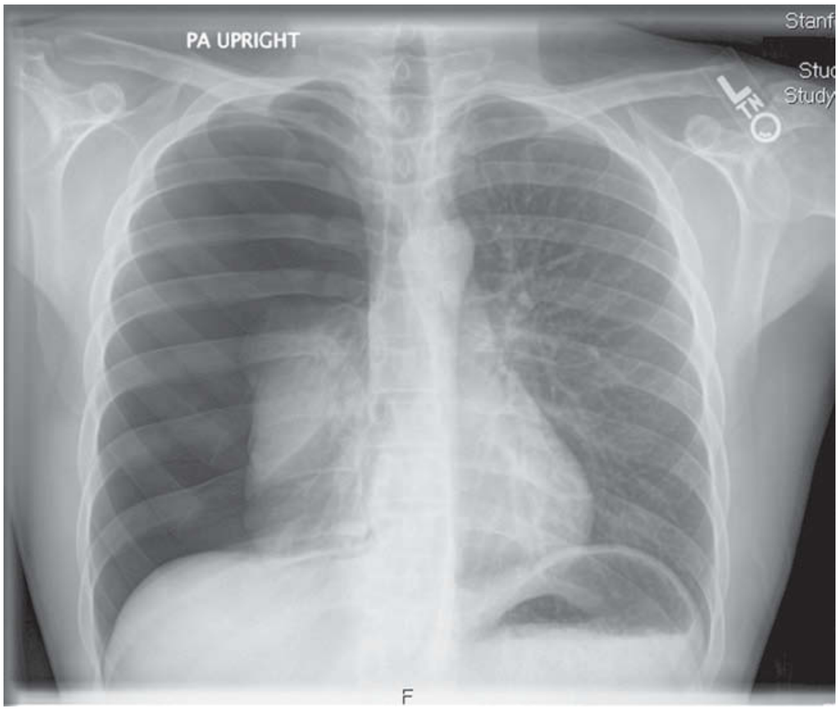

Figure 38.2
Pneumothorax. PA chest radiograph demonstrating a large spontaneous pneumothorax on the right, with nearly complete collapse of the underlying lung. Courtesy: Kathryn Stevens, MD.

Laboratory studies

Arterial blood gas

Arterial blood gases (ABGs) are not routinely necessary in all dyspneic patients. They may be useful in patients with unexplained dyspnea, altered mental status, suspected acidosis, or serious illness. An ABG can detect hypoxemia (PO_2 <90–95), hypercarbia (PCO_2 >45), metabolic acidosis (bicarb <20), acidosis (pH <7.35), or alkalosis (pH >7.45). Renal disease or metabolic disorders can lead to acidosis that may result in compensatory hyperventilation and dyspnea.

The ABG may be used to calculate the *alveolar-arterial gradient* (A-a gradient). This is the calculated difference between the concentrations of alveolar and arterial O_2.

> The alveolar oxygen concentration, PAO_2, is calculated from the alveolar gas equation:
>
> $$PAO_2 = FIO_2\ (PB - PH_2O) - PaCO_2/RQ$$
>
> FIO_2 = inspired oxygen fraction; PB = barometric pressure; PH_2O = water vapor pressure (47 at sea level); RQ = respiratory quotient, which is the rate of CO_2 production/rate of oxygen consumption (a constant 0.8).

The A-a gradient is merely the difference between the alveolar and arterial blood PO_2, or $PAO_2 - PaO_2$. Normally, this difference is 2–10 mmHg. This value increases with age, and may be as high as 30 mmHg in elderly patients. Abnormal A–a gradients may be found in conditions such as PE, although this finding is neither sensitive nor specific.

D-dimer

The ᴅ-dimer is a product of blood clot breakdown and is typically elevated in patients with thromboembolic disease. The ᴅ-dimer is nonspecific and may be elevated in conditions other than PE, such as pneumonia, chronic inflammatory states, and neoplastic conditions. In patients with low clinical risk for PE, a negative ᴅ-dimer assay makes the diagnosis of PE unlikely.

Cardiac enzymes

Cardiac enzymes (troponin, CK–MB, myoglobin) should be ordered if ACS is a possible cause of dyspnea. Dyspnea is a common presentation of ACS in the elderly and diabetics. Furthermore, prolonged dyspnea, hypoxia and increased work of breathing may result in cardiac ischemia.

B-type natriuretic peptide

B-type natriuretic peptide (BNP) levels may help diagnose CHF in patients with undifferentiated dyspnea. BNP is a good screening tool because of its high sensitivity and high negative predictive value. BNP levels are generally increased in patients presenting with CHF, whereas a level <100 pg/mL is very accurate for excluding CHF in patients with dyspnea. BNP may be falsely elevated in some patients without CHF (such as pneumonia); therefore, BNP levels need to be interpreted in the context of the patient's clinical presentation.

White blood cell count

Although an elevated white blood cell (WBC) count and/or left shift can occur with infection, malignancy and inflammatory processes, patients with a pulmonary infection such as pneumonia may have a normal WBC and differential. However, a WBC count with differential can be helpful in certain situations. A markedly elevated WBC or significantly abnormal differential suggests an infectious process or malignancy, especially in association with an abnormal chest X-ray. An abnormal peripheral smear showing immature forms or blasts suggests malignancy. A markedly decreased WBC raises the possibility of immunosuppression and increased susceptibility to infection. A patient with an infiltrate on chest X-ray and a very low absolute neutrophil count (ANC) is at greater risk for opportunistic infections, which will influence the selection of antibiotics.

Hemoglobin or hematocrit

The hemoglobin (Hgb) or hematocrit (Hct) may also be helpful in the evaluation of dyspnea. A markedly low Hgb or Hct can cause dyspnea due to inadequate delivery of O_2 to the cells or tissue. Conversely, a markedly elevated Hgb or Hct can occur in with cyanotic congenital heart disease or polycythemia, conditions that may present with dyspnea.

Electrolytes

Serum electrolytes may be indicated in patients who have dyspnea or tachypnea to evaluate for metabolic acidosis, diabetic ketoacidosis, or significant dehydration.

Glucose

A Dextrostix, Accu-Chek, or point-of-care glucose test (confirmed by a serum glucose) is indicated when hyperglycemia or diabetic ketoacidosis is suspected as the cause of dyspnea or tachypnea.

Pregnancy test

In women of childbearing age, a pregnancy test may be indicated. Pregnant women have an increased risk of PE and pre-eclampsia, two life-threatening disorders that can present with dyspnea.

Other laboratories

In patients for whom anticoagulation is being considered (e.g., ACS or PE), a baseline prothrombin time (PT) and partial thromboplastin time (PTT) are indicated. These studies should also be ordered for patients on warfarin or heparin. Liver function tests may be done if ascites or hepatomegaly is present. In some patients with pulmonary infections, blood cultures and sputum Gram stain and culture may be warranted. In patients in whom pulmonary malignancy is a concern, sputum for cytology should be obtained.

Additional specialized studies

Chest computed tomography

Helical chest CT has emerged as the modality of choice for the diagnosis of PE (Figure 38.3). Chest CT has the advantage over V/Q scan in that it may identify pulmonary or thoracic abnormalities in addition to PE.

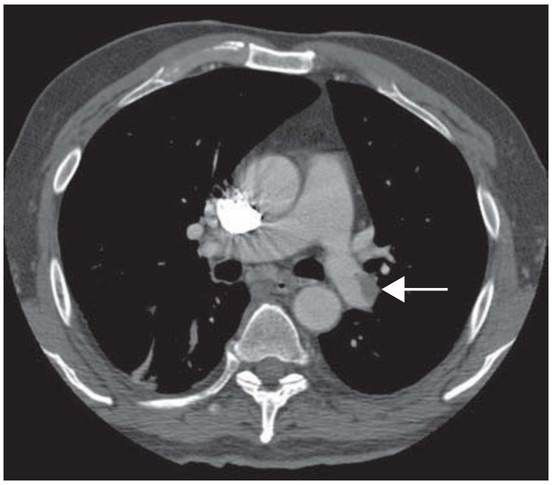

Figure 38.3
Axial CT section through the thorax following intravenous contrast, showing a large thrombus within the left main pulmonary artery. Courtesy: S.V. Mahadevan, MD.

Ventilation/perfusion scans

Ventilation/perfusion (V/Q) scans were once commonly ordered to diagnose PE, despite the test's limitations. In patients with an abnormal chest X-ray, there is a high incidence of indeterminate readings. Similarly, up to 40% of patients with a high pretest probability of PE but low probability V/Q scan have a PE. Recognizing these limitations, the V/Q scan is currently reserved for patients with contrast allergies, renal insufficiency (creatinine >1.5), or pregnancy (Figure 38.4).

Echocardiogram

The echocardiogram may diagnose a pericardial effusion or cardiac tamponade. In less critical situations, the echocardiogram may diagnose heart failure, valvular disease, congenital heart disease, hypertrophic obstructive cardiomyopathy (HOCM), cardiac tumors, or PE.

Peak expiratory flow rate

The peak expiratory flow rate (PEFR), also called the peak flow (PF), can monitor a patient's response to bronchodilator therapy. Measurements of PF are noninvasive, easy to obtain, and cost-effective. The results are very dependent on patient cooperation and effort. Trends in PEFR are more useful than absolute values in most cases.

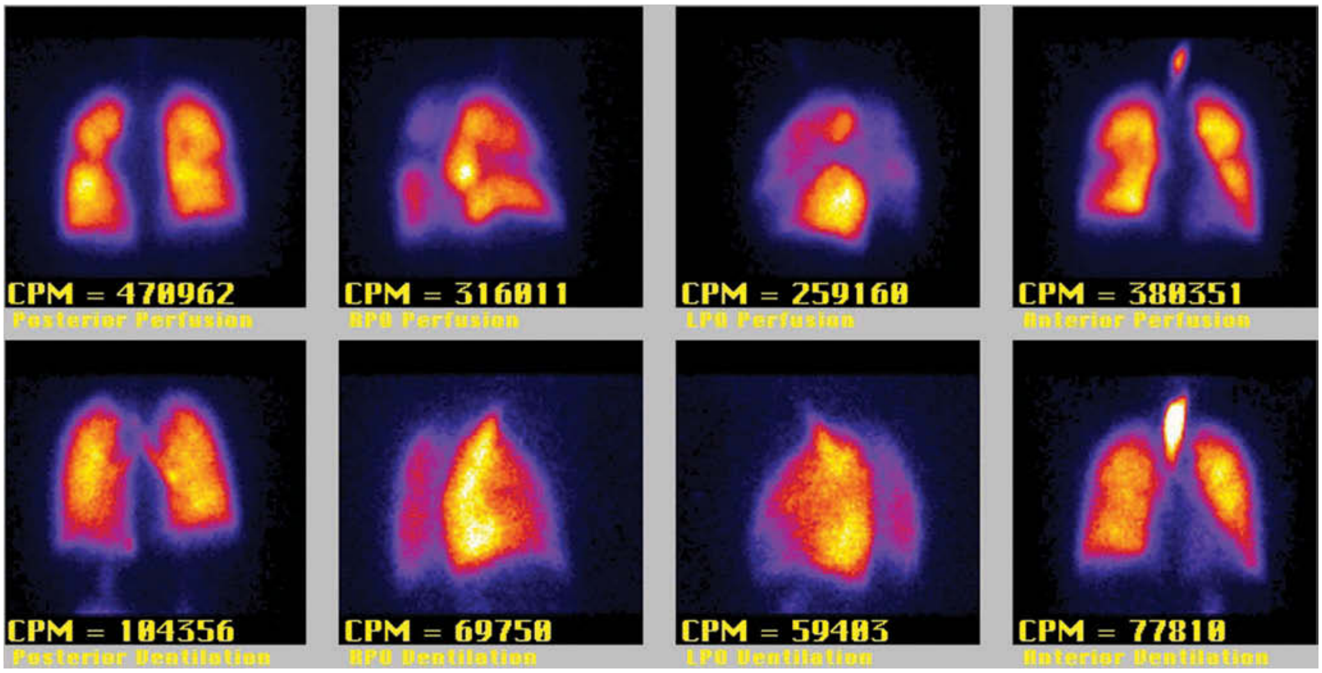

Figure 38.4
Lung ventilation/perfusion (V/Q) scan. The bottom row shows different views of the ventilation study. The top row shows the same views of the perfusion study. Heterogeneous perfusion is observed in both lung fields, with multiple peripheral wedge-shaped defects present with preserved ventilation (mismatched defects), consistent with high probability for pulmonary embolism. Courtesy: Carina Mari Aparici, MD and Sanjiv Gambhir MD, PhD.

General treatment principles

The patients's general appearance is key to determining which patients need immediate lifesaving measures (Table 38.6). Evaluation of the ABCs (airway, breathing, circulation) is the first priority. Does the patient have a patent airway? Is the patient in respiratory failure? Does the patient have respiratory distress and/or shock requiring emergent treatment?

Patients in the ED being evaluated for significant, life-threatening causes of dyspnea should be placed in a monitored bed with continuous HR, RR, blood pressure (BP) and ECG monitoring, in addition to continuous pulse oximetry. Supplemental O_2 should be administered to any patient suffering from hypoxemia, respiratory distress, respiratory failure, or shock, even if the patient has COPD. It is more important to provide O_2 to the tissues than to be concerned about suppressing the hypoxic respiratory drive in COPD patients. After the administration of supplemental O_2, allow the patient to sit upright, unless there are contraindications such as an acute cervical spine injury. The upright position makes it easier to breathe by decreasing the work of breathing. This position decreases "abdominal breathing," aids accessory muscle use, and decreases pulmonary congestion in patients with heart failure.

Noninvasive positive pressure ventilation (NIPPV), bilevel positive airway pressure (BiPAP), or continuous positive airway pressure (CPAP), may be instituted in select patients with respiratory distress. The physician should be prepared to intubate a patient who fails a brief trial of NIPPV.

If bradypnea or apnea occurs, ventilation may be assisted with a bag-mask device prior to definitive airway management. Emergent airway management may be indicated if airway obstruction, altered mental status, shock, inability to speak, or inadequate ventilation is present. Endotracheal intubation provides a definitive airway, with laryngeal mask airway (LMA), fiberoptics, cricothyrotomy, or transtracheal jet ventilation serving as alternatives in the event of unsuccessful intubation. Emergent airway management is discussed in detail in Chapter 2.

Table 38.6 Treatment for the dyspneic patient

Maintain the airway
Give O_2 (even if chronic obstructive pulmonary disease is present)
Continuous monitoring of pulse oximetry, HR, BP, ECG
Sit patient upright (decreases work of breathing, may decrease pulmonary congestion in CHF) if no acute cervical spine injury
If possible airway obstruction: do not lie patient flat, lean head backward, or use a tongue blade in mouth
May try NIPPV in some patients
Emergency airway management, if needed: endotracheal or nasotracheal intubation, laryngeal mask airway, transtracheal jet ventilation, cricothyrotomy (surgical)

BP: blood pressure; CHF: congestive heart failure; ECG: electrocardiogram; HR: heart rate; NIPPV: noninvasive positive pressure ventilation.

Acute asthma exacerbation

Pharmacologic agents used for the treatment of an acute asthmatic episode are known as "rescue medications." Inhaled beta-2-selective agonists are the initial therapeutic agents for all exacerbations. These agents cause bronchial smooth muscle relaxation and bronchodilation. The most commonly used beta-agonist is albuterol. Inhaled beta-agonists are generally preferred over other routes of administration (such as subcutaneous) because of fewer side effects and better efficacy. Anticholinergic agents, such as ipratropium, cause bronchodilation via inhibition of vagal tone from blocking muscarinic receptors in the airway smooth muscle. The beta-agonist albuterol and the anticholinergic ipratropium can be given in combination. Corticosteroids are useful because of their antiinflammatory effects; these include blocking the release of inflammatory mediators (which attract polymorphonuclear leukocytes), blocking increased capillary permeability, and increasing the "receptiveness" (and thus "availability") of beta-adrenergic receptors. High doses of corticosteroids for short durations are used for moderate to severe exacerbations, and in mild exacerbations in poorly controlled or steroid-dependent asthmatics. Inhaled corticosteroids are used for prophylaxis in most patients with asthma. Antibiotics are given for concurrent bacterial infections. With severe asthma or status asthmaticus, medications such as magnesium, epinephrine, and heliox may be administered in addition to those discussed.

Acute exacerbation of chronic obstructive pulmonary disease

The therapy for acute exacerbations of COPD is the same as for asthma: inhaled beta-adrenergic agents, anticholinergic bronchodilators, and corticosteroids. Antibiotics are commonly given to patients experiencing COPD exacerbations, as bacterial infections are more frequent.

Pneumonia

Specific therapy for pneumonia depends on the underlying etiologic agent. Bacterial infections are treated with antibiotics. The choice of antimicrobial therapy is influenced not only by the suspected causative organism, but also by age, comorbidities and clinical severity. Empiric antibiotic therapy is also based on whether the pneumonia is "community-acquired" or "hospital-acquired." Aspiration pneumonias are particularly difficult to treat, often occurring in debilitated patients, alcoholics, patients with changes in consciousness, and those who cannot protect their airway. Pneumonia in an HIV-positive patient differs from pneumonia in a geriatric nursing home patient with numerous comorbidities (e.g., diabetes, CHF, COPD, renal failure, or risk of aspiration). An atypical pneumonia in an otherwise healthy young adult has a relatively benign (typically outpatient) course. Supportive treatment is generally the rule for viral pneumonia, although neuraminidase inhibitors (zanamivir or oseltamivir) can be used for severe pneumonia due to influenza. Amantadine and rimantadine are not recommended in the United States due to increased resistance. Ribavirin (antiviral) and immunotherapy with respiratory syncytial virus immune globulin (RSVIG) may be warranted in immunocompromised patients with severe RSV pneumonia.

Congestive heart failure

Emergency treatment of CHF includes removal of the precipitating factor(s), identification and correction of the underlying etiology, and redistribution and removal of excess fluid in the lungs. Three helpful mnemonics summarizing the treatment of CHF are provided in Table 38.7 (these treatment modalities are not listed in particular order of use).

Table 38.7 Mnemonics for the treatment of CHF

UNLOAD ME
- U: upright position
- N: nitrates/nitroprusside
- L: lasix
- O: oxygen
- A: ACE inhibitors and aspirin
- D: dopamine/dobutamine, dialysis if appropriate
- M: morphine sulfate
- E: endotracheal intubation (if needed)

MOIST AND DAMP
- M: morphine sulfate
- O: oxygen
- I: inotropic support
- S: sitting upright, salt restriction
- T: tourniquets (rotating)
- A: aspirin
- N: nitrates/nitroprusside
- D: diuretics, dialysis if appropriate
- D: digoxin
- A: ACE inhibitors
- M: monitors (cardiac, pulse oximetry, BP)
- P: positive pressure ventilation, phlebotomy

LMNOP
- L: lasix
- M: morphine
- N: nitrates/nitroprusside
- O: oxygen
- P: position (upright), positive pressure ventilation

ACE: angiotensin-converting enzyme; BP: blood pressure; CHF: congestive heart failure.

In the setting of acute pulmonary edema, 100% O_2 must be provided, and often delivered under positive pressure. The patient is maintained in the upright sitting posture, with legs dangling if possible to assist with fluid redistribution away from the lungs.

Many clinical manifestations of CHF result from hypervolemia and expansion of the interstitial fluid volume. Diuretics are commonly used to treat CHF and contribute to excess fluid removal and redistribution. In acute emergencies, loop diuretics (e.g., furosemide) are administered IV. This route results in rapid and reliable absorption, resulting in increased venous capacitance (venodilation), which decreases venous return, in addition to reducing circulating blood volume by its renal diuretic effect. The usual starting dose for furosemide is 20–80 mg IV, depending on prior exposure, chronic therapy, and renal function (larger doses are required in patients with renal insufficiency).

Nitroglycerin may be administered sublingually or IV to decrease pulmonary congestion and peripheral resistance through veno- and arteriodilation. This also causes afterload reduction and redistributes fluid from the lungs. High-dose nitroglycerin has proven more efficacious in treating acute exacerbations of CHF than high-dose diuretics, although a synergistic effect between high-dose nitroglycerin and low-dose diuretic is likely. In acute CHF emergencies, when systolic blood pressure (SBP) is high due to increased sympathetic tone and intense vasoconstriction, sodium nitroprusside may be administered IV. Patients receiving nitroprusside must have continuous monitoring, preferably with an arterial line.

Morphine sulfate decreases adrenergic vasoconstrictor stimuli to arteriolar venous beds. It also reduces anxiety. Caution is warranted, as it may cause CNS and respiratory depression.

Dopamine and dobutamine have a limited role in the acute management of CHF exacerbations. These inotropic agents may be necessary when patients present in acute respiratory distress accompanied by hypotension, and are not responding to other therapeutic agents. Dopamine, which increases renal blood flow in low doses according to some authorities, can increase peripheral tone if a patient is hypotensive. Dobutamine has been demonstrated to improve cardiac contractility and output, which may assist with fluid redistribution. However, dobutamine can cause tachycardia and dysrhythmias, and should be used with caution.

Angiotensin-converting enzyme (ACE) inhibitors cause peripheral vasodilation, decreasing systemic pressure that may lead to regression of left ventricular hypertrophy (LVH). The effect of ACE inhibitors may be partly due to a decrease in myocardial angiotensin II production and cardiac remodeling. The role of ACE inhibitors in the acute management of CHF remains controversial. Some emergency physicians advocate sublingual ACE inhibitor administration for acute presentations of CHF. Contraindications to ACE inhibitors include pregnancy, prior ACE inhibitor-induced angioedema and hyperkalemia.

Bronchodilators, such as beta-2-agonists, may be of limited benefit in patients presenting in acute pulmonary edema, as there is often mild to moderate bronchospasm secondary to fluid congestion. Caution is warranted, however, as these agents may increase HR, agitation or anxiety, thereby worsening dyspnea.

Digoxin has no definitive role in the acute management of CHF. Digitalis glycosides are commonly used in chronic therapy to increase myocardial contractility and improve ventricular emptying. Digoxin toxicity should be considered with acute or chronic digoxin therapy, especially in association with concomitant dehydration and renal insufficiency.

Beta-blockers slow HR (increasing the time available for coronary flow and filling of the left ventricle), decrease myocardial O_2 demand, and lower BP (causing a regression of LVH). The use of beta-blockers in acute

CHF is controversial; most authorities do not recommend their use in the ED unless diastolic dysfunction exists, or if tachycardia or excess catecholamines is causing the CHF exacerbation. When needed, short-acting beta-blockers (such as esmolol or metoprolol) are recommended. Although not part of emergency care, chronic beta-blocker therapy has been shown to improve LV function and long-term survival. Contraindications include bradycardia or known atrioventricular (AV) block, severe lung disease or bronchospasm, and previous adverse response to beta-blockers. They should be used with extreme caution in asthmatics and patients with COPD.

For chronic renal failure patients on dialysis, acute CHF exacerbations due to fluid overload with resultant respiratory distress are common. Emergent dialysis may be the only treatment that can successfully remove the fluid responsible for respiratory distress, although many of the therapies mentioned previously may provide some benefit.

It should be noted that not all of these drugs are used for every patient presenting in CHF. The choice of pharmacologic agents depends on many variables, including the clinical situation, vital signs, comorbid conditions (angina, hypertension, diabetes), and whether the heart failure is due to systolic or diastolic dysfunction.

In addition to pharmacologic therapy, lifestyle modifications are recommended for CHF patients. These include smoking cessation, decreasing alcohol intake, salt restriction, water restriction if hyponatremic, weight reduction, and cardiac rehabilitation if stable.

Pulmonary embolism

Therapy for PE is anticoagulation. The standard anticoagulant regimen is the simultaneous initiation of heparin (either unfractionated or low-molecular-weight) and oral warfarin in stable patients. Major side effects of heparin are bleeding and heparin-induced thrombocytopenia (HIT), often associated with thrombosis. IV thrombolytic therapy may be used for PE that causes hemodynamic compromise or respiratory distress. Thrombolytics accelerate clot lysis, but their use is associated with an increased risk of intracranial and GI bleeding. Contraindications to their use include patients at very high risk of bleeding or unstable patients needing immediate intervention, such as surgery or insertion of a vena cava filter.

Central airway obstruction

Management of central or upper airway obstruction entails ensuring both oxygenation and ventilation. Following this, treatment depends on the etiology. If a serious allergic or anaphylactic reaction is suspected, epinephrine (aerosolized, intramuscular or IV) is the treatment of choice. IV diphenhydramine, IV corticosteroids, and sometimes IV H_2-blockers are also helpful, although oral administration is possible if the patient can tolerate this route. If the cause is a bacterial infection (e.g., epiglottis, retropharyngeal abscess), IV antibiotics are warranted. With epiglottitis, management may vary depending on age and severity. Pediatric patients and critically ill adults need their airway emergently secured, which is generally done in the operating room (OR) by the most experienced personnel. In selected stable adult epiglottitis patients, admission to an intensive care unit (ICU) for IV antibiotics and close monitoring for airway obstruction is appropriate. Some patients with retropharyngeal abscess may need surgical drainage in the OR by a head and neck (ENT) surgeon. If the etiology is a foreign body, removal via laryngoscopy or bronchoscopy may be indicated. If a mass is responsible for the obstruction, then surgery, radiation therapy, or chemotherapy are possible treatment options. Rapid involvement of specialists is often mandatory and may be lifesaving in these circumstances.

Special patients

Elderly

Cardiopulmonary causes of dyspnea increase in incidence with advancing age. Typically, older patients have multiple comorbidities (e.g., COPD, CHF) limiting their cardiopulmonary reserve; any additional disorders such as pneumonia are poorly tolerated. Any physiologic stress on the heart or lungs may result in increased morbidity and mortality.

It is usually more difficult to identify the causes of dyspnea in the elderly for many reasons. Signs and symptoms may be nonspecific, mild, and even absent. Elderly patients may have an acute MI without chest pain. Their baseline ECG is often abnormal (with hypertrophy, previous MI, or bundle branch block), such that diagnosing an acute MI by ECG alone may be challenging. Laboratory studies may be abnormal at baseline. For example, in patients with renal failure, the troponin is often elevated even if an acute MI is not present. Mortality rates of patients with elevated troponin due to any cause are greater than the rates of matched patients with normal troponin levels.

Pregnant

Dyspnea is extremely common in pregnancy, occurring in three-fourths of pregnant women by 30 weeks gestation. This physiologic dyspnea of pregnancy occurs gradually, usually with exertion, and is not severe. The cause of dyspnea during pregnancy is due to the physiologic effects of an elevated diaphragm and postural-dependent alterations in blood flow. Also, hyperventilation from increased circulating progesterone and an increased sensitivity to CO_2 results in dyspnea. The physiologic dyspnea of pregnancy rarely increases in severity during the final weeks before delivery. However, dyspnea during pregnancy that is acute, severe, progressive, occurs at rest, or occurs with other symptoms or signs of cardiopulmonary disease is abnormal and deserves further evaluation. Pregnant women are at increased risk for two life-threatening causes of dyspnea – PE and pre-eclampsia.

Patients who are pregnant, postpartum, have had a recent miscarriage or abortion, or have had recent gynecologic or pelvic surgery are at increased risk for PE. Additionally, women who have a history of multiple episodes of fetal demise (e.g., multiple miscarriages) may have a coagulopathy that predisposes them to miscarriage and PE. For all pregnant patients who present with shortness of breath or chest pain, PE should be considered a diagnostic possibility. Unrealistic concerns over potential fetal harm from radiation exposure should not dissuade clinicians from doing appropriate radiologic studies when warranted. Limiting fetal radiation exposure can be accomplished by:

1. Use of a lead shield (apron) over the abdomen.
2. Eliminating the ventilation portion of the VQ scan in patients with normal perfusion and decreasing the dose of the perfusion component by 50%.
3. Using a low-dose CT pulmonary angiography protocol.

Disposition

Patients in respiratory distress and those requiring supplemental O_2 to maintain adequate oxygenation need hospital admission. Disposition generally depends on the underlying etiology and response to therapy. For example, a dyspneic patient with asthma, COPD, or CHF who responds to therapy in the ED, is no longer dyspneic (or returns to his or her baseline), and has an acceptable pulse oximetry reading, a normal respiratory rate, and no signs of respiratory distress, may be discharged home on appropriate therapy with close follow-up. If any of the above are abnormal (dyspnea remains, hypoxia, increased work of breathing, PF significantly below baseline), then admission is warranted. A useful test of pulmonary function is a trial of ambulation, with assessment of a patient's dyspnea and vital signs (especially pulse oximetry) after ambulation. A patient who can't walk without dyspnea but is comfortable at rest is not ready for discharge and may need hospital admission. Admission is also indicated in a patient with dyspnea secondary to ACS, such as unstable angina or acute MI, even if he or she responds to therapy in the ED and is no longer dyspneic.

Disposition may be affected by the clinical situation and comorbidities. In an otherwise healthy young adult without respiratory distress and normal vital signs, pneumonia is generally well tolerated and can be managed as an outpatient. A geriatric patient with COPD or CHF has limited cardiopulmonary reserve, so pneumonia in such a patient may require admission for initial therapy and observation. Similarly, an asthmatic child with a poor social situation, unreliable caregivers, or who may not receive his discharge medications should be considered for admission.

Pearls, pitfalls and myths

- Patients with apparently normal breathing (e.g., normal respiratory rate and respirations) may have a serious etiology for their dyspnea.
- All patients with dyspnea deserve an evaluation including a thorough history, physical examination and diagnostic tests. Specifically, pulse oximetry, a chest X-ray and an ECG (if cardiac disease is a concern) should be strongly considered.
- A normal pulse oximetry and ABG do not rule out significant disease, such as PE or ACS.
- Absence of chest pain does not rule out ACS or PE in patients with dyspnea.
- Dyspnea with hypotension or shock is an ominous combination. These patients need emergent evaluation and immediate treatment.
- Dyspnea and chest pain in adults is usually due to a cardiopulmonary etiology, and deserves further evaluation and careful disposition.
- Although dyspnea during pregnancy is common, pregnant women with acute or unexplained severe dyspnea deserve further evaluation, especially for PE.
- Necessary imaging studies should not be avoided in pregnant patients if they are indicated.
- Do not assume hysterical patients have psychogenic dyspnea; patients with life-threatening causes of dyspnea may be hysterical because of hypoxia.
- Do not assume that just because a patient has a history of a chronic cardiopulmonary disease that his acute episode of dyspnea is due to chronic disease. For instance, patients with CHF can develop a PE, patients with COPD can have acute pneumonia, and asthmatics can have a pneumothorax.
- Ambulation is a functional "test" that can provide tremendous information about a patient's respiratory status. Vital signs and the amount of dyspnea should be evaluated during and following ambulation, and compared with baseline, if possible.

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39 Shortness of breath in children

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Scope of the problem

Difficulty breathing is one of the most common reasons for children to visit the emergency department (ED). Upper and lower respiratory tract disease account for approximately 10% of pediatric ED visits and comprise 20% of pediatric hospital admissions. The necessary ED interventions can be as simple as parental reassurance for a mild cold to intubation for respiratory failure. Although most children recover completely from episodes of difficulty breathing, permanent pulmonary or brain injury may occur, especially in very young infants or chronically ill children.

Anatomic essentials

Infants and young children have relatively narrow airways with high resistance. If the diameter of these small airways is decreased, the work of breathing can increase dramatically. The airways can narrow due to inflammation (e.g., asthma, chemical pneumonitis, bacterial tracheitis, croup), bronchospasm (e.g., asthma, bronchiolitis), extrinsic compression (e.g., esophageal foreign body, retropharyngeal abscess), excessive mucus and secretions with airway plugging (e.g., bronchiolitis, bacterial tracheitis, pneumonia), or mechanical obstruction (e.g., aspirated foreign body). Infants have a pliable chest wall and immature diaphragm, which also contribute to respiratory fatigue and failure. Increased work of breathing may restrict a child's ability to feed, with resultant dehydration and respiratory muscle fatigue. This in turn may lead to respiratory failure and the subsequent need for mechanical ventilation.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 39.1).

History

Conditions that cause difficulty breathing in children have many similar features. Many are preceded by complaints such as rhinorrhea, low-grade fever, and other symptoms of upper respiratory tract infection. The history, however, can be used to determine the overall likelihood of some diagnoses.

How old is this child?

The likelihood of some disorders depends greatly on age. For example, pertussis is primarily seen in infants less than 6 months of age who are partially immunized. However, adults are now also presenting with this disease. Croup is a disease of young children, such as toddlers and preschool

Table 39.1 Shortness of breath in children red flags

History	Concerning diagnosis
Acute onset of cough and wheezing	Foreign body
Acute onset of fever and dysphagia	Epiglottitis, bacterial tracheitis, retropharyngeal abscess
History of prematurity, congestion, and difficulty breathing or apnea	Bronchiolitis
Paroxysmal cough, apnea	Pertussis, foreign body
Progressive sore throat and ear pain	Peritonsillar abscess
Chest pain and fever	Myocarditis, pericarditis
Examination finding	**Concerning diagnosis**
Wheezing	Bronchiolitis, asthma, foreign body, myocarditis
Stridor	Foreign body, croup, bacterial tracheitis, laryngomalacia
Rales	Pneumonia, bronchiolitis
Uvular displacement	Peritonsillar abscess
Drooling without fever	Foreign body
Sniffing or tripod position	Epiglottitis, bacterial tracheitis, severe croup, foreign body, severe asthma

groups. Bronchiolitis is generally not diagnosed past the age of 2 years. Infants who do not yet crawl, usually less than 9 months of age, seldom aspirate foreign bodies.

How long have these symptoms been going on?

The time course of the illness may be helpful. Abrupt-onset conditions include foreign body aspiration and chemical pneumonitis from hydrocarbon ingestion. Disorders such as bacterial tracheitis and epiglottitis may present after a few days of mild upper respiratory tract symptoms, and then have a rapid worsening. Croup tends to begin with mild coughing for a day or two before the parents notice a barking cough late in the evening. Bronchiolitis and asthma exacerbations typically worsen over a few days. Pertussis has three phases: catarrhal, paroxysmal and convalescent. The catarrhal stage is usually associated with a history of 7–10 days of rhinorrhea and upper respiratory tract symptoms. The second phase, the paroxysmal stage, is followed by 2–4 weeks of a relatively severe staccato cough often accompanied by post-tussive vomiting and periods of cyanosis. The third phase is the convalescent phase, which typically lasts 1–2 weeks, but in some cases may persist up to 6–10 weeks.

Have there been any fevers or ill contacts?

Fever or ill contacts tend to suggest an infectious etiology. Retropharyngeal and peritonsillar abscesses typically present with fever. Bronchiolitis usually presents with a fever, whereas an asthma exacerbation does not (unless the aggravating factor is an upper respiratory tract infection). Bacterial tracheitis and epiglottitis tend to have higher fevers than croup and upper respiratory tract infections. Recent antipyretics or the presence of an immune-compromising condition may affect the presenting temperature. Ill contacts suggest a highly infectious agent such as pertussis, respiratory syncytial virus (RSV), influenza, or influenza-like illness (e.g., H1N1).

Does the child have a cough? What does it sound like?

Some conditions cause children to have a distinctive cough. A barking, seal-like cough in a toddler is characteristic of croup, but can also be seen in children with bacterial tracheitis. A young infant that coughs in bursts or paroxysms followed by periods that are relatively free of coughing may have pertussis. A persistent, repetitive cough with an abrupt onset or unilateral wheezing may be due to an aspirated foreign body. A cough associated with bilateral wheezing suggests a bronchospastic process, such as asthma or bronchiolitis.

How is the child feeding/urinating?

It is important to assess the hydration status of a young child with difficulty breathing. Something as simple as nasal congestion in a young infant who is an obligate nasal breather can result in serious dehydration. Some children are simply breathing too fast to feed. The increased work of breathing, tachypnea and insensible fluid losses from the lungs may contribute to dehydration. Other findings such as sweating with feeds are suggestive of congestive heart failure. A history of feeding honey to a young infant may suggest botulism.

How is the child handling his or her secretions?

Although all young infants drool, excessive drooling may suggest epiglottitis, retropharyngeal abscess, or an esophageal foreign body. These conditions typically inhibit swallowing and lead to increased secretions.

Where was this child when you noticed a problem?

If the child was left unattended in a garage or under a sink, a chemical ingestion or foreign body aspiration should be suspected. It is important to recognize that toddlers can drown in a shallow bucket or tub and can present with respiratory distress due to immersion.

Past medical

There are several conditions that may alter the likelihood of certain diagnoses. If a child has a history of atopy, including eczema, environmental allergies or asthma, an exacerbation of asthma is more likely if the child is wheezing. Many children with a history of prematurity and intubation in the neonatal intensive care unit have bronchopulmonary dysplasia and are prone to lung infections that can become severe. Children with known cardiac defects may wheeze when in congestive heart failure, or have a cardiac cause for their difficulty breathing. Children with laryngo- or tracheomalacia may become more acutely ill with croup than other children. Clinicians should also inquire about chronic lung conditions such as cystic fibrosis.

Medications

One of the most common complaints in the ED is that “cough medicine” does not work. Many cough medicines given to children are expectorants that actually make children cough more than they would otherwise. The Food and Drug Administration has recently placed a “black box” warning on cough medications in children less than 2 years of age due to reports of seizures, altered mental status and death in this age group. Other medications, particularly for chronic conditions such as asthma, may give some indication of the child’s underlying health. A child with asthma who has used inhaled beta-agonists a few times in his or her life may respond to a respiratory infection better than a child with severe asthma who is frequently on oral steroids in addition to beta-agonists.

Immunizations

Immunization status is an important general marker of the overall health care of a child; however, the presence of a complete set of immunizations seldom has a significant impact on the identification or management of children with difficulty breathing. Due to herd immunity, infections with *Haemophilus influenzae* type b (Hib) are uncommon, even in unimmunized children. A child who has not received the pneumococcal vaccine may be more likely to have pneumonia due to an invasive strain of the organism.

Family

Some conditions, like asthma and cystic fibrosis, may be familial. Therefore, a family history may prove helpful. It is also important to ask about other ill contacts at home during the history.

Social

Occasionally a social history will be revealing. A history of homelessness may suggest tuberculosis. Day care or school may expose children to a greater number of viral respiratory illnesses. Smoke exposure in the home may exacerbate chronic conditions such as asthma. Recent travel overseas can expose the child to a multitude of different illnesses.

Physical examination

General appearance

An assessment of the degree to which a child is having difficulty breathing can be made upon entering the examination room. Infants should be removed from car seats or infant carriers, and all young children should be undressed. There are three main components to the initial general assessment: the work of breathing, the degree of dehydration, and the mental status. An experienced clinician can assess these three components in a matter of seconds.

Work of breathing

Assessing the work of breathing is perhaps the key to the initial evaluation of difficulty breathing in children. This assessment does not require a stethoscope and can be made upon entering the child's room. A happy, playful, well-appearing child generally has a normal work of breathing. Signs of increased work of breathing include retractions, grunting, nasal flaring, head bobbing, sitting forward to maintain an open airway, and tachypnea (respiratory rates may approach 100 breaths per minute in infants). When the child can no longer sustain the increased work of breathing, he or she will characteristically appear fatigued, develop altered mental status, and have periods of apnea, irregular respirations, or a markedly decreased respiratory rate.

Degree of dehydration

The degree of dehydration may indirectly indicate the overall impact of the child's increased work of breathing. A child who is working too hard or is too sick to feed requires more aggressive fluid resuscitation than a child who is feeding well. A decrease in the number of wet diapers per day may indicate dehydration. Other signs and symptoms of dehydration include a sunken fontanel or eyes, dry tongue and mouth, tachycardia, and either irritability or lethargy.

Mental status

A child's mental status can be rapidly assessed by watching her interact with a parent or caregiver, play with a toy, or interact with her environment (i.e., pull things off the wall, climb around the room). It can also be assessed during the patient evaluation. A bright, alert, interactive, smiling, playful child can be readily differentiated from one who is listless, limp, disinterested and tired-appearing.

Vital signs

There are five main vital signs that should be assessed in all children with difficulty breathing.

Respiratory rate

Although there are published tables of normal respiratory rates for children, these values are seldom helpful in the ED. The respiratory rate is quite variable and can depend on several factors that may change rapidly. Crying, fever, anxiety, hypoxia, increased work of breathing, shock, dehydration and pain can make a child breathe faster than normal. Counting the respiratory rate for only a few seconds and then using a multiple of that count to generate breaths per minute may inaccurately reflect the true respiratory rate. Rough guidelines for tachypnea suggest that a normal respiratory rate during the first 2 months of life is in the 30–60 breaths per minute (bpm) range. Respiratory rates > 60 bpm for a sustained period of time (while the infant is calm) are nearly always abnormal. Very rapid rates in the 80–100 bpm range may be seen. As infants age, the normal respiratory rate decreases. Older infants and toddlers typically have normal respiratory rates in the 20–40 bpm range. School-aged children usually have respiratory rates in the 20s. More ominous than tachypnea is bradypnea (slow respirations). Bradypnea typically signals respiratory fatigue that may require prompt intubation and mechanical ventilation. Infants in the few couple months of life should not be breathing slower than about 30 bpm. Young children should not breathe more slowly than 20 bpm. Adolescents have the same respiratory pattern as adults.

Heart rate

The heart rate is variable in the same way as the respiratory rate. Multiple transient factors may result in tachycardia. These include crying, fever, anxiety, increased work of breathing, shock, dehydration and pain. In young infants with difficulty breathing, a heart rate around 200 bpm is not uncommon. Supraventricular tachycardia is suggested by very rapid heart rates (at least 220 bpm in the infant and > 180 bpm in children). Persistent tachycardia (while the patient is calm and relaxed) most commonly represents dehydration, but may also be seen in early shock and myocarditis.

Pulse oximetry

Using a noninvasive sensor, a painless assessment of the child's oxygenation can be performed with a bedside pulse oximeter. This "fifth vital sign" should be routinely assessed in all children with respiratory complaints. Subtle alterations in oxygenation that are not typically picked up on physical examination may be appreciated with pulse oximetry. Pulse oximetry has several limitations, however. It cannot be used to exclude respiratory conditions such as pneumonia. Due to small digits and vasoconstriction in acutely ill infants, the pulse oximeter may have difficulty picking up a signal and generating useful output. The pulse oximeter frequently falsely alarms when there is excessive movement, the device fails to sense the pulse, or the probe falls off. Typically, young children have pulse oximetry readings in the high 90s or 100% at sea level, and in the low 90s at significant elevation. Many physicians will select a value in the low 90s as their threshold for administering oxygen.

Temperature

A good deal of fever phobia exists among parents and health care providers. In the setting of difficulty breathing, a fever typically suggests an infectious etiology. Foreign body aspiration or ingestion, for example, does not usually present with a fever.

Blood pressure

Although blood pressure measurements are commonly obtained on patients in the ED, the role of blood pressure measurement in young infants is controversial. Young children often have transiently elevated blood pressures in the ED that have no clinical significance. Low blood pressures are seen in critically ill patients. Young children do not exhibit low blood pressure even when they are in early shock, due to their excellent capacity to peripherally vasoconstrict. The preferred formula to use for normal systolic blood pressure in children is 90 + (2 × age in years), which correlates to the 50th percentile of measurements. Be careful using the formula 70 + (2 × age in years) for blood pressure, as it correlates to only the fifth percentile for systolic blood pressure in children.

Head, eyes, ears, nose and throat

Head

In young infants, a sunken fontanel may suggest dehydration.

Eyes

The concurrent presence of conjunctivitis in the setting of clinical pneumonia is suggestive of an infection with *Mycoplasma pneumoniae*, and may indicate the need for a macrolide antibiotic (e.g., erythromycin). Newborns with *Chlamydia* pneumonia may have a history of chlamydial eye infection. These patients typically present without a fever and have a staccato-like cough. Patients with viral respiratory infections can also present with conjunctivitis.

Ears

The inappropriate diagnosis of otitis media may lead to the unnecessary use of antibiotics in the setting of a viral upper respiratory tract infection. The most specific way to check for otitis media is with an insufflator to assess tympanic membrane mobility, as the ears may appear red due to fever or crying.

Nose

Copious rhinorrhea is often associated with upper respiratory tract infections. Rhinorrhea in the setting of wheezing is consistent with reactive airway disease exacerbated by a viral upper respiratory tract infection or bronchiolitis. Significant rhinorrhea is not typically seen in cases of peritonsillar or retropharyngeal abscess. Nasal flaring and grunting can be seen in young infants with respiratory distress.

Throat

The oropharyngeal examination may be particularly helpful in identifying cases of peritonsillar abscess. Dryness of the mucous membranes may indicate dehydration. A gentle examination of the posterior pharynx is indicated unless the child is in extremis. In the past, oropharyngeal examination was discouraged due to the concern for respiratory compromise if the child had epiglottitis. However, these concerns have not been validated, especially as the incidence of epiglottitis has been markedly decreased due to the Hib vaccine.

Neck

Asymmetric cervical adenopathy is typically seen with peritonsillar abscesses.

Pulmonary

The vast majority of information about the pulmonary examination is obtained without a stethoscope during the assessment of general appearance and work of breathing. The pulmonary examination should identify the presence or absence of retractions, asymmetry of breath sounds, a prolonged expiratory phase or wheezing suggesting bronchospasm, and the presence or absence of rales. Palpation and percussion have no appreciable role in the chest examination of an infant or young child.

The physical examination should include an assessment of stridor, wheezing, and abnormal or asymmetric breath sounds. Conditions that partially occlude the upper airway (epiglottitis, esophageal foreign body, croup and bacterial tracheitis) typically cause stridor. *Stridor* is an abnormal sound made during inspiration. Conditions that partially occlude the lower airways, such as asthma and bronchiolitis, typically cause wheezing and a prolonged expiratory phase. *Wheezing* is an abnormal sound made during expiration. Localized lung involvement as seen in pneumonia or aspirated foreign bodies may simply cause one side of the chest to sound different than the other. Rales can be heard in chemical pneumonitis, bronchiolitis, influenza, near-drowning, and less commonly in congestive heart failure. With poor air exchange, there may be few or no breath sounds (the "quiet" wheezer).

Cardiac

There are two main objectives in performing a cardiac examination on a child with difficulty breathing: identifying pathologic murmurs and identifying rate-related problems. Heart murmurs are quite common in young children; many have a known murmur that is of no clinical significance. Benign murmurs may also be heard in the setting of fever and shock. These "flow murmurs" are typically heard in systole, do not last for its entire duration, and are decrescendo in pattern. Pathologic murmurs may be acquired or newly discovered congenital problems. Subtle features (which may be difficult to assess in a loud ED on a tachycardic, ill child) include an S3 and absence of a split S1. Not-so-subtle features, such as a loud, blowing, holosystolic murmur, are unlikely to be missed. Patients with myocarditis may present with wheezing, so it is important to remember the caveat that "not all that wheezes is asthma."

Abdomen

A meaningful abdominal examination on a child who is having difficulty breathing can be difficult. Depending on the work of breathing, a young child with respiratory distress may preferentially sit up to breathe. It is imperative to keep the child in the position of comfort; laying the patient down to perform a thorough abdominal examination may worsen the patient's condition. Abdominal breathing is common in children with respiratory distress. It is important to feel for an enlarged liver, which may be associated with congestive heart failure. In children, gastric distension can also lead to respiratory compromise, which is especially important to remember in children who have received bag-mask ventilation.

Extremities

Capillary refill is quite dependent on ambient temperature. Abnormal capillary refill is usually defined as greater than 2 seconds duration. Although prolonged capillary refill may represent shock, peripheral vasoconstriction due to an infant being cold will also manifest as prolonged capillary refill. Cyanotic extremities may be seen in hypoxic children.

Neurologic

The neurologic examination performed on a child with difficulty breathing is typically brief and global. The physician should immediately note how the child interacts with his or her environment. A child who is lying down with obviously labored breathing and fatigue can be easily distinguished from a child who is playing, smiling, and, if old enough, sitting up and talking comfortably. As a child tires from a prolonged increase in the work of breathing, mental status typically deteriorates. The child will appear tired and less interactive, and may stare somewhat blankly forward, no longer seeming to recognize or care about the environment.

Altered mental status in a child with difficulty breathing can result from related concurrent conditions. For example, a child with pneumonia may develop sepsis. This may progress to septic shock, causing tachypnea initially, then respiratory depression and altered mental status. Children seem to tolerate modest degrees of hypoxia, but if acutely and severely hypoxic, mental status may be depressed. Fluid losses typically occur from the inability to feed, insensible losses with exhalation, and increased work of breathing. These fluid losses may lead to dehydration that can result in depressed mental status. This is especially true if dehydration progresses to hypovolemic shock.

Differential diagnosis

Table 39.2 describes causes of shortness of breath in children.

Diagnostic testing

Radiologic studies

Chest X-ray

Lobar or interstitial infiltrates suggest pneumonia. Unilateral hyperexpansion suggests an aspirated foreign body (Figure 39.1). Symmetric hyperexpansion can be seen in asthma and bronchiolitis. An esophageal foreign body may be discovered or confirmed (Figure 39.2). Serial chest X-rays may show progression of disease in conditions such as chemical pneumonitis and near-drowning. Special chest X-rays such as bilateral decubitus or inspiratory and expiratory films may identify unilateral hyperexpansion suggestive of an aspirated foreign body. Additional findings such as pneumothorax, pneumomediastinum and masses can be seen. Chest X-rays are typically unnecessary unless there is reasonable clinical suspicion of one of the above diagnoses.

Neck X-rays

Soft tissue neck X-rays may be helpful in identifying the cause of shortness of breath. They should be ordered selectively to identify prevertebral soft tissue swelling suggestive of a retropharyngeal abscess (Figure 39.3) or an upper esophageal foreign body (usually a coin). A narrowed tracheal shadow may be noted in cases of croup,

Table 39.2 Differential diagnosis of shortness of breath in children

Diagnosis	Clinical presentation	Work-up
Aspiration/chemical pneumonitis (e.g., hydrocarbon ingestion)	Coughing and gagging at the time of ingestion, followed by wheezing and tachypnea. Cyanosis may be present as well as hypoxia assessed by pulse oximetry. Mental status may be depressed. Fever may develop. Accidental and small-volume hydrocarbon ingestions predominate in young children. Adolescents may present after intentional, large-volume ingestions.	Prevent spontaneous emesis if possible and do not induce emesis. Avoid activated charcoal to further decrease the risk of vomiting and aspiration. Attempt to identify the exact compound ingested and contact a poison control center. A chest X-ray should be obtained looking for bronchovascular markings, bibasilar and perihilar infiltrates.
Asthma	Wheezing and respiratory distress are the hallmarks of an asthma exacerbation. Fever may be present if an infection is the trigger for the exacerbation. Sitting upright with a hyperexpanded chest is common. Severe cases may have an absence of wheezing due to poor air exchange. A known history of asthma, hay fever, and/or eczema (i.e., atopy) is typical. Most will have a prior history of wheezing or asthma.	Pulse oximetry may reveal hypoxia. Response to bronchodilators may be helpful. In cases in which there is poor air exchange, wheezing may become much more prominent *after* albuterol. Chest X-ray may be helpful if there is fever to assess for concurrent lobar pneumonia.
Bacterial tracheitis	In many ways, the presentation of bacterial (or membranous) tracheitis is like severe croup. The same age group as croup (toddlers and preschoolers) is typically affected. A prodrome of rhinorrhea and a barking cough is common. Bacterial tracheitis, however, presents with a high fever, ill appearance, and sometimes a productive cough. Children usually require prompt intubation.	Bacterial tracheitis is a clinical diagnosis; there is no specific laboratory or radiographic work-up. If obtained for other reasons, a lateral neck X-ray may demonstrate an exudate within the trachea or a narrowed tracheal shadow (steeple sign).
Bronchiolitis	The hallmark of bronchiolitis is wheezing. The typical age group is <24 months. The degree of respiratory distress ranges from minimal ("happy wheezer") to severe, with respiratory fatigue and failure requiring prompt intubation. Apnea may be the first sign seen in a small number of very young infants. Some infants seem to improve with beta-agonist therapy, whereas others do not.	Bronchiolitis is a clinical diagnosis, and is the most common diagnosis made in infants who are wheezing for the first time. A test for RSV is available as a nasal washing. This test is primarily used for hospitalized infants to identify those needing respiratory isolation from other children. About half of the cases of bronchiolitis are positive for RSV. A chest X-ray may be normal, or may reveal peribronchial cuffing or bilateral hyperexpanded lung fields.
Croup	Most commonly affects toddlers and preschool children. Prodrome of a few days of rhinorrhea and cough. Barky cough and stridor develop. In mild cases, the stridor is present only when the child is agitated or crying. More severe cases have stridor at rest. Low-grade fever is common. Marked improvement after exposure to cool night air (usually on the way to the ED) is common. Typically, these children have a nontoxic appearance.	In general, the diagnosis of croup is made on clinical grounds and no diagnostic work-up is indicated. Neck X-rays may be used to assess for esophageal or tracheal foreign body. Children with croup will usually have a narrowed tracheal shadow (steeple sign).
Epiglottitis	The number of cases of epiglottitis has declined due to the widespread use of the *Haemophilus influenzae* type b vaccine. Most commonly affected are preschool and young school-aged children. Abrupt onset of fever with dysphagia, drooling, refusal to speak, muffled voice, and sitting upright in the "sniffing" or tripod position are common features.	Minimal agitation of the patient. Lateral neck X-ray should demonstrate an enlarged epiglottitis. Be prepared for airway obstruction – be ready to intubate (likely to be very difficult) or use needle cricothyroidotomy jet ventilation. Emergent ENT consultation for direct visualization of the epiglottis in the operating room is indicated.
Foreign body aspiration (Figure 39.1)	The presentation of a child with a foreign body in the airway can be quite subtle or very dramatic. The most common symptoms are choking, coughing, a sense of breathlessness, and wheezing. Abrupt onset of symptoms while eating may be expected, as peanuts and other foods are commonly aspirated objects. About 80% of foreign body aspirations occur in toddlers and preschoolers.	The work-up depends on the severity of symptoms. Children with severe respiratory distress and impending airway obstruction are best managed in the operating room by a bronchoscopist. Aspirated foreign bodies are seen on chest X-ray less than 20% of the time. Inspiratory/expiratory, forced expiratory views, or bilateral decubitus X-rays may be needed to show air trapping and unilateral hyperexpansion, suggestive of aspirated foreign body.
Foreign body — esophagus (Figure 39.2)	Esophageal foreign bodies that are in the upper esophagus may compress the airway "from behind" and cause respiratory distress. Symptoms may include drooling and stridor. An abrupt onset of symptoms and the absence of fever are common.	Coins are the most commonly identified esophageal foreign bodies and are visible on X-ray.

(*continued*)

Table 39.2 Differential diagnosis of shortness of breath in children (*cont.*)

Diagnosis	Clinical presentation	Work-up
Muscle weakness (e.g., infant botulism, Guillain–Barré syndrome)	Muscle weakness in a previously well child is an uncommon event. Difficulty breathing arises when the respiratory muscles no longer can sustain the work of breathing. The onset is usually gradual and progressive. However, when the work of breathing can no longer be sustained, respiratory failure and the need for intubation arise suddenly.	There is no specific work-up for this in the ED. Ventilatory support is the primary concern and focus. Elevated PCO_2 on a blood gas may occur too late to be of clinical utility.
Myocarditis and congestive heart failure	Acquired congestive heart failure in children is a relatively rare event. A history of a recent viral infection is possible but nonspecific. Tachycardia that fails to improve or worsens with the administration of IV fluids is suggestive of congestive heart failure. Rales, pedal edema and jugular venous distention (commonly seen in adults) are atypical in young children. More typical symptoms include an enlarged liver and sweating during feedings. Fever may or may not be present.	An enlarged cardiac silhouette and cephalization on chest X-ray may be seen. Although uncommonly performed on children in the ED, echocardiography is helpful identifying cardiac dysfunction. The role of laboratory studies commonly used in adults with heart disease (e.g., troponin, B-type natriuretic peptide) is unclear in children.
Near-drowning	Presentation varies with patient severity. The child may be initially asymptomatic and develop shortness of breath and cough over a few hours. Alternatively, the child may present in a coma due to anoxic brain injury and be intubated at the scene.	In the awake, well-appearing child, a chest X-ray should be obtained to look for developing infiltrates. A second X-ray obtained a few hours later may show progressively worsening infiltrates. In the critically ill child, the initial chest X-ray may show florid pulmonary edema and interstitial infiltrates.
Peritonsillar abscess	Sore throat that progressively worsens in severity is common. This is typically seen in older children. Drooling is uncommon. A muffled voice may be present. This diagnosis is usually made by examining the posterior pharynx and seeing asymmetric swelling next to the tonsils and uvular deviation away from the swelling.	There is no specific work-up. Diagnosis is initially made by visual inspection of the posterior oropharynx. Diagnosis is confirmed by needle aspiration or during incision and drainage.
Pertussis	Presentation is primarily based on age. The classic presentation has been described in young children, consisting of mild rhinorrhea and cough followed by severe paroxysms of cough (with a characteristic "whoop") associated with vomiting. Immunizations have made this "classic" presentation of whooping cough rare. Young infants present with fever and repetitive paroxysms of cough, and may have associated seizures, pneumonia, or encephalopathy. However, infants may not have the characteristic "whoop." They may present with apnea, subconjunctival and intracranial hemorrhages.	The definitive test is culture of the etiologic agent, *Bordetella pertussis*, from nasopharyngeal mucus. This does not help with ED management as the test result takes about 2 weeks. The repetitive nature of a cough in a young infant suggests the diagnosis. The chest X-ray may identify associated pneumonia or an irregular heart border.
Pneumonia	A wide range of clinical presentations can be seen in children with pneumonia. An otherwise well-appearing child may present with a mild cough and fever. Alternatively, the child may present in septic shock with respiratory failure.	The hallmark of pneumonia is an infiltrate on chest X-ray. The combination of cough and fever with an infiltrate on chest X-ray is indicative of pneumonia.
Retropharyngeal abscess (Figure 39.3)	Children <3 years of age typically develop the abscess in the setting of suppurative cervical lymphadenopathy. An older child may fall with something in his mouth and develop an abscess following penetration of the posterior oropharynx.	Diagnosis can be difficult. A lateral neck X-ray may show prevertebral soft tissue swelling. Confirmation with a neck CT is usually necessary.
Upper respiratory tract infection/ nasal congestion	Although not usually a problem in toddlers and school-aged children, nasal congestion can lead to difficulty breathing in young infants since they are obligate nasal breathers.	The diagnosis is clinical and made by examination.

CT: computed tomography; ED: emergency department; ENT: ear, nose and throat; IV: intravenous; PCO_2: partial pressure of carbon dioxide; RSV: respiratory syncytial virus.

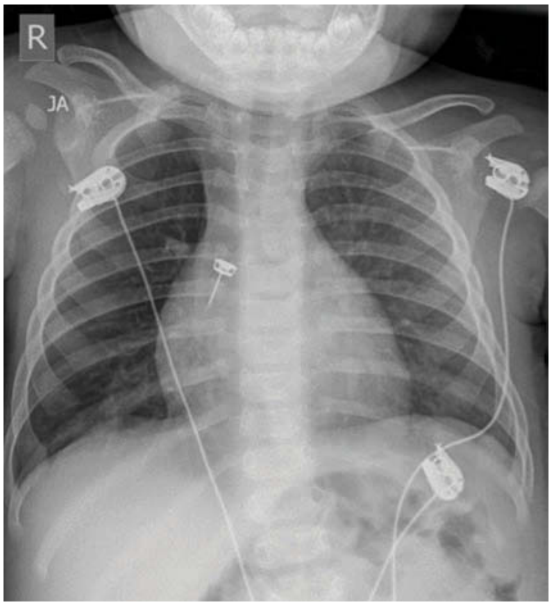

Figure 39.1
Right lung hyperexpansion. This child has aspirated a right-sided radiolucent foreign body (earring in the right mainstem bronchus). Courtesy: Bev Newman, MD.

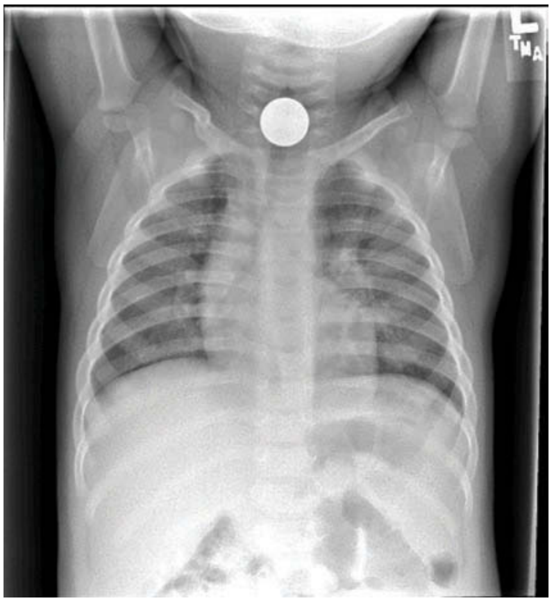

Figure 39.2
An esophageal coin. This child presented with the abrupt onset of difficulty breathing and drooling. Courtesy: Gus M. Garmel, MD.

but is not used to make the diagnosis. Exudate visible in the trachea on X-ray is suggestive of bacterial tracheitis.

Laboratory studies

Specific testing

Some tests may identify the etiologic agent, although the utility of these tests in the ED is questionable. There is a nasal wash test for RSV, the organism identified in about 70% of bronchiolitis cases. This test is performed on nasopharyngeal mucus, with results available within an hour in most hospitals. Because RSV is highly infectious, the primary purpose of this test is to detect infected children prior to hospitalization and place them in respiratory isolation. The use of this test for children treated as outpatients is controversial. There is also a nasopharyngeal swab test for pertussis. Currently, rapid tests are available for influenza.

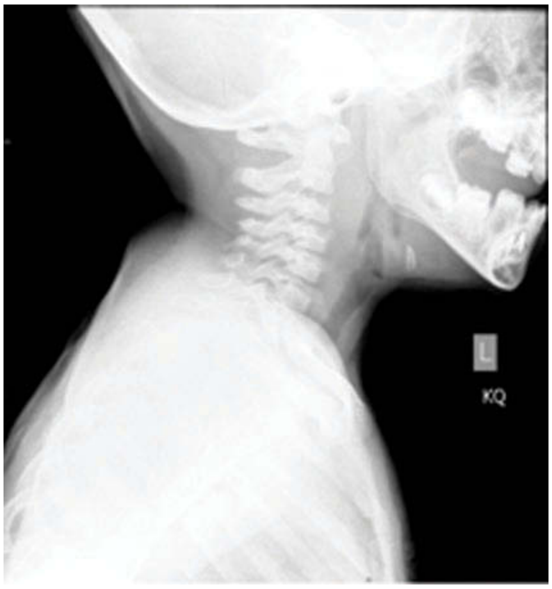

Figure 39.3
A child with a retropharyngeal abscess. Note the prevertebral soft tissue swelling. Courtesy: S.V. Mahadevan, MD.

Arterial blood gas

An arterial blood gas is rarely indicated for infants and children with difficulty breathing. If carbon monoxide poisoning is suspected, as in smoke inhalation, an arterial blood gas with co-oximetry can be used to determine the percent carboxyhemoglobin. It may also be used to guide management. If methemoglobinemia is suspected, as seen in teething gel ingestion, co-oximetry can be used to determine the percent methemoglobinemia, and may guide therapy with respect to administering methylene blue. Arterial blood gases may also be used after intubation to evaluate and appropriately adjust ventilator settings. In the vast majority of cases, pulse oximetry and the clinical examination are used to monitor the patient and guide therapy. The degree to which a child has respiratory fatigue determines when to intubate a child with respiratory distress, *not* the partial pressure of carbon dioxide ($PaCO_2$) from an arterial blood gas.

Peak flow

Peak flow is a bedside test in which a child is asked to blow as hard as they can with their lips closed tightly around an L-shaped tube. One portion of the tube has a sliding gauge that is measures the force of the child's exhalation.

The numeric output from these peak flow meters has been advocated for home assessment of chronic asthma. The role of peak flow meters in young children is controversial. Infants and young children who cannot forcefully exhale on command cannot use peak flow meters. The numeric output is effort-dependent, and school-aged children who are unfamiliar with the peak flow meter may not perform adequately enough to provide useful information. However, peak flow measurements in experienced school-aged asthmatics can guide acute ED management.

General treatment principles

There are several general treatment principles that apply to all children with difficulty breathing.

General treatment

- Oxygenation
- Respiratory support
- Rehydration
- Identification of patients who require procedural intervention

Oxygenation

Ensuring adequate oxygenation is an important principle in managing children with difficulty breathing. Oxygenation can be reliably assessed using pulse oximetry. Oxygen delivery can be achieved in a relatively non-threatening manner with a parent holding oxygen tubing near the patient's face ("blow by"). Alternatively, infant and child-sized oxygen delivery tubing and masks can be used.

Respiratory support

Although a child may have adequate oxygenation, this typically is not the same as adequate ventilation. The child may not be able to overcome excessive work of breathing and may fatigue. In these instances, although the pulse oximetry readings may be normal, the carbon dioxide level is typically rising. This rise in carbon dioxide may lead to mental status changes, decreased respiratory rate and, finally, bradycardia and apnea. If a child presents in respiratory failure or with apnea, immediate ventilatory assistance with a bag-mask device is indicated. If a child is more slowly deteriorating, a trial of noninvasive ventilatory support such as nasal continuous positive airway pressure (CPAP) or a bi-level positive airway pressure (BiPAP) mask may be tried. Ultimately, a child in respiratory failure will need to be endotracheally intubated and placed on mechanical ventilation. In anticipation of respiratory failure, the emergency physician should ready all appropriate personnel and equipment for intubation.

Rehydration

A child who has an increased work of breathing will have increased fluid losses from the lungs. These children typically have difficulty feeding and taking in adequate fluid volume. In general, children become dehydrated more rapidly than adults due to higher metabolic demands and decreased intake. IV access should be obtained early in the ED course unless discharging the child home is expected after the initial evaluation. If the child appears to be in shock, volume support with 20 mL/kg boluses of normal saline is indicated.

Identification of patients who may need procedural intervention

Identifying foreign bodies may be straightforward or subtle. Once identified, all aspirated foreign bodies and most high esophageal coins will need removal, usually by a specialist. Retropharyngeal abscesses are usually drained by ENT consultants in the operating room. Young infants are obligate nasal breathers and typically feed with a nipple in their mouth for several minutes without interruption. Nasal passage narrowing from nasal mucus or a viral upper respiratory tract infection may disrupt feeding and cause a surprising degree of distress in a neonate or young infant. Bulb suctioning the nose may help clear secretions, and may dramatically improve the ease of breathing and feeding for these babies.

Specific treatment

Asthma

Asthma has been increasing both in prevalence and severity for the past few decades. Asthma is the most common chronic childhood disease and accounts for up to 1.8 million annual ED visits There are several treatments for acute exacerbations of asthma that can be lifesaving. The two major pathophysiologic components of acute asthma exacerbations are bronchospasm and airway inflammation. The primary treatments are directed toward these features.

Bronchospasm is currently treated with albuterol, a relatively beta-2–specific bronchodilator. There are two main methods of delivery that seem to have similar efficacy. Albuterol may be administered as a nebulized solution or as an inhaler with a spacer (and mask in young infants). Terbutaline, a beta-2 agonist, can be administered subcutaneously or intravenously in children with severe asthma (10 mcg/kg bolus followed by a maintenance dose of 0.4 mcg/kg/min titrated to 6–10 mcg/kg/min). In a sense, children will drive the dosing through their pulmonary tidal volume.

Inflammation is primarily treated with steroids. Commonly administered steroids include prednisone, prednisolone, methylprednisolone and dexamethasone. The dosing is typically 1–2 mg/kg/day once daily (or divided into two equal doses) for 5 days for outpatients, except for dexamethasone, which is dosed from 0.15–0.6 mg/kg/dose for 1–2 days. Several studies have shown that single or two-dose dexamethasone is as effective as standard 5-day therapy with prednisolone or prednisone. The advantage of dexamethasone is that parents miss less days of work, children miss fewer days of school, compliance is increased, and there is less associated vomiting. Oral steroids are as effective as IV/IM steroids, and therefore parenteral medication should be reserved for those patients who are in extremis or who cannot tolerate oral medication.

Intravenous magnesium sulfate (50–75 mg/kg up to 2–4 g) has been shown to be effective in children with severe asthma exacerbations and can be used as an adjunct in these patients. Due to its bronchodilating effects, ketamine is an excellent induction agent for children who require rapid sequence intubation.

Bronchiolitis

Bronchiolitis is a clinical syndrome that comprises a group of presumed viral lung infections in children. Respiratory syncytial disease accounts for 70% of all cases of bronchiolitis, although parainfluenza, adenovirus and influenza can also be contributing agents. As bronchiolitis is not a uniform disease, treatment responses are variable; the literature is varied with regard to treatment recommendations.

Therapeutic options include nasal and deep suctioning, steroids, beta-agonists and nebulized epinephrine. The use of suctioning as an immediate intervention cannot be undervalued. Many infants with bronchiolitis have marked nasal congestion and secretions, and simple maneuvers such as nasal and deep suctioning can immediately improve their respiratory status.

The utility of steroid therapy in patients with bronchiolitis is controversial. The effectiveness of steroids is thought to be highest in cases in which the child has underlying reactive airway disease (usually bronchopulmonary dysplasia or asthma). However, steroids are not routinely indicated in the management of infants and children with bronchiolitis.

Beta-agonists seem to work for some children and not for others. A trial of two or three nebulized albuterol treatments may be undertaken in the ED. If effective, treatment may be continued as an outpatient using an inhaler with a spacer and mask, or as an inpatient with a nebulizer. If ineffective, further treatments are not usually helpful.

The use of nebulized epinephrine is currently controversial. Clinical experience suggests that some children respond well to nebulized epinephrine (at least transiently), whereas others do not. However, recently performed, well-designed studies have failed to show a significant benefit.

Antiviral treatment (e.g., ribavirin) has no role in the ED. A recent review of hypertonic saline for acute bronchiolitis revealed a lack of evidence to support its routine use in ED patients. However, nebulized 3% saline in combination with beta-agonists appeared to reduce hospital length of stays and clinical severity scores in patients admitted for bronchiolitis.

Croup

The treatment of croup is based on the severity of symptoms. A child with only a history of a barky cough yet no demonstrable cough in the ED is typically discharged for observation at home. A child with a demonstrable barky cough and no stridor at rest is treated with steroids as an outpatient. Historically, a single dose of dexamethasone has been given. Dosing ranges from 0.15–0.6 mg/kg, and the routes include oral, intravenous, intramuscular (IM) and nebulized. The IM route is rarely indicated because the oral route is just as effective; therefore, IM dexamethasone should be reserved for children in extremis or those unable to tolerate the oral route.

If a child has a demonstrable barky cough and stridor at rest, nebulized epinephrine may be administered in addition to dexamethasone. The dose of nebulized solution is 0.05 mL/kg (up to 0.5 mL) of 2.25% racemic epinephrine diluted in 3 mL of normal saline. Because only half of the racemic epinephrine solution is biologically active and 1:1,000 epinephrine is available in every crash cart, nebulized 1:1,000 epinephrine is now being used at a dose of 0.5 mL/kg (up to 5 mL) diluted in 3 mL of normal saline. Although cool mist has been advocated and used historically, clinical efficacy is doubtful. A recent, well-designed study suggested no demonstrable benefit.

If stridor persists at rest or multiple doses of racemic epinephrine are necessary, admission is typically warranted. If the stridor at rest resolves after nebulized epinephrine treatment, the child is usually observed in the ED for 2–3 hours. If stridor at rest returns, the child requires admission. If stridor at rest does not return and the child is well-appearing and tolerates oral fluids, discharge home is appropriate with close outpatient follow-up. There has been concern that children treated with nebulized epinephrine would "rebound" and substantially worsen after an initial period of clinical improvement; however, it is important to note that patients do not rebound to a condition worse than their initial clinical presentation.

Pneumonia

The treatment of pneumonia in children is primarily based on the likely etiology given the age of the child. Low socioeconomic status, poor nutrition, underlying disease, crowded living conditions and smoke exposure have all been implicated in increased risk of pneumonia. Common pathogens of pneumonia vary by age and are listed in Table 39.3.

In neonates, pneumonia is treated like all potentially serious bacterial infections, with intravenous ampicillin (100 mg/kg per dose) and cefotaxime (100 mg/kg per dose) plus admission to the hospital. In young infants, particularly those with minimal temperature elevation, repetitive coughing and a history of conjunctivitis, *Chlamydia* is the presumed etiology. Inpatient treatment for infections suspected to be caused by *Mycoplasma pneumoniae* or *Chlamydia pneumoniae* includes intravenous erythromycin (10 mg/kg, maximum 4 g) or azithromycin (5 mg/kg, maximum 500 mg). Well-appearing infants may be managed as outpatients with oral erythromycin or azithromycin.

For children between the first few months of life and 5 years of age, the most likely bacterial etiology of lobar pneumonia is *Streptococcus pneumoniae*. Treatment for this age group is with IV cefuroxime or ceftriaxone

(50 mg/kg per dose) for children admitted to the hospital, or oral amoxicillin (45–90 mg/kg per dose twice daily) for children discharged home. Presumed viral pneumonia (clinically indistinguishable from bacterial and along the spectrum of bronchiolitis) is not treated with antibiotics. So-called "atypical" pneumonia is most common in children older than 5 years of age, and macrolide antibiotics (e.g., azithromycin 10 mg/kg on day 1 – not to exceed 500 mg – and 5 mg/kg once daily for 4 subsequent days – not to exceed 250 mg per day) are appropriate.

The most common reason for children outside the neonatal period to be admitted is hypoxia. The pulse oximetry threshold that determines clinically significant hypoxia warranting admission is unknown. It has been suggested that children with room air pulse oximetry readings <90–92% should be admitted to the hospital.

Table 39.3 Common pathogens involved in pneumonia in children

Neonate (<30 days)	Group B streptococcus, *Listeria monocytogenes*, *Escherichia coli*, *Klebsiella*, *Staphylococci*, *Haemophilus influenzae* type B
1–3 months of age	RSV, *Streptococcus pneumoniae*, *Haemophilus influenzae* type B, *Staphylococcus aureus*, *Chlamydia*
3–24 months of age	RSV, parainfluenza, *Streptococcus pneumoniae*, *Haemophilus influenzae* non-type B
Toddlers	*Streptococcus pneumoniae*, *Haemophilus influenzae*, *Mycoplasma pneumoniae*; adenovirus, influenza
School age	*Mycoplasma pneumoniae*, *Streptococcus pneumoniae*, influenza, adenovirus

RSV: respiratory syncytial virus.

Special patients

Bronchopulmonary dysplasia

Former premature infants, particularly those with lung disease identified in the neonatal intensive care unit and those with prolonged mechanical ventilation, may have bronchopulmonary dysplasia. These children have reactive airways and often wheeze when they get upper respiratory tract infections. If they develop bronchiolitis, particularly if they test positive for RSV, they can have severe disease and may progress to respiratory failure. Similarly, pneumonia that is well-tolerated by other children may make children with bronchopulmonary dysplasia critically ill.

Cystic fibrosis

Although systemic and genetic, cystic fibrosis primarily manifests as a pulmonary disorder. These children have frequent lung infections and may have relatively unusual pneumonias caused by Gram-negative organisms, including *Pseudomonas aeruginosa*.

Sickle cell disease

A child with sickle cell disease may develop *acute chest syndrome*. These children typically present with fever, cough and chest pain. They may or may not be hypoxic. In most instances, it is difficult to distinguish lung infarction from a bacterial pneumonia; the two conditions may co-exist. These children are usually admitted to the hospital for hydration, beta-agonist administration, analgesia and pain control, and parenteral antibiotics.

Children with tracheostomies

Children with tracheostomies may have mechanical complications. Tracheostomy dislodgment may lead to cardiopulmonary arrest. Plugging of the tracheostomy tube with mucus may occur and lead to profound respiratory distress.

Disposition

Consultation

Although subspecialty consultation is infrequently needed in pediatric cases of difficulty breathing, it is prudent to speak with the primary care physician or the pulmonologist who has been caring for the child. These caregivers can often offer invaluable information about the patient and the care plan. Consultation (usually from an ENT specialist or a pediatric surgeon) for the removal of aspirated or esophageal foreign bodies is usually indicated. ENT surgeons are usually consulted to drain retropharyngeal abscesses in the operating room. Emergency physicians often perform needle drainage of peritonsillar abscesses in the ED if comfortable with this procedure.

Monitoring

For moderately ill children, it is sometimes difficult to predict whether they will improve or deteriorate. Close monitoring with frequent re-examinations and continuous pulse oximetry is prudent. Anticipating and responding to respiratory distress is a key management principle.

Intensive care unit admission

Any child who is endotracheally intubated will be admitted to an intensive care setting. Other children in whom it is desirable to aggressively treat without intubation (particularly asthmatics) may also be appropriate for the intensive care unit. Furthermore, any patient in whom impending airway compromise is possible (e.g., epiglottitis, retropharyngeal abscess, bacterial tracheitis) should be admitted to the intensive care unit.

Ward admission

Children who are dehydrated and require IV fluids should be admitted to the hospital. Children with croup and stridor at rest 2 hours after treatment with nebulized epinephrine should be admitted. Children with pneumonia who are suspected of being septic should be admitted to the hospital.

Young children with bronchiolitis who are slowly worsening should be admitted. Willwerth et al. recommended the following criteria for hospital admission of infants with bronchiolitis: (1) Full-term infants less than 1 month of age; (2) Premature infants (<37 weeks) who are less than 48 weeks post-conception; and (3) patients with witnessed apnea. Others recommend hospital admission for infants less than 2 months of age due to the risk of apnea.

Asthmatic children who require nebulized beta-agonist therapy more frequently than every 3 or 4 hours should strongly be considered for admission to the hospital. Due to a risk of apnea, an infant with a history and examination consistent with pertussis should be admitted for observation. Although the specific pulse oximetry value that constitutes clinically significant hypoxia is controversial, in general, hypoxic children should be given oxygen and admitted to the hospital.

Discharge

Many children who present with the complaint of difficulty breathing can be discharged home after treatment in the ED. Patients with croup who have been treated with racemic epinephrine should be observed prior to discharge. However, for children who present with mild symptoms and no stridor at rest, a dose of dexamethasone is all that is necessary prior to discharge. Infants over 2 months of age with bronchiolitis may be discharged if they are without respiratory distress and able to feed without difficulty. It is imperative that any child who presents with difficulty breathing has close follow-up the next day in order to ensure improvement on the regimen recommended by the ED provider.

Pearls, pitfalls and myths

Pearls

RSV bronchiolitis in young infants may initially present with apnea

Before the onset of wheezing, infants in the first few months of life may present after a brief period of apnea.

Inspiratory/expiratory, forced expiratory and decubitus chest X-rays may help identify cases of foreign body aspiration

More than 80% of aspirated foreign bodies are not directly visible on X-ray. Indirect findings should be sought. Due to a ball-valve mechanism, air trapping occurs on the side with the foreign body. Unilateral hyperexpansion may be seen on the chest X-ray, and this phenomenon may be exaggerated in an expiratory or decubitus film. In patients who cannot cooperate for inspiratory and expiratory radiographs, a forced expiratory view can be obtained by the radiology technician.

A child in moderately severe respiratory distress should be allowed to assume a position of comfort unless you are ready to aggressively manage the airway

Although we often remove children from their parents' arms to perform parts of the physical examination, agitating a child with a tenuous airway may lead to airway occlusion. Aggressive and prompt airway management may be required. If not prepared to immediately manage the airway of a young infant, keep the child in a position of comfort until a definitive management plan is in place.

Severe asthma may present without wheezing due to poor air exchange

In order to make the musical sound of wheezing, enough air has to pass through the airways. If there is no air movement, wheezing may not be heard.

A decrease in pulse oximetry does not necessarily indicate a worsening clinical condition in an asthmatic just starting bronchodilator therapy in the ED

As bronchodilator therapy is initiated, areas of lung that were previously "shut down" open up. There is a lag between the time when the blood flow normalizes in a region of lung and when the airways open up. A transient ventilation–perfusion mismatch develops, and the patient's oxygenation may actually fall briefly before improving.

Pitfalls

Slowing respirations may imply getting better or getting worse

As a patient improves, tachypnea usually normalizes. However, if a patient is worsening and developing respiratory fatigue, their respiratory rate will also fall. A slower respiratory rate has to be assessed in relation to the overall clinical appearance.

Vomiting and hydrocarbon ingestion

Hydrocarbons in the stomach and intestines are usually well-tolerated. Hydrocarbons in the lungs can cause a devastating chemical pneumonitis. Vomiting offers another chance for the hydrocarbon to go into the lungs.

Racemic epinephrine and dexamethasone

If a patient requires racemic epinephrine, always administer a dose of dexamethasone; otherwise, the patient may rebound and require additional doses of racemic epinephrine.

Myths

Wheezing means asthma

A frequently repeated mantra is "all that wheezes is not asthma." Other conditions, such as aspirated foreign bodies, chemical pneumonitis, bronchiolitis, cardiac disease and gastroesophageal reflux, often cause wheezing.

All wheezing children need albuterol

A trial of a bronchodilator is a reasonable intervention in young children with wheezing and respiratory distress. However, if the cause is not asthma, bronchodilators are often ineffective. If a child improves with a bronchodilator, it is reasonable to continue with additional doses. If a child has a foreign body or chemical pneumonitis, repeated doses of bronchodilators are not indicated. Although commonly administered, the effectiveness of bronchodilators in many cases of bronchiolitis is unclear.

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40 Syncope

Amal Mattu, MD

Scope of the problem

Syncope is defined as a transient loss of consciousness and postural tone caused by an abrupt decrease in cerebral perfusion, with subsequent spontaneous recovery. When recovery occurs prior to complete loss of consciousness, the episode is referred to as pre- or near-syncope. Syncope and pre-syncope are generally considered the same condition at different points along a continuum. Therefore, the emergency department (ED) evaluation and work-up for both are similar.

Syncope is a common presenting complaint in the ED, accounting for as many as 3% of all ED visits. According to the National Hospital Ambulatory Medical Care Survey: 2006 Emergency Department Summary, 288,000 patients were admitted to the hospital from the ED with the discharge diagnosis of syncope or collapse (ICD-9-CM code 780.2). This was 1.9% of all admissions from the ED that year. In the United States, $750 million are spent each year to diagnose and treat syncope. The differential diagnosis of syncope includes both benign and life-threatening etiologies. Emergency physicians must have a sound knowledge of diagnostic considerations in order to perform a comprehensive and cost-effective work-up, and make an appropriate disposition.

Pathophysiology

Consciousness is maintained through the proper functioning of the cerebral hemispheres and the reticular activating system (RAS). Syncope occurs when there is dysfunction of either both cerebral hemispheres or the RAS. Proper function of these structures depends on cerebral metabolism and delivery of oxygen and glucose. Disruption of this metabolism can occur due to generalized systemic hypoperfusion (e.g., cardiac dysrhythmia, hypovolemia with orthostasis), localized cerebral hypoperfusion (e.g., transient ischemia attack [TIA], stroke), systemic hypoxia, or hypoglycemia.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 40.1).

Table 40.1 Syncope red flags

History	**Concerning diagnosis**
Elderly (older)	Cardiac dysrhythmia, ACS
Palpitations	Cardiac dysrhythmia
Associated with severe headache	SAH or ICH
Associated with abdominal or back pain	Ruptured AAA or ectopic pregnancy
Associated with dyspnea	Pulmonary embolism
Associated with chest pain	ACS, pulmonary embolism, aortic dissection
Examination finding	**Concerning diagnosis**
Documented hypotension	Ruptured AAA or ectopic pregnancy, ACS, dysrhythmia, aortic dissection, sepsis, GI bleed or other blood loss
Head or face trauma	True syncope
Diaphoresis	Cardiac or vascular etiology, hypotension
Confusion	CNS event, hypoglycemia
Pulsatile abdominal mass	AAA
Vaginal bleeding	Ruptured ectopic pregnancy
Cardiac murmur	Aortic stenosis, hypertrophic CM
Wheezing	Pulmonary embolism
Pallor, melena, rectal bleeding	GI or other blood loss
Rales, JVD, peripheral edema	Cardiac disease or event, CHF

AAA: abdominal aortic aneurysm; ACS: acute coronary syndrome; CM: cardiomyopathy; CHF: congestive heart failure; CNS: central nervous system; ECG: electrocardiogram; GI: gastrointestinal; ICH: intracranial hemorrhage; JVD: jugular venous distention; SAH: subarachnoid hemorrhage.

History

Syncope has many causes, some benign and others life-threatening. As many as 45% of cases of syncope remain undiagnosed after a complete work-up. However, most etiologies of syncope that *are* established are made during the initial work-up in the ED. Only a minority of cases are diagnosed during inpatient admission. Therefore, a thorough history, physical examination and work-up in the ED are essential to identifying the cause.

What were you doing when you "passed out?"

A sudden change in position from supine to standing suggests syncope due to orthostatic hypotension (orthostatic syncope). However, syncope while supine indicates a cardiac etiology or a seizure (not a true cause of syncope, but generally included in the differential diagnosis). Syncope during exertion may also indicate orthostatic syncope, but may instead be caused by cardiac outflow obstruction (e.g., hypertrophic cardiomyopathy or aortic stenosis). Syncope associated with sudden head-turning, tight neck collars, or shaving the neck may be caused by carotid sinus hypersensitivity. Syncope that occurs shortly after exposure to pain or that is associated with strong emotions (e.g., fear, anxiety) is termed vasovagal or vasomotor syncope. Forceful prolonged coughing, defecation, micturition and weight-lifting (Valsalva maneuvers) can induce syncope by increasing intrathoracic pressure, leading to decreased venous return to the heart and decreased cardiac output.

What symptoms do you remember just before you "passed out?" What symptoms did you experience after you "woke up?"

Severe headache preceding a syncopal episode suggests an intracranial bleed, especially subarachnoid hemorrhage. Chest pain, palpitations, or dyspnea suggest cardiovascular etiologies, such as acute coronary syndrome (ACS), dysrhythmia, pericardial tamponade, or aortic dissection. A pulmonary cause, such as pulmonary embolism (PE), must also be considered. Aortic dissection is often accompanied by upper back pain. Abdominal or low back pain may indicate a ruptured abdominal aortic aneurysm (AAA) or ectopic pregnancy. A sense of progressive lightheadedness, weakness and "tunnel vision" immediately prior to the syncopal episode is often associated with vasomotor and orthostatic syncope, although this prodrome does not exclude more life-threatening causes. In contrast, the absence of any prodromal symptoms is often associated with a dysrhythmia. If the patient describes an aura, a seizure is probable. Patients reporting a prodrome of lightheadedness, nausea, diaphoresis and tremulousness should be evaluated for hypoglycemia.

Were there any witnesses?

Witnesses to the episode can help distinguish between certain causes of syncope. Witnesses should be questioned regarding the duration of unconsciousness. True syncope is generally associated with unconsciousness lasting no more than seconds to minutes. Prolonged unconsciousness before waking is more likely due to a seizure (postictal state) or drug effects (e.g., alcohol). Witnesses may report "seizure" activity. Tonic–clonic movements lasting for several seconds are common in cases of syncope; however, if the movements last more than 20–30 seconds, a seizure is the more likely cause. Witnesses should also be questioned regarding the mental status of the patient after waking. Seizures are associated with postictal confusion typically lasting more than 5 minutes. Syncope, on the other hand, is associated with a rapid return to a normal level of consciousness. Witnesses may also help describe the patient's activities and complaints immediately before and after the syncopal episode. Patients who reported feeling ill or lightheaded to witnesses, or who appeared pale or diaphoretic prior to the episode more likely experienced true syncope. The absence of these symptoms and signs, however, has limited utility.

Have you been feeling ill lately?

Recent nausea, vomiting, anorexia, or diarrhea suggests orthostatic syncope due to intravascular volume depletion. If the patient has had abdominal pain, vomiting, or diarrhea, specific questions pertaining to hematemesis or melena may indicate gastrointestinal blood loss as a cause of orthostatic syncope. Anemia from chronic blood loss is associated with a prolonged sense of fatigue, malaise and dyspnea with exertion. Recent episodes of chest pain or palpitations may indicate a cardiac cause of syncope. Recent abdominal or back pain in the days preceding the syncopal episode may suggest an expanding AAA or ectopic pregnancy prior to rupture.

What medications have you been taking? Any drugs or alcohol?

Many medications have been implicated in inducing syncope (Table 40.2). The primary mechanism through

Table 40.2 Drugs associated with syncope

Antidysrhythmics
Anticonvulsants
Antidepressants
Antiparkinsonism agents
Antipsychotics
Benzodiazepines
Beta-adrenergic blocking agents
Calcium channel blocking agents
Diuretics
Hypnotics
Narcotics
Nitrates
Phenothiazines

Adapted from De Lorenzo RA. Syncope. In Marx JA (ed). *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 7th ed. Mosby Elsevier, Philadelphia, PA, 2010.

which medication-induced syncope occurs is from excessive bradycardia or vasodilation. Patients who abuse illicit drugs and alcohol may also report that they "passed out." The period of unconsciousness associated with these drugs and alcohol is more prolonged before recovery than most other causes of syncope. Alchohol consumption, even in moderation, causes vasodilation and therefore predisposes individuals to syncope from other causes.

Has this ever happened before? Did you see a doctor? Was there any work-up?

Many types of benign syncope have a repeated pattern of occurrence. However, some life-threatening causes of syncope may also be associated with prior episodes, including dysrhythmias, hypertrophic cardiomyopathy and aortic stenosis. Information from prior work-ups helps rule in or exclude some life-threatening etiologies (e.g., prior echocardiography results will provide information regarding hypertrophic cardiomyopathy or aortic stenosis; prior cardiac catheterization results will help risk-stratify patients for suspected ACS).

Has any member of your family died suddenly?

Some of the life-threatening causes of syncope, including hypertrophic cardiomyopathy, Jervell Lang–Nielson, Romano–Ward and the Brugada syndromes, are associated with a family history of sudden death. Also, there may be a family predilection for subarachnoid hemorrhage (SAH). Emergency physicians should lower their threshold to perform a more extensive work-up in patients who report this family history.

Associated symptoms

Cardiopulmonary

Chest or upper back pain prior to or following a syncopal episode warrant strong consideration for ACS, aortic dissection, PE, or pericardial tamponade. Acute dyspnea also warrants consideration of PE and pericardial tamponade. Palpitations suggest a dysrhythmia.

Neurologic

The presence of a headache (especially of abrupt onset) prior to the syncopal episode suggests the possibility of intracranial hemorrhage, most commonly SAH. The presence of a headache after recovery is less specific, as many patients will report a post-syncopal (or post-seizure) headache. However, if the headache is severe or persistent, intracranial hemorrhage should be ruled out. Diplopia, dysarthria, ataxia, or vertigo prior to or after the syncopal episode indicates a posterior circulation stroke or TIA. Any neurologic complaint that is accompanied by chest or upper back pain should be considered an aortic dissection until proven otherwise.

Gastrointestinal

The presence of nausea, vomiting, anorexia, or diarrhea raises concern for orthostatic syncope due to intravascular volume depletion. Hematemesis or blood in the stool suggests gastrointestinal blood loss may be causing orthostatic syncope. Abdominal or low back pain may indicate a ruptured AAA.

Gynecologic

Ruptured ectopic pregnancy can cause sufficient blood loss to induce orthostatic syncope. The abrupt pain from a ruptured ectopic pregnancy can also cause vasomotor syncope. Clues to the diagnosis of ruptured ectopic pregnancy include known or suspected first-trimester pregnancy, irregular vaginal bleeding, and abdominal, pelvic, or low back pain. Frequent and heavy vaginal bleeding can induce severe anemia and also cause orthostatic syncope.

Extremities

Leg pain or swelling suggests deep venous thrombosis (DVT) and syncope due to PE. Leg pain may also be caused by acute vascular insufficiency, often associated with aortic dissection or AAA. Unilateral extremity weakness suggests a cerebrovascular cause of syncope.

Past medical

Cardiovascular

Cardiac risk factors increase the likelihood of cardiac causes of syncope, especially ACS and dysrhythmia. Hypertension and atherosclerotic coronary vascular disease also predispose to aortic dissection and AAA, respectively.

A history of previous dysrhythmia warrants work-up for recurrent atrial or ventricular dysrhythmias. A history of Wolff–Parkinson–White syndrome should prompt strong consideration of tachydysrhythmia-induced syncope.

Aortic stenosis is associated with exertional syncope. Advanced aortic stenosis can cause syncope with even minimal exertion. Valvular rupture or worsening incompetence may cause syncope from decreased cardiac output.

Pulmonary

Patients who report recent surgeries, immobilization, recent or current pregnancy, or other risk factors for thromboembolism should be evaluated for possible PE. Prior episodes of DVT or PE, as well as a family history, also predispose individuals to recurrent PE.

Physical examination

A comprehensive physical examination should be performed in all patients with syncope. Emphasis should be placed on the cardiovascular and neurologic examinations.

General appearance

If the patient appears pale, ashen or diaphoretic at presentation, a life-threatening cause of syncope should be considered until proven otherwise. This is especially true in older individuals. Intravascular volume depletion with shock due to a ruptured AAA or ectopic pregnancy, aortic dissection, gastrointestinal bleeding, sepsis, or severe dehydration are diagnostic possibilities. Shock may also be caused by an ACS or persisting dysrhythmia.

Vital signs

Hypotension indicates significant intravascular volume depletion or cardiogenic shock due to ACS or dysrhythmia. The heart rate can also indicate the presence of a brady- or tachydysrhythmia. A rapid heart rate may be caused by a dysrhythmia, or it may be a compensatory response to shock. Tachypnea is nonspecific, but may be a marker for PE or shock. A fingerstick glucose and pulse oximetry should be obtained as part of the routine vital sign assessment in patients with syncope as well. If the fingerstick glucose is noted to be low during the initial evaluation, hypoglycemia-induced syncope is suggested. This diagnosis should only be made if the history is supportive (i.e., the patient exercised without eating for many hours). Unconsciousness due to insulin-induced hypoglycemia is unlikely to resolve without administration of exogenous glucose. Hypoxia noted on pulse oximetry suggests PE. Orthostatic vital signs are often checked in patients with syncope; however, these have poor sensitivity and specificity for intravascular volume depletion, especially in elderly patients. Up to 30% of cardiac-related syncope cases will have positive orthostatic vital sign changes (heart rate increase of more than 30 beats per minute or systolic blood pressure fall of more than 20 mmHg). Fever or mild hypothermia may occur in the presence of sepsis or other focal infections. Low-grade fever may occur in patients with PE.

Head and neck

The head and neck should be assessed for signs of trauma that may have occurred during the syncopal episode. Pupillary or facial asymmetry may indicate a cerebrovascular cause of syncope. On the other hand, bilateral pupillary constriction or dilation suggests a medication side effect or drug ingestion. Most narcotics induce pupillary constriction, whereas anticholinergics and sympathomimetics may cause pupillary dilation. Carotid pulses should be assessed for symmetry and bruits. Some authors have suggested carotid sinus massage (CSM) to assess for carotid sinus hypersensitivity. However, this should only be done in patients who have a history suggestive of this diagnosis (e.g., syncope while shaving), and in whom the risk of carotid atherosclerotic plaques is negligible. The mouth should be assessed for evidence of trauma, such as tongue-biting, which is more common after seizures than syncope.

Cardiovascular

The cardiac examination is the most important part of the physical examination in patients presenting with syncope. The ventricular rate should be assessed, and may indicate the presence of a tachy- or bradydysrhythmia. The heart rhythm should be assessed for regularity. An irregular rhythm suggests atrial fibrillation, premature complexes, or second-degree atrioventricular (AV) block. Beware, however, that a normal heart rate and rhythm does not exclude a transient dysrhythmia as the possible cause of syncope. Carefully auscultate for murmurs. Aortic stenosis is associated with a crescendo–decrescendo systolic murmur, heard loudest at the upper sternal border and radiating to the carotid arteries. Hypertrophic cardiomyopathy is also associated with a systolic murmur, but the intensity is greatest at the lower sternal border or apex. The intensity of the murmur of hypertrophic cardiomyopathy generally increases with Valsalva maneuvers and decreases with the Trendelenburg position and squatting. The vascular examination is crucial as well. Assess the quality of upper and lower extremity pulses. Unequal pulses between the upper extremities suggest aortic dissection in the appropriate clinical scenario.

Pulmonary

Abnormal lung sounds suggest a pulmonary cause of syncope. Rales can be caused by pulmonary edema due to myocardial ischemia or infarction. Focal wheezes can be caused by a PE. Diffuse wheezes may be due to multiple pulmonary emboli or pulmonary edema.

Abdomen

The abdomen should be assessed for tenderness. Consider a ruptured AAA or ectopic pregnancy in a patient with syncope and abdominal tenderness. An enlarged pulsatile abdominal aorta may be appreciated in some cases of aortic aneurysm.

Pelvic

The pelvic examination is useful in a patient in whom ruptured ectopic pregnancy is a consideration. Any female of childbearing age with syncope who reports pelvic or abdominal pain, vaginal bleeding, or is known to be pregnant in the first or second trimester should have a pelvic examination to assess for vaginal bleeding, adnexal masses, or adnexal and uterine tenderness.

Rectal

The rectal examination should be considered a routine part of the physical examination in a patient presenting with syncope. Gross blood or melena suggests significant gastrointestinal bleeding. The presence of occult blood may indicate chronic gastrointestinal blood loss and anemia.

Neurologic

A detailed neurologic examination should be performed on all patients who present following a syncopal episode. A depressed level of consciousness may be caused by persistent shock, hypoxia, hypoglycemia, seizure (postictal state), stroke, intracranial bleed, or drug/medication effect. Vertigo, ataxia, dysarthria, or any focal neurologic deficits indicate a stroke or intracranial hemorrhage. Neurologic symptoms or signs accompanied by chest or upper back pain should be considered an aortic dissection until proven otherwise.

Differential diagnosis

Table 40.3 lists a number of causes of syncope.

Table 40.3 Differential diagnosis of syncope

Causes of syncope	Symptoms	Signs	Work-up
Acute coronary syndrome	Chest pain or other “anginal equivalent” is usually present. Elderly patients and diabetics are prone to more atypical and painless presentations.	Patients usually appear uncomfortable; signs of heart failure (hypoxia, rales) may accompany ACS.	ECG and cardiac biomarker testing is warranted. Patients should be admitted. Isolated syncope without anginal symptoms or ECG abnormalities is very unlikely due to ACS.
Aortic dissection	Classically, patients with AD present with sudden onset of “ripping” or “tearing” chest pain with radiation to the upper back. Up to 15% present with syncope, but the majority will have preceding or post-syncope chest or upper back pain.	Although classically these patients are severely hypertensive, two-thirds will be normotensive or hypotensive. Suggestive findings include unequal pulses in the extremities or associated neurologic deficits.	CXR is neither sensitive nor specific enough to be a definitive test for the diagnosis. If the diagnosis is suspected based on history and physical examination, CT of the great vessels with IV contrast should be obtained. Alternatively, emergent aortography or TEE can be performed with equal or greater sensitivity, although they are usually less available in the ED.
Aortic stenosis	Syncope in patients with AS usually occurs with exertion. Severe AS may produce syncope with even mild amounts of exertion. Patients are usually elderly.	The main physical examination finding is a systolic crescendo–decrescendo murmur heard best at the upper sternal border that radiates to the carotid arteries.	The diagnosis of AS is established during echocardiography. The ECG usually demonstrates left atrial and left ventricular enlargement, but these are nonspecific findings.
Carotid sinus hypersensitivity	Syncope is associated with maneuvers that result in direct pressure applied to the carotid sinus (e.g., shaving, tight-fitting collar), and sudden turning of the neck.	The vital signs and examination after recovery is normal. CSM may reproduce syncope, but this maneuver is reserved for patients that have negligible risk of carotid atherosclerotic disease.	No specific work-up is indicated, although patients should be warned about factors associated with recurrence.
Dysrhythmia	The classic presentation is an abrupt onset of syncope without prodrome. On the other hand, palpitations or chest pain may precede the syncopal episode.	Tachycardia, bradycardia, or irregular pulses are typical. Signs of heart failure may be present if the dysrhythmia is severe or persistent.	The ECG may show an obvious dysrhythmia or more subtle findings, including signs of Brugada syndrome (right bundle branch block or incomplete right bundle branch block pattern with ST-segment elevation in right precordial leads), WPWS (short PR interval, wide QRS interval and delta-wave), or prolonged QT interval; the ECG may also be completely normal. Admission for cardiac monitoring is recommended.
Hypertrophic cardiomyopathy	Syncope in these patients often occurs with exertion, but may also occur at rest. Patients are often young athletes, although the average age at diagnosis is 30–40 years.	Examination is usually notable for a systolic murmur heard best at the lower sternal border or apex. The murmur intensity increases with maneuvers that decrease venous return, such as Valsalva or standing, and decreases with maneuvers that increase venous return, such as Trendelenburg or squatting.	The diagnosis of hypertrophic cardiomyopathy is definitively established during echocardiography.

(*continued*)

Table 40.3 Differential diagnosis of syncope (*cont.*)

Causes of syncope	Symptoms	Signs	Work-up
Intracranial hemorrhage	Patients usually report the presence of a severe or sudden headache prior to the syncopal episode, or a severe persistent headache after the episode. Some patients may report neurologic abnormalities as well.	A detailed neurologic examination may reveal subtle or overt neurologic deficits. Subarachnoid hemorrhage, however, may be associated with a normal examination.	CT of the head is diagnostic for intraparenchymal hemorrhage and will detect more than 90% of subarachnoid hemorrhages. Lumbar puncture should be performed in patients who have a headache associated with syncope if the CT is negative.
Medication effects	Syncope due to medications may occur in a fashion similar to that of orthostatic syncope. Associated weakness and lightheadedness tend to be more persistent. However, patients usually report a recent change in or addition to their medication regimen.	Orthostatic hypotension or persistent bradycardia is common. Toxicity due to anticholinergic medications may be associated with warm, dry, erythematous skin and dilated pupils. Narcotic toxicity is associated with a persistent decreased level of consciousness and miosis.	Signs of medication toxicity, especially medications that have type IA antidysrhythmic effects, often manifest abnormalities on the ECG; therefore, an ECG should always be obtained. Medication levels should be checked when appropriate.
Orthostatic syncope	This type of syncope, associated with intravascular fluid depletion (dehydration, acute or chronic blood loss) occurs when there are abrupt changes in body position from supine to standing or with walking.	Intravascular fluid depletion may be associated with tachycardia and/or hypotension. Orthostatic vital sign changes are common, though not 100% sensitive or specific. Signs of blood loss may be present.	Serum electrolyte testing is indicated if the patient reports ongoing vomiting or diarrhea. Hemoglobin testing is indicated if the patient reports blood loss (e.g., hematemesis, melena, heavy vaginal bleeding) or if there is evidence of blood loss on the examination (e.g., fecal occult blood).
Pulmonary embolism	PE is associated with syncope in 10–15% of cases. Patients usually complain of dyspnea or pleuritic chest pain. If the source of the embolism is from a DVT, the patient may also complain of leg pain or swelling.	The majority of patients will have tachypnea (respiratory rate ≥20/min). Approximately half will also have tachycardia. Lung sounds are variable; wheezing is common, but the lungs may be clear.	CXR is often obtained to evaluate for alternative diagnoses. Further work-up for PE varies between physicians. Testing may involve CT of the lungs with IV contrast, V/Q scanning, D-dimer testing, or any combination of the above. Pulmonary angiography is considered the gold standard test.
Pericardial tamponade	Patients typically complain of dyspnea. Chest pain associated with pericarditis may precede the development of tamponade.	Tachycardia and hypotension are very common. The lungs are usually clear. Jugular venous distension is typical unless the patient is hypovolemic.	The ECG often shows evidence of low voltage or *electrical alternans.* CXR typically shows massive cardiomegaly. Definitive diagnosis is made with emergent echocardiography (diastolic collapse of the right atrium and right ventricle).
Ruptured ectopic pregnancy	Syncope occurs in 10–15% of patients with ruptured ectopic pregnancy. Patients usually report abdominal pain, pelvic pain, low back pain, or vaginal bleeding.	Hypotension may be present if significant blood loss has occurred. The examination may be notable for abdominal and adnexal tenderness, with vaginal blood on the pelvic examination.	Any woman of childbearing age who presents with syncope in association with abdominal, low back or pelvic pain, or vaginal bleeding should have immediate pregnancy testing. If the test is positive, obtain an immediate abdominal and pelvic ultrasound, as well as a quantitative serum hCG level, type and Rh, and "pre-op" labs.
Ruptured abdominal aortic aneurysm	As many as 10–15% of patients with ruptured AAA present with syncope. These patients usually complain of abdominal or low back pain prior to or after the syncopal episode.	Classic findings include hypotension and an enlarged, palpable pulsatile abdominal aorta. However, more than one-third of patients are normotensive, and more than two-thirds do not have an enlarged palpable aorta noted on initial examination. Femoral pulses may be unequal, but this finding is not sensitive.	If a ruptured AAA is suspected, surgical consultation should be obtained immediately. Bedside ultrasound is helpful in evaluating for the presence of an aneurysm. If a bedside ultrasound is not available, abdominal CT with IV contrast can be obtained. However, if the patient is hemodynamically unstable and the diagnosis is highly suspected, immediate vascular surgical consultation is warranted.

(*continued*)

Table 40.3 Differential diagnosis of syncope (*cont.*)

Causes of syncope	Symptoms	Signs	Work-up
Stroke or transient ischemic attack	Stroke/TIA are rare causes of syncope. A stroke that induces a loss of consciousness (disrupting perfusion to the bilateral cerebral hemispheres or RAS) is unlikely to be associated with a normal neurologic examination after recovery. One exception is the posterior circulation stroke or TIA, associated with dysarthria, diplopia, ataxia, or vertigo. In the absence of these symptoms, stroke and TIA are unlikely.	Syncope due to stroke will invariably be associated with neurologic deficits associated with the posterior circulation of the brain.	CT of the head should be performed in patients with syncope who have neurologic deficits on regaining consciousness. However, if a detailed neurologic examination is normal, the yield of CT is extremely low and may not be warranted.
Seizure	Patients with seizures often report a preceding aura. No other self-reported symptoms are particularly helpful in distinguishing seizures from true syncope.	If the patient presents during the postictal period, seizure is most easily diagnosed based on the confusion that slowly resolves. Another characteristic that has been shown to be more specific for seizure (vs. true syncope) is evidence of tongue-biting. Loss of bowel/bladder control is fairly nonspecific.	Patients with a first-time seizure usually require an extensive ED work-up, including laboratory testing, drug and alcohol screening, and CT (or MRI) of the head. Patients with a history of prior seizures who have a recurrent uncomplicated seizure followed by a brief postictal period usually require nothing more than anticonvulsant level testing.
Vasomotor (also referred to as "vasovagal" or "vasodepressor")	Includes episodes associated with emotional factors (e.g., sudden exposure to fear or anxiety) and situational factors (e.g., micturition). The episode is usually preceded by a prodrome of progressive lightheadedness and tunnel vision. Recovery occurs quickly after the patient assumes a supine position.	If the heart rate is obtained at first onset of symptoms, transient bradycardia is usually noted. Patients are often diaphoretic, cold and clammy during the first few minutes following recovery, but the remainder of the examination is normal.	No specific work-up is indicated other than a thorough history and physical examination.

AAA: abdominal aortic aneurysm; ACS: acute coronary syndrome; AD: aortic dissection; AS: aortic stenosis; CSM: carotid sinus massage; CT: computed tomography; CXR: chest X-ray; DVT: deep venous thrombosis; ECG: electrocardiogram; ED: emergency department; hCG: human chorionic gonadotropin; IV: intravenous; MRI: magnetic resonance imaging; PE: pulmonary embolism; RAS: reticular activating system; TEE: transesophageal echocardiography; TIA: transient ischemic attack; V/Q: ventilation–perfusion; WPWS: Wolff–Parkinson–White syndrome.

Diagnostic testing

A "shotgun" approach to diagnostic testing in the patient with syncope wastes valuable time and resources. The electrocardiogram (ECG) and bedside glucose are perhaps the most important diagnostic tests routinely indicated in these patients. All other tests should be obtained based on concerns raised during the detailed history and physical examination.

Electrocardiogram

The ECG should be considered a routine part of the evaluation of a patient presenting with syncope. An abnormal ECG is the most important predictor of cardiac causes of syncope, dysrhythmias and adverse outcomes. Emergency physicians should evaluate the ECG for evidence of tachydysrhythmias, bradydysrhythmias and AV blocks. The presence of any of these abnormalities suggests a dysrhythmia as the cause of the syncopal episode. The ECG should also be evaluated for evidence of acute myocardial ischemia (ST-segment elevation or depression, inverted T waves, or new intraventricular conduction abnormalities or bundle branch blocks). PE may be associated with sinus tachycardia, inverted T waves (especially in the right precordial leads), a rightward axis, Q waves in lead III, and tall R waves in lead V_1 (Figure 40.1). The "classic" $S_1Q_3T_3$ ECG pattern is commonly described, but these findings are infrequently present. Pericardial tamponade is suggested by the combination of tachycardia plus low QRS voltage; electrical alternans is present in one-third of cases (Figure 40.2). Close attention should be paid to the ECG intervals. A short PR interval in combination with a slightly prolonged QRS interval and delta waves is diagnostic of Wolff–Parkinson–White syndrome

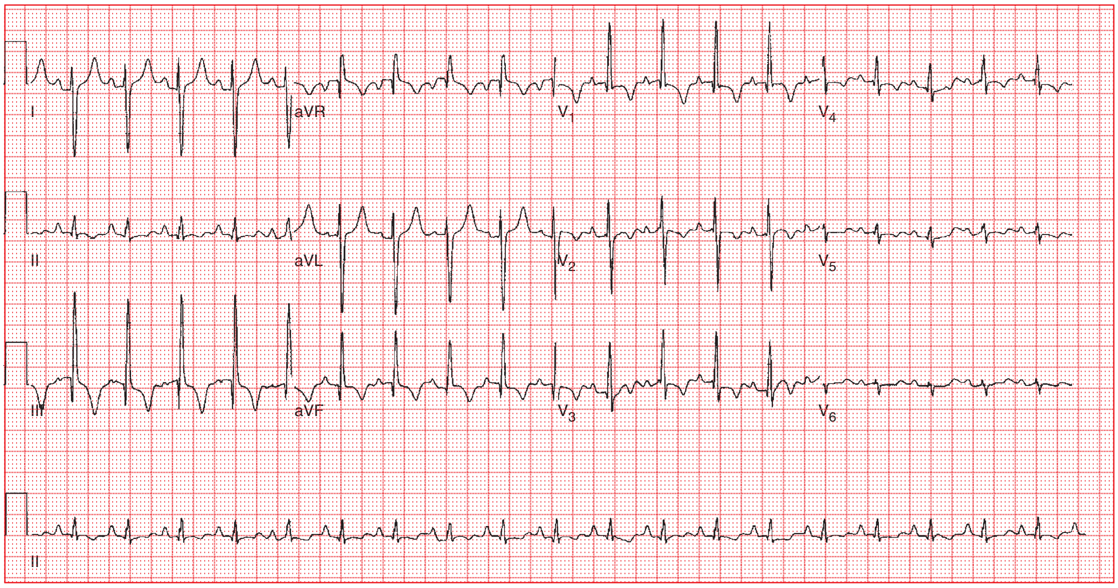

Figure 40.1
ECG in a patient with pulmonary embolism demonstrating the "classic" $S_1Q_3T_3$ pattern and rightward axis shift. T wave inversions in leads V_1–V_3 are indicative of right heart strain.

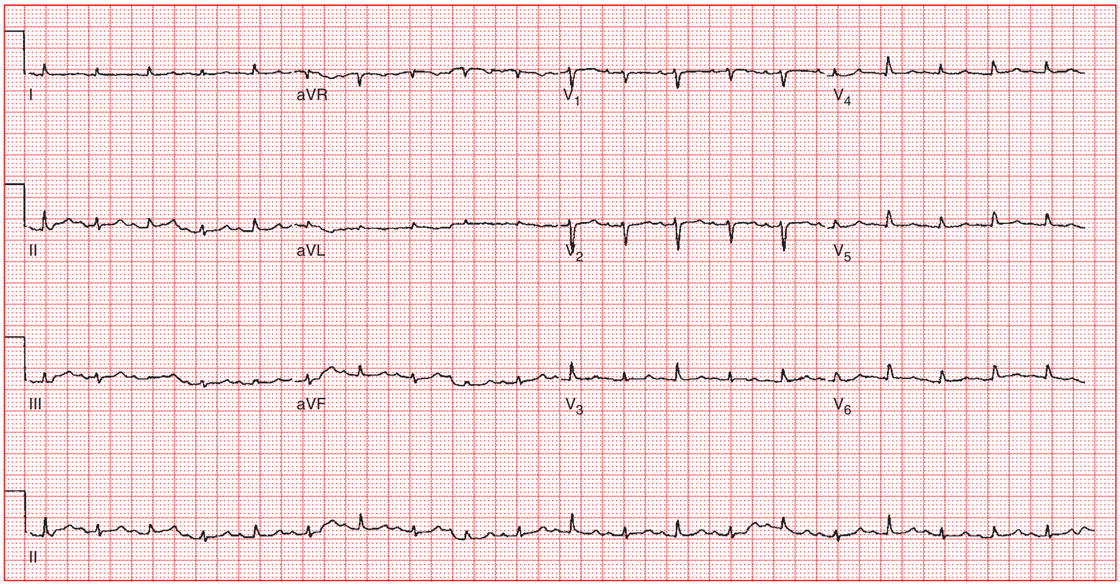

Figure 40.2
ECG in a patient with cardiac tamponade demonstrating low QRS voltages and electrical alternans.

(Figure 40.3), and increases the likelihood of tachydysrhythmias. Prolongation of the QT interval, which may be caused by electrolyte abnormalities, medications, or congenital defects, predisposes to polymorphic ventricular tachycardia. Less common conditions that predispose to ventricular tachydysrhythmias include hypertrophic cardiomyopathy and Brugada syndrome. Hypertrophic cardiomyopathy is identified on the ECG when large-amplitude QRS complexes, tall R waves in the right precordial leads, and deep narrow Q waves in the inferior or lateral leads are present (Figure 40.4). The Brugada syndrome is identified on the ECG by the presence of a complete or incomplete right bundle branch block pattern with ST-segment elevation in the right precordial leads (Figure 40.5). Hypertrophic cardiomyopathy and Brugada syndrome are confirmed with Doppler echocardiography and electrophysiologic testing, respectively. Nonspecific ECG abnormalities, such as premature atrial or ventricular complexes, sinus tachycardia, and first-degree AV block, have no diagnostic significance in an asymptomatic patient.

Laboratory studies

In general, laboratory studies should be ordered when the history or physical examination suggests a high likelihood of abnormality.

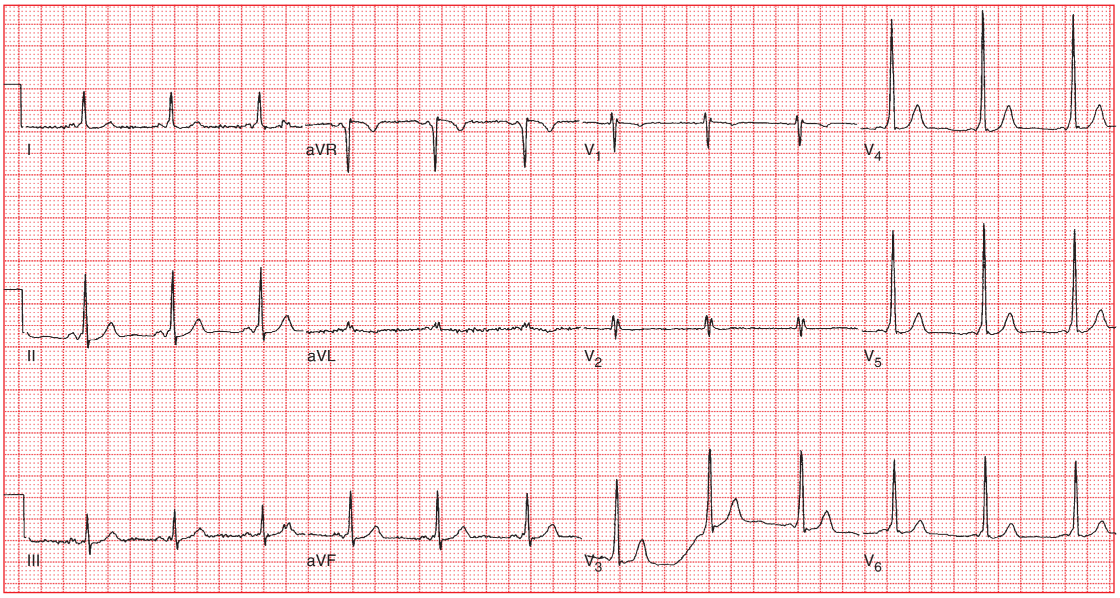

Figure 40.3
ECG in a patient with Wolff–Parkinson–White (pre-excitation) syndrome demonstrating short PR intervals and delta waves.

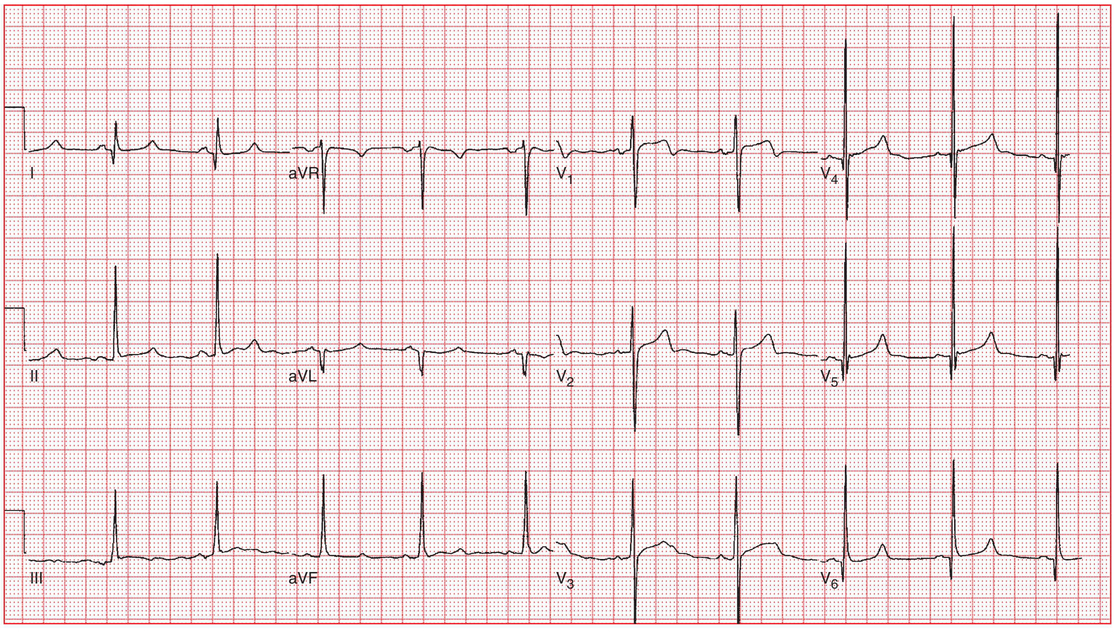

Figure 40.4
ECG in a patient with hypertrophic cardiomyopathy demonstrating high QRS voltage, left ventricular hypertrophy (LVH), and a "pseudo-infarct pattern" (Q waves in V_4–V_6).

Glucose

Routine serum glucose testing is of low yield in a patient who is asymptomatic on arrival to the ED. However, patients with persistent lightheadedness, nausea, tremulousness or diaphoresis should have their glucose tested at the bedside. A point-of-care glucose should be obtained in all diabetics and alcoholics presenting with syncope.

Complete blood count

The complete blood count (CBC) is rarely helpful in evaluating patients with syncope. The white blood cell count is nonspecific. Hemoglobin and hematocrit levels are useful when the history or physical examination suggests blood loss (most commonly from a gastrointestinal or vaginal source). Patients with renal failure, human

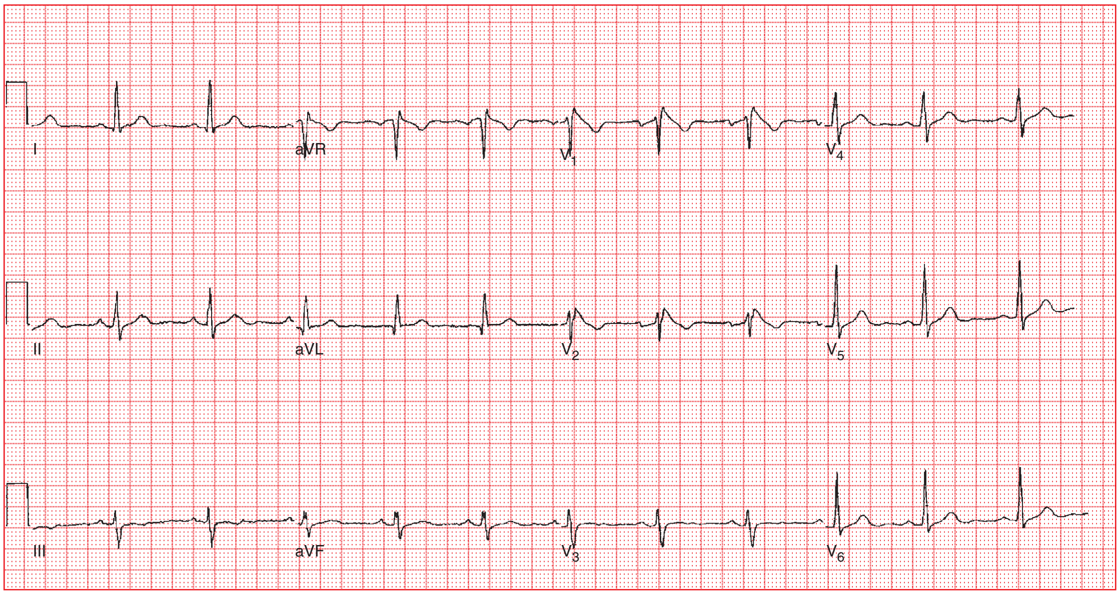

Figure 40.5
ECG in a patient with Brugada syndrome demonstrating coved ST-segment elevation in leads V_1 and V_2, and right bundle branch pattern (absence of terminal S waves in leads I and V_6).

immunodeficiency virus, sickle cell anemia, and other chronic diseases are predisposed to anemia and warrant routine CBC testing.

Electrolytes

Routine electrolyte testing is rarely helpful in the evaluation of a patient with syncope. However, patients taking diuretics, with profound vomiting and/or diarrhea, and the elderly warrant electrolyte testing. Electrolytes are often abnormal in patients with renal failure and diabetes. An electrolyte abnormality may be responsible for prolonged QT syndrome when this finding is present on the ECG.

Cardiac biomarkers

Patients who report concurrent chest pain or other potential anginal symptoms warrant cardiac biomarker testing. In the absence of these symptoms, however, the routine use of cardiac biomarkers in patients with syncope has a very poor yield. Syncope is a rare isolated manifestation of ACS, although it is a possible presenting symptom in PE.

Pregnancy test

Female patients of childbearing age who present with vaginal bleeding or abdominal, pelvic, or low back pain and syncope should be tested for pregnancy. However, in the absence of any of these symptoms, pregnancy testing is unlikely to be helpful.

Radiologic studies

Routine radiologic imaging for patients with syncope is not indicated. However, the history and physical examination may help determine which patients require focused imaging.

Chest radiography

Routine chest radiography for patients with syncope is not cost-effective and rarely leads to a definitive diagnosis. Patients who complain of dyspnea, chest or upper back pain, or who are hypoxic, tachypneic, or have abnormal lung sounds should receive chest radiography as an initial screening tool to evaluate for pneumonia, pneumothorax, or PE. The most common radiographic findings in PE include elevation of a hemidiaphragm, atelectasis and small pleural effusions; less common findings include pulmonary infiltrates and peripheral oligemia. The chest radiograph may also display evidence of congestive heart failure (CHF) associated with ACS, large pericardial effusions (massive cardiomegaly, water bottle–shaped heart), and aortic dissection in patients with chest or upper back pain (widened mediastinum, left pleural effusion, blurring of the aortic knob, rightward tracheal deviation, pleural cap).

Computed tomography of the chest

Patients with suspected aortic dissection or PE based on history and physical examination should have diagnostic imaging of the great vessels or lungs, respectively. Computed tomography (CT) is emerging as the diagnostic test of choice for both conditions, in part due to the accessibility of CT in most EDs. However, emergency physicians should remember that the test is not 100% sensitive; therefore, if clinical suspicion of either diagnosis is high, additional testing may be indicated even after a negative CT.

Computed tomography of the brain

Patients who experience syncope following recent head trauma should have a CT of the brain. Emergency physicians should also consider CT of the brain in patients who experience head trauma during the syncopal episode, although studies evaluating the utility of this test in this situation are lacking. Any patient with syncope who experiences a severe headache prior to or after the syncopal episode should have a brain CT as part of the work-up for intracranial hemorrhage. Also, patients who experience new seizures, report focal neurologic symptoms, or have new focal neurologic deficits should have emergent CT of the brain. Overall, neurologic causes of syncope are rare, especially in patients without focal neurologic symptoms or deficits. Routine brain CT is not indicated in these patients.

Echocardiography

Emergent echocardiography is indicated in cases of suspected pericardial tamponade or acute valvular dysfunction (e.g., post–myocardial infarction papillary muscle rupture). Echocardiography may also be helpful in evaluating wall motion abnormalities in suspected ACS or right heart strain in suspected massive PE. However, it is rarely required for diagnosis of either condition in the ED. Echocardiography is also diagnostic for aortic stenosis and hypertrophic cardiomyopathy, but obtaining it emergently for either condition is rarely necessary.

General treatment principles

Due to the many life-threatening etiologies of syncope, treatment should begin with assessment of the ABCs (airway, breathing, circulation). Once initial stabilization has occurred, further treatment will be guided by the presumed etiology. Patients with an uncertain diagnosis should be monitored and observed in the ED. Bed rails should be maintained in an upright position, and protective padding placed in the event of a seizure. Placement of an intravenous (IV) line is prudent in case emergent medications or IV fluids are required.

Special patients

Elderly

Elderly patients are known to present with atypical symptoms and signs, even in the presence of deadly diseases. Many patients greater than 75 years of age have dyspnea rather than chest pain as their symptoms of ACS. A small but significant proportion of elderly patients will experience painless aortic dissection. Elderly patients are more likely than younger patients to have cardiovascular causes of syncope, such as ACS, dysrhythmias, aortic dissection, ruptured AAA, and aortic stenosis. Vital signs can be misleading; cardiac medications may blunt an expected tachycardic response to acute blood loss, and this population is prone to false-positive and false-negative orthostatic vital signs. Polypharmacy is common in elderly patients as well, increasing the likelihood of medication-related syncope. Elderly patients are more likely to have electrolyte abnormalities, anemia and intracranial abnormalities. Emergency physicians should maintain a lower threshold for ordering laboratory studies (including cardiac biomarkers) and CT of the brain, although a detailed history and physical examination are still most useful for predicting the yield of these tests in the ED. Because elderly patients experience a much higher mortality rate from their causes of syncope, a liberal policy for routine admission in order to receive an expedited work-up or possibly identify the cause is warranted.

Pacemakers

Patients with artificial cardiac pacemakers who present with syncope should have at least 24 hours of cardiac monitoring and an evaluation for pacemaker malfunction. This usually requires cardiologist consultation and admission.

Pregnant

Patients who are pregnant, especially during their second and third trimester, are prone to vasomotor and orthostatic syncope. However, pregnancy also increases the risk for PE. Therefore, pregnant patients who present with or after syncope should be evaluated for PE if they are tachypneic, hypoxic, or report chest pain. Cardiotocography (fetal monitoring) is reasonable for patients whose gestational age is greater than 20 weeks.

Pediatric

Syncope in pediatric patients is rare. The most common causes are vasomotor syncope and syncope related to dehydration. Adolescents who report exertional syncope should be assessed for hypertrophic cardiomyopathy. In addition to hypertrophic cardiomyopathy, an ECG should be obtained in the pediatric population to assess for evidence of Wolff-Parkinson-White, prolonged QT, and Brugada syndromes.

Disposition

Immediate surgical/obstetric consultation

Patients with suspected aortic dissection, ruptured AAA, or pericardial tamponade warrant immediate surgical consultation. Patients with suspected ectopic pregnancy should have immediate gynecologic consultation. All patients with catastrophic causes of syncope requiring surgical intervention need two large-bore IV lines, a broad panel of "pre-op" labs, and a type and crossmatch for at least 4 units of blood.

Table 40.4 Syncope rules to predict short-term serious outcomes

FED 30 90	
F	Failure (congestive heart failure)
E	ECG abnormalities
D	Dyspnea (shortness of breath)
30	Hematocrit < 30%
90	Systolic blood pressure < 90 mmHg (at any time)

Modification of San Francisco Syncope Rules from Quinn JV, Stiell IG, McDermott DA, et al. Derivation of the San Francisco Syncope Rule to predict patients with short-term serious outcomes. *Ann Emerg Med* 2004;43(2):224–32. Modified by Gus M. Garmel, MD.

Admission

All patients with a suspected cardiovascular, pulmonary, or neurologic cause of syncope should be admitted. Patients with cardiac risk factors should be considered for admission for cardiac monitoring. Although there is no specific age at which routine admission of patients experiencing syncope occurs, most experts recommend that patients greater than 50 years of age should be strongly considered for routine admission to monitor for dysrhythmias and evaluate for other cardiac causes. Clinical decision instruments have been published to determine those patients with syncope at greatest risk for serious short-term outcomes (Table 40.4). However, validation studies have demonstrated conflicting results. Consequently, these decision instruments should be considered guidelines for consideration rather than firm rules for determining disposition.

Discharge

The most common cause of syncope in young patients with a normal physical examination and ECG is vasomotor syncope. Patients with this benign condition can be discharged with close outpatient follow-up. These patients should be counseled to maintain adequate hydration, as hypovolemia increases the likelihood of syncope. Patients should also be instructed to lie down immediately if they begin to experience a similar prodrome as that which preceded their syncopal episode. In general, all patients who are discharged after a syncopal episode should follow-up with their primary care physician or cardiologist for re-evaluation within 1 week. In addition, patients should be instructed to avoid swimming, driving, climbing, or operating heavy machinery until further evaluation. Patients who have an uncomplicated seizure with a known seizure disorder should be counseled to maintain proper compliance with medications and to follow up with their physician for reassessment of their medication regimen.

Pearls, pitfalls and myths

Pearls

- Abrupt onset of syncope without prodromal symptoms should prompt strong suspicion of a dysrhythmia.
- Up to 30% of cardiac-related syncope cases have positive orthostatic vital sign changes.
- The ECG is useful not only for identifying myocardial ischemia and dysrhythmias, but also for evaluating for Wolff–Parkinson–White syndrome, pericardial tamponade, prolonged QT syndrome, hypertrophic cardiomyopathy, PE and Brugada syndrome.

Pitfalls

- Gastrointestinal bleeding may be missed because a rectal examination was not performed.
- Hypertrophic cardiomyopathy may be missed because a murmur (or changes in the murmur with provocative maneuvers) was not detected in a young patient.
- PE and SAH are commonly overlooked as potential causes of syncope.

Myths

- CT of the head should be performed on all patients who present after syncope.
- Serum electrolyte and CBC testing must be performed on all patients who present after syncope.
- Orthostatic vital sign abnormalities are highly specific for dehydration and rule out cardiac causes of syncope.

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41 Toxicologic emergencies

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Scope of the problem

The American Academy of Poison Control Centers (AAPCC) reported 2.4 million human exposures to toxic agents in 2007. The demographics of human poisoning show two peaks of exposure. The first peak is in children from 1–3 years of age, reflecting accidental ingestions. The second peak is in the elderly due to the increased risk of toxicity from multiple prescription drugs. Intentional overdose for abuse or suicidal intent is seen throughout the adolescent and adult populations.

The most common exposures are analgesics (e.g., acetaminophen [APAP], ibuprofen, aspirin) and cleaning products. The agents responsible for the most fatalities are sedative/hypnotics, opioids, analgesics, antidepressants, cardiovascular agents and drugs of abuse. Carbon monoxide (CO) and drugs of abuse are responsible for many deaths never reported to a poison control center (PCC). Data show that analgesics, antidepressants and cardiovascular agents are also associated with significant morbidity.

Most patients who are poisoned will recover with supportive care alone. However, some may require specific antidotes or other therapeutic measures under the guidance of a regional PCC or medical toxicologist. The identification and treatment of poisoning emergencies is part of the unique and specialized knowledge of emergency physicians and medical toxicologists. Emergency physicians are on the front line of caring for these patients and must be familiar with the principles of poisoning diagnosis and management.

Anatomic essentials

There are three basic physiologic concepts required to understand the effects of toxic agents on the human body: absorption, distribution and elimination. In addition, each toxic substance has specific mechanisms of toxicity and effects on organ systems that are not described in this chapter but can be found in some of the references.

Absorption is defined as the process by which a drug or chemical enters the body. Drugs can be absorbed through a variety of pathways, including the lungs, skin, muscle tissue, oral mucosa, stomach, intestine, rectal mucosa and eyes. The route of absorption directly affects its rate and extent. Absorption is also affected by the formulation of the agent and by host factors, such as skin thickness or gastric motility.

Distribution is the process by which a drug or chemical that has reached the bloodstream is transported to peripheral tissues. Distribution is a complex process with a large number of variables. Volume of distribution (V_d) is the apparent volume into which a drug distributes. It reflects the drug's distribution into all of the body tissues. A drug that is distributed only in the plasma will have a V_d near that of total body water (between 0.6 and 1 L/kg). Drugs that are heavily concentrated in the body fat will have a V_d much greater than that of total body water.

Elimination is the removal of a drug or chemical from the body. Elimination can occur either by excretion or through biotransformation to one or more metabolites. The lungs, kidneys and liver are responsible for the majority of drug elimination. Elimination is affected by integrity of organ systems, age, saturation of key enzyme processes, and the specific properties of the drug.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 41.1).

History

The primary goal of the history is to identify whether a poisoning occurred and which toxic agents were responsible. This can range from a simple to extremely complex process, depending on the ability and willingness of the patient to answer historical questions, and the extent to which corroborating information is available. Historical information should be solicited from the patient, family members, companions, friends, emergency medical services (EMS), law enforcement, or other available witnesses.

Questions for the patient

What drugs or pills did you take or what chemicals were you exposed to?

The answers provided may be your quickest way to a diagnosis and treatment plan. The question can be phrased to reflect what you know about the patient's history. For example, you could ask: What pills did you take? What did the pills look like? What chemicals were you working with at your job? What drugs were you injecting? The core of this question is to get the patient's best description of which agent(s) and what context he was exposed. Recognize that poisoned patients may not be reliable historians because of alterations of consciousness or due to suicidality; additional history should be obtained from other sources, if possible.

Table 41.1 Toxicologic emergencies red flags

History	Concerning diagnosis
Suicide attempt	Multiple substances, co-ingestion, delayed action
Tricyclic antidepressant or sodium channel blocker ingestion	High morbidity and mortality
Beta-blocker or calcium channel blocker	High morbidity and mortality
Vomiting with altered LOC	Airway compromise, aspiration
Lithium, aspirin, theophylline, toxic alcohols	May require dialysis
Mushroom or acetaminophen ingestion	Delayed morbidity and mortality, liver problems, coagulopathy
Examination finding	**Concerning diagnoses**
Altered LOC	Airway compromise
Prolonged QRS or terminal R in aVR	Sodium channel blockade
Recurrent hypoglycemia	Sulfonylurea ingestion
Tachypnea, altered LOC	Salicylate toxicity
Miosis, bradypnea, depressed LOC	Opioid ingestion
Tachycardia, mydriasis, dry skin	Anticholinergic toxidrome
Tachycardia, mydriasis, moist skin	Sympathomimetic toxidrome
Bradycardia, miosis, SLUDGE	Cholinergic toxidrome

LOC: level of consciousness; SLUDGE: salivation, lacrimation, urination, defecation, gastric emptying

What time did the ingestion occur?

Timing of exposure is critical in order to determine expected signs and symptoms based on the natural history of the exposure. It also helps guide therapy, such as gastric decontamination. For agents that can be measured in the blood (e.g., APAP), time of ingestion is essential to interpret drug levels. The patient's response can be corroborated by family, friends, time last seen well, or other sources.

How and why did the exposure occur?

Patients may come into contact with toxic agents for a variety of reasons. The exposure may be a suicide attempt or gesture, a medication error, or an accidental, work-related, or environmental exposure. Understanding the reason for the exposure may significantly influence your plan of management, with the classic example being the suicidal patient for whom it is necessary to consider co-ingestants, as well as the need for psychiatric evaluation.

What symptoms have you had since the exposure?

Initially, open-ended questions can be used to find out the patient's symptoms, which can be followed up with specific questions looking for clinical features of likely exposures. In many cases, treatment of an exposure is based on the severity of the patient's symptoms.

Questions for the family, friends, emergency medical services and law enforcement

What drugs, pills, or chemicals did the patient ingest or was the patient exposed to? What time did the ingestion occur? How and why did the exposure occur?

These questions should always be asked to family, friends, EMS, or other witnesses to confirm the patient's history and to provide additional details or a different perspective. In patients with an altered level of consciousness, history of seizure, or concern about suicidality, this information is extremely important.

What other medications, agents, drugs, or chemicals were noted at the scene? Did you bring in any pill bottles or containers?

In the situation where the type of exposure, quantity of drug or other details are missing, having EMS or law enforcement bring pill bottles, chemical containers, material safety data sheets (MSDS), or other objects from the scene can provide needed information.

What other pills, drugs, agents, or herbals does the patient have access to (ie., in the home, from family members or friends)?

A complete list of agents that the patient takes or has access to is invaluable in narrowing your differential and interpreting diagnostic tests. Frequently, patients may provide inaccurate information regarding the types of medications or doses, and a list of possible agents allows the provider to look for signs, symptoms and laboratory findings that do not fit with the patient's reported exposure. Certain drugs such as lithium, digoxin, or warfarin, if available to the patient, may prompt additional laboratory testing. Many over-the-counter preparations are abused, as well as common products sold for other purposes.

What did the home or scene look like?

A description of the scene may provide clues to the cause of exposure, the patient's state of mind, or symptoms prior to the exposure.

How has the patient's condition changed over time? What signs or symptoms have you noticed since the exposure?

These two questions provide a picture of the patient's clinical course since ingestion, which may help with diagnosis, triage and assessment of the severity of exposure. They may also indicate possible required interventions.

Past medical

Additional sources of information include the patient's pharmacy and regional PCCs. Key items from the medical history include prior exposures, psychiatric illnesses, prior suicide attempts, social history (including drugs of abuse, occupational history), general medical problems and allergies. Questions related to dosing, signs and symptoms, or other features of the patient's clinical course may be indicated in certain overdose situations, and should be discussed with your regional PCC.

Physical examination

The physical examination may provide valuable clues to the diagnosis of an unknown ingestion, as well as determine severity of symptoms. The examination initially focuses on the ABCs (airway, breathing, circulation). Repeated assessment of the ABCs and neurologic status is essential to identify early changes and anticipate increasing toxicity.

General appearance

The poisoned patient should be examined for evidence of trauma, general level of consciousness, and ability to maintain adequate airway and breathing. Odors can provide valuable clues to possible exposures. Some classic examples of odors that may be found in poisoned patients include bitter almonds with cyanide, fruity smell of ketoacidosis from any cause, garlic from organophosphates, and rotten eggs from hydrogen sulfide.

Vital signs

Vital signs (including heart rate, blood pressure, respiratory rate, oxygen saturation, and temperature) may indicate the severity or stability of the poisoned patient at any point in time. In addition, specific constellations of vital signs can provide clues to the diagnosis (Table 41.2). However, it is important to remember that when the patient is in extremis, the expected constellation of vital signs for a particular poisoning may not be present. This is especially true in mixed ingestions, or ingestions in patients on certain medications or with underlying medical conditions.

Airway

Examination of the airway is the first step in determining the severity of a poisoning or overdose. Alteration in level of consciousness is a primary cause of airway compromise and is commonly seen in a variety of different exposures. Patients should be assessed for level of consciousness and the ability to protect their airway. The physical examination should also look for signs of upper airway obstruction, including sonorous breathing or loss of upper airway muscle tone. Because of the high frequency of both airway compromise and emesis in poisoned patients, aggressive airway management is often indicated early in the management of poisoned patients.

Eyes

The size of the pupils can provide additional diagnostic clues in the poisoned patient. Miosis (small pupils) can be caused by opioids, organophosphates, clonidine, phenothiazines and sedatives. Mydriasis (large or dilated pupils) can be caused by sympathomimetics, cocaine, many hallucinogens and anticholinergics. Horizontal nystagmus can be caused by lithium, ethanol, carbamazepine, phenytoin and other anticonvulsants. Phencyclidine (PCP) causes a vertical or rotatory nystagmus not typically seen with other ingestions.

Cardiovascular

Examination of the heart should be performed to search for bradycardia from cardiotoxic overdose or tachycardia from a variety of exposures. A new murmur in a patient with a history of intravenous (IV) drug use may represent endocarditis. Abnormal rhythms found on examination should be further defined with a 12-lead electrocardiogram (ECG). The peripheral pulses and skin should be examined for signs of shock, including mottling, delayed capillary refill, or weak or absent distal pulses.

Pulmonary

Assessment of respiratory system begins with inspection of respiratory rate, depth and effort. Auscultation of the lung fields should be performed to assess for symmetry and abnormal sounds. Drugs such as opioids, tricyclic

Table 41.2 Important toxicologic causes of abnormal vital signs

Bradycardia/hypotension	Beta-blockers, calcium channel blockers, clonidine, digoxin, organophosphates, ethanol, opioids, other sedatives
Tachycardia/hypertension	Sympathomimetics, cocaine, anticholinergics, theophylline, nicotine, thyroid hormone
Hyperthermia	Salicylates, anticholinergics, sympathomimetics, sedative withdrawal, neuroleptic malignant syndrome, serotonin syndrome
Tachypnea	Salicylates, metabolic acidosis, paraquat, chemical pneumonitis
Bradypnea	Sedatives, ethanol, opioids

antidepressants (TCAs) and cholinergic agents may produce pulmonary edema that can be auscultated during careful pulmonary examination. Wheezing or rales may also be heard in patients following aspiration of hydrocarbons or exposure to irritant toxic gases. Toxic agents producing a metabolic acidosis will cause a compensatory increase in respiratory rate and depth, which can be appreciated.

Neurologic

A complete neurologic examination looking for focal findings of the cranial nerves or extremities is extremely important to rule out structural brain injury in any patient with altered mental status. Toxicologic causes of altered mental status less frequently cause focal neurologic deficits. If focal neurologic findings are present, a structural lesion is more likely and imaging of the brain should be performed. Other important findings in poisoning may include muscle rigidity (serotonin syndrome, neuroleptic malignant syndrome or strychnine), tremors (lithium, sympathomimetics or sedative withdrawal), and fasciculations (organophosphate poisoning).

Skin

A careful skin examination can provide important diagnostic information. The patient should be undressed and the entire skin surface examined. Look for needle track marks, nail changes with heavy metal poisoning, spider or snake bites, trauma, or paint residue from "huffing." The oral cavity can provide clues if residual pill fragments, odors, burns from caustic exposures, or bite marks from seizure activity are identified. Very dry skin is seen with anticholinergic poisoning, whereas diaphoretic skin is seen with sympathomimetics, organophosphates and salicylates. Bullae or skin blisters may occur in some cases of barbiturate overdose.

Abdomen

A thorough abdominal examination is important for evaluation of other diagnoses in the differential and complications of caustic ingestions. In the poisoned patient, hyperactive bowel sounds can be heard with organophosphate poisoning. Diminished bowel sounds can help make the diagnosis of anticholinergic poisoning. Abdominal examination and bowel activity may also provide information related to treatment approaches and expectations related to toxin clearance.

Pelvic and rectal

If there is any suspicion of a foreign body or drug packets, a rectal and pelvic examination should be performed to search for these, as rupture may be lethal.

Belongings and clothing

The patient's personal belongings and clothing should be searched to identify potentially useful information. This might include medication bottles, loose pills, drug paraphernalia, pharmacy records, or other documentation. If a patient is altered or comatose, personal information and emergency contacts may be found.

Differential diagnosis

The correct diagnosis of a patient with a toxic exposure is most often learned from the history using directed questions and alternative sources. When the history does not provide a diagnosis, the patient's signs and symptoms help identify the possible exposure. Classic constellations of signs and symptoms linked with particular poisonings are called *toxidromes*. These can be very helpful in single overdoses with a classic clinical picture. In cases of multiple exposures, complications related to timing, or underlying illness, classic toxidromes may be of limited value. Four common toxidromes occur with cholinergic, anticholinergic, sympathomimetic and opioid agents (Table 41.3). A common mnemonic for the cholinergic toxidrome is SLUDGE: salivation, lacrimation, urination, defecation, gastric emptying. For anticholinergic exposures, ANTI-SLUDGE can be used (the opposite of cholinergic symptoms), but the mnemonic "blind as a bat, mad as a hatter, hot as a hare, red as a beet, and dry as a bone" is commonly used. The mnemonic SALT has been proposed as a tool to assist in the recognition and treatment of toxicity from sodium channel blocking agents (Table 41.4). TCAs are a commonly recognized sodium channel blocker, but a number of

Table 41.3 Common toxidromes

Toxidrome	Pupils	Skin/HEENT	Cardiovascular	Pulmonary	CNS	GI/GU
Cholinergic	Miosis	Diaphoresis Salivation Lacrimation	Bradycardia	Increased secretions	Coma Seizures	Emesis Urination Diarrhea
Anticholinergic	Mydriasis	Dry skin	Tachycardia	Variable	Delirium	Decreased motility Urinary retention
Sympathomimetic	Mydriasis	Diaphoresis	Tachycardia Hypertension	Variable	Agitation Seizures	Variable
Opioid	Miosis	Variable	Bradycardia Hypotension	Hypoventilation	Hypnosis Coma	Decreased motility

CNS: central nervous system; HEENT: head, eyes, ears, nose, throat; GI: gastrointestinal; GU: genitourinary.

other agents have similar effects: cocaine, diphenhydramine, propranolol and benzonatate. Other important poisonings to recognize that have identifiable clinical findings are salicylates, nonsteroidal antiinflammatory drugs (NSAIDs), digoxin, beta and calcium channel blockers, lithium, and hypoglycemic agents.

Table 41.4 SALT mnemonic for the diagnosis of sodium channel blockade

S	Shock
A	Altered level of consciousness
L	Long QRS
T	Terminal R in lead aVR

Diagnostic testing

Although laboratory testing can be helpful in diagnosing, defining and treating acute toxic exposures, the most important therapeutic decisions are those regarding airway management, circulatory support and decontamination, which do not depend on ancillary testing. Treatment of critically ill patients should proceed based on the clinician's best judgment, and should not be delayed while awaiting the results of ancillary testing.

Electrocardiogram

The ECG may be helpful in both diagnosis and prediction of toxicity in acute overdose. A 12-lead ECG is recommended in any cardiotoxic overdose, including digoxin, other antidysrhythmics, phenothiazines, beta-blockers, calcium channel blockers, cocaine and other sympathomimetics, and TCAs or other sodium channel blockers. It can be helpful in an unknown overdose to look for cardiotoxic agents.

Sodium channel blocking agents, including TCAs, have characteristic ECG changes that help with diagnosis and suggest an increased risk of complications. In TCA overdose, the ECG commonly shows a sinus tachycardia from anticholinergic effects, which alone does not predict serious toxicity. ECG findings of a QRS interval greater than 100 milliseconds or a large terminal R wave in aVR in a TCA-poisoned patient predict an increased risk of both seizures and ventricular tachycardia (Figures 41.1 and 41.2).

Radiologic studies

There are no routine radiologic studies for patients with toxic ingestions or exposures. A KUB (kidneys, ureters, bladder) film can be helpful in visualizing radiopaque drugs, drug packets, and other foreign bodies. "Body stuffers" ingest poorly wrapped single packets of drugs to avoid detection; these are rarely seen on plain radiographs. "Body packers" (also known as "mules") ingest large numbers of well-wrapped drug packets as a technique to transport drugs without detection; these large numbers of packets are usually seen on a KUB. Other agents can be seen occasionally depending on quantity, concentration, and time of ingestion. These potentially visible agents can be remembered by the mnemonic CHIPES (Table 41.5). A KUB can be helpful in iron ingestions, both for initial detection of the ingestion as well as following the progress of gastrointestinal (GI) decontamination. Chest radiographs are indicated in patients with potential aspiration of hydrocarbons or other agents that can cause chemical pneumonitis.

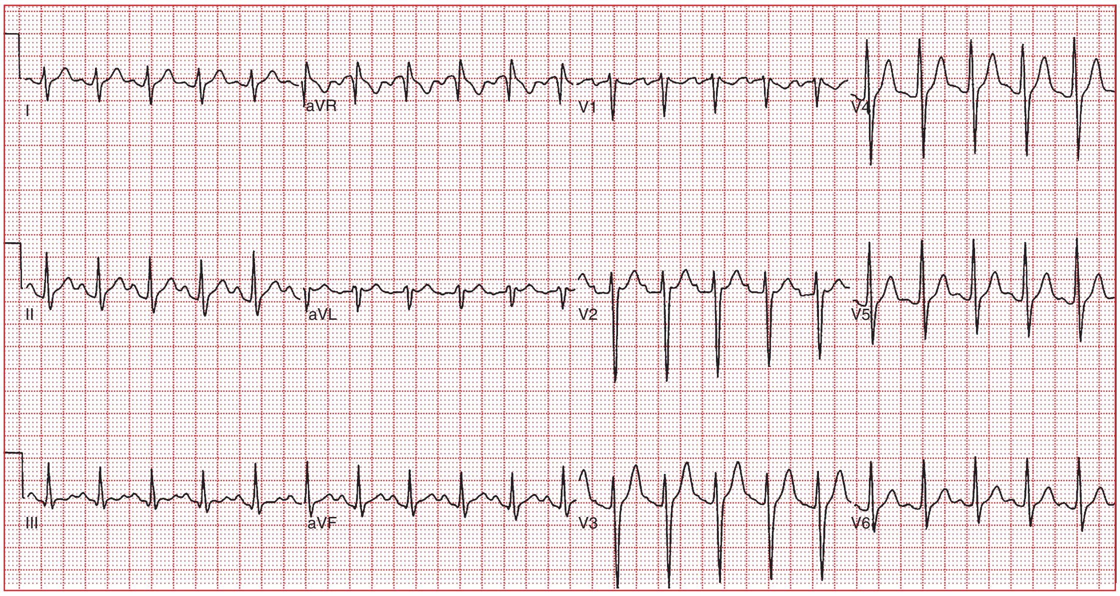

Figure 41.1
Electrocardiogram of tricyclic antidepressant–poisoned patient showing tachycardia, a widened QRS, a rightward QRS axis, and a large terminal R wave in aVR. Courtesy: Amal Mattu, MD.

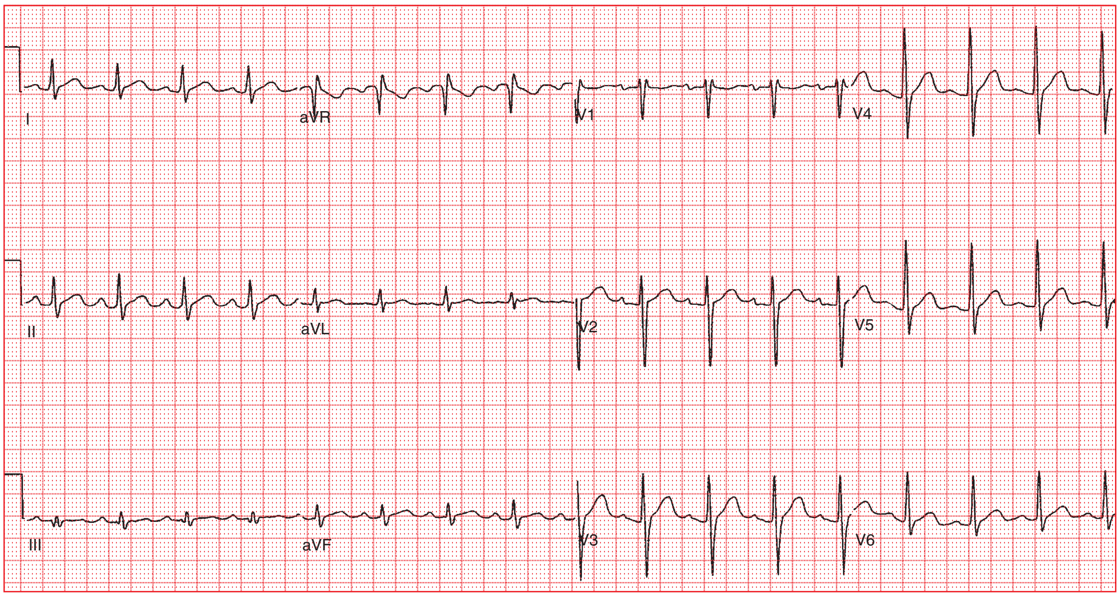

Figure 41.2
Electrocardiogram of the same patient following administration of three ampules of sodium bicarbonate showing resolution of abnormalities. Courtesy: Amal Mattu, MD.

Table 41.5 CHIPES mnemonic for potentially visible agents on plain radiograph

C	Chloral hydrate, calcium carbonate
H	Heavy metals
I	Iron, iodinated compounds
P	Psychotropics, potassium preparations, packets of drugs
ES	Enteric-coated and slow-release formulations

Laboratory tests

The first laboratory test performed in any patient with altered mental status should be a bedside glucose. Additional testing in almost all ingestions should include a urine pregnancy test in females of childbearing age and blood chemistries. The electrolytes, blood urea nitrogen (BUN), creatinine and glucose can be used to search for metabolic acidosis, renal insufficiency, and significant sodium or potassium abnormalities. All of these potentially impact the management of a poisoned patient. In a patient with a metabolic acidosis, the electrolytes are used to calculate the anion gap and help to narrow the differential diagnosis.

Anion gap = Na – (Cl + bicarbonate)

Normal range is 8–12 meq/L.

The differential diagnosis of an anion gap metabolic acidosis can be remembered using the mnemonic MUDPILECATS (Table 41.6).

Table 41.6 Differential diagnosis of an anion gap metabolic acidosis using the MUDPILECATS mnemonic

M	Methanol, metformin
U	Uremia
D	Diabetic ketoacidosis
P	Paraldehyde, phenformin
I	Iron, isoniazid
L	Lactic acidosis
E	Ethylene glycol
C	Cyanide, carbon monoxide
A	Alcoholic ketoacidosis
T	Toluene
S	Salicylates, seizure

The calculated osmolal gap can narrow the differential diagnosis in a patient with a metabolic acidosis. An elevated osmolal gap is suggestive of poisoning with methanol, ethylene glycol, mannitol or isopropanol. The test's utility is limited by its inability to identify the specific cause of the increased gap, and because a normal osmolal gap does NOT rule out poisoning with any of these agents, as individuals have a wide range of normal values.

Osmolal gap = Measured osmoles^a – (2 × Na)
+ (Glucose/18)
+ (BUN/2.8)
+ (EtOH/4.6)

^{a}Freezing point depression technique.

Na: sodium; BUN: blood urea nitrogen; EtOH: ethanol. Normal osmolal gap range is < 10 mOsm.

APAP is a common over-the-counter medication contained in a variety of combination products. Patients may refer to it as "aspirin" or "pain killers." Because APAP toxicity is initially clinically silent, treatment must be started before the patient manifests signs of hepatic toxicity (Figure 41.3). Therefore, measurement of serum

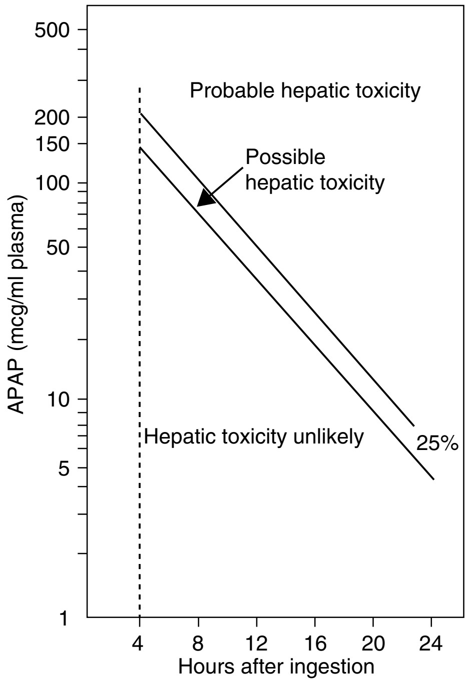

Figure 41.3
Acetaminophen toxicity nomogram. The Rumack-Matthew nomogram, relating expected severity of liver toxicity to serum acetaminophen concentrations. Reprinted with permission and adapted from Smilkstein MJ, Bronstein AC, Linden C, et al. Acetaminophen overdose: A 48-hour intravenous *N*-acetylcysteine treatment protocol. *Ann Emerg Med* 1991;20(10):1058.

APAP levels is indicated in any suspected overdose or unknown exposure.

Controversy exists regarding the ordering of routine salicylate levels in overdose patients. Salicylate toxicity typically produces recognizable signs and symptoms in serious overdose, including tachypnea, tinnitus and altered mental status, so screening levels may not routinely be indicated. However, because this is a common, potentially life-threatening ingestion with subtle early signs of toxicity, routine levels may be appropriate in selected situations. These include early signs of altered mentation, tachypnea, or suspected multiple drug overdose.

Quantitative levels of specific drugs such as carbamazepine, lithium, phenobarbital, carbon monoxide and digoxin are indicated in patients when there is a clinical suspicion of exposure or overdose based on the history or exam. They should not be ordered routinely as screening tests.

Qualitative urine drug screens, often referred to as “tox screens,” test for the presence of multiple drugs or their metabolites in urine. These tests most commonly identify drugs of abuse, such as opioids, benzodiazepines, cocaine, amphetamines, barbiturates, marijuana and PCP. Qualitative tests will be positive with “therapeutic” levels or significant ingestions. The amount of drug present cannot be inferred from the result of qualitative screens; these tests are useful only for identifying the presence or absence of this limited group of drugs. In several studies, these tests had limited impact on the management of the unknown overdose and are not routinely recommended in overdose patients. Recognizing these limitations, drug screens can determine which drugs a patient has been exposed to, and may be helpful in creating an accurate differential diagnosis or arranging for appropriate psychiatric follow-up or substance abuse counseling.

General treatment principles

There are four main components of treatment for the poisoned patient: supportive care, antidotes, gastric (and other) decontamination, and enhanced elimination. These can be remembered using the mnemonic SAGE.

S	Supportive care: ABCs and the “coma cocktail”
A	Antidotes: specific therapy for certain exposures
G	Gastric (and other) decontamination: removal from stomach, skin, eyes
E	Enhanced elimination: includes dialysis, urinary excretion, hemofiltration

Supportive care

The majority of patients with toxic exposures will survive with supportive care alone, which includes airway management, supplemental oxygen and ventilation, and normalization of blood pressure using IV crystalloid fluids and inotropes or vasopressor agents (if necessary). Every overdose patient should be monitored initially with continuous cardiac, noninvasive blood pressure and pulse oximetry monitoring. IV access should be established in all patients. Patients with unstable vital signs or potentially serious ingestions require two large-bore IV lines. Patients can deteriorate rapidly, and early airway management with rapid sequence intubation may be lifesaving.

During the initial evaluation of any patient with altered mental status, consideration should be given to using the “coma cocktail.” The coma cocktail typically consists of naloxone to reverse opioid toxicity, a fingerstick glucose and supplemental dextrose if hypoglycemia is found, and thiamine to prevent or treat Wernicke’s encephalopathy. Historically, the coma cocktail included empiric treatment with D50 (dextrose) and physostigmine (Antilirium), although neither of these practices is currently recommended. In patients with a reversible overdose such as opioid intoxication from heroin, the patient should be oxygenated and ventilated with a bag-mask device until naloxone can be administered and take effect. The mnemonic DON’T highlights these four elements of the coma cocktail.

D	Dextrose
O	Oxygen
N	Naloxone
T	Thiamine

Naloxone is a specific opioid antagonist that lasts 60–90 minutes. When given IV, intramuscular (IM), subcutaneous (SQ), intranasally, by nebulizer or via an endotracheal tube, it will result in a rapid return to consciousness and spontaneous ventilation in the opioid-poisoned patient. The initial dose of naloxone ranges from 0.1 mg for a chronic opioid abuser to 0.4 mg for an acute overdose.

Naloxone should be titrated so that spontaneous ventilation is adequate and the individual is alert enough to provide a medical history. The dose may be increased up to 2–10 mg in patients who fail to respond to the initial dose if the clinical suspicion of opioid intoxication remains high. The only significant side effect from naloxone in adults is acute opioid withdrawal, which is non–life-threatening but may cause discomfort for the patient.

There are no good data to support the use of thiamine in all patients with altered mental status, and it does not need to be given before glucose. Treatment of hypoglycemia takes priority. Risk factors for thiamine deficiency include malnutrition, alcoholism, extreme diets, or a history of previous deficiency. Such patients should be given thiamine during the first 12 hours or immediately if they have altered mental status. The typical adult dose of thiamine is 100 mg IV or IM.

Flumazenil, a reversal agent for benzodiazepines, is not part of this coma cocktail. It can cause seizures in patients with some ingestions, including TCAs, cocaine and sympathomimetics, and can cause acute withdrawal in patients who are tolerant to benzodiazepines. In addition, benzodiazepine overdose can be safely managed with supportive care. Benzodiazepines are protective against seizures, so reversing the protective effect is not advised for mixed overdoses.

Antidotes

Antidotes are an exciting part of toxicology, despite being of limited utility in most cases. It is important to be aware of the toxins that respond to antidote therapy, so that appropriate consideration can be given to this treatment modality. A list of important toxins and their antidotes is provided (Table 41.7).

Table 41.7 Important toxic substances and antidotes

Toxic substance	Antidote
Acetaminophen	*N*-acetylcysteine
Beta-blockers	Glucagon
Calcium channel blockers	Calcium, glucagon, insulin/dextrose
Carbon monoxide	Oxygen, hyperbaric oxygen
Cyanide	Cyanide kit *or* hydroxycobalamin hydrochloride
Digoxin	Digibind Fab-fragments
Hydrofluoric acid	Calcium
Iron	Deferoxamine
Isoniazid	Pyridoxine
Local anesthetics	Intravenous lipid emulsion
Methanol/ethylene glycol	Ethanol or fomepizole
Methemoglobinemia	Methylene blue
Opioids	Naloxone
Organophosphates	Atropine and pralidoxime
Snake bite (Crotalidae)	Crotalidae polyvalent immune Fab
Sulfonylureas (oral hypoglycemics)	Glucose, octreotide
Tricyclic antidepressants and other sodium channel blockers	Sodium bicarbonate and hypertonic saline

Gastric and other decontamination

Decontamination of the GI system (after an oral exposure), or the skin and eyes (after a dermal exposure) is critical to reduce the amount of absorbed drug and reduce potential toxicity. All health care providers should use appropriate protective gear when indicated in order to protect against possible exposure.

Skin decontamination begins with removal of all the patient's clothing. Any dry powder or debris should be brushed off first, as some chemicals produce an exothermic reaction when mixed with water. The skin is then washed with warm, soapy water until no evidence of contamination remains. In an ocular exposure, the eyes should be irrigated with generous amounts of normal saline or tap (shower) water until symptoms are relieved and pH returns to normal (in cases of chemical exposure).

There are four basic techniques for decontamination of the GI system: induced emesis with syrup of ipecac, gastric lavage, activated charcoal, and whole bowel irrigation. A significant amount of ingested toxic material may remain in the GI tract even with optimal application of these techniques. Given their limited effectiveness, these gastric decontamination techniques have been utilized less frequently in recent years for acute overdose.

Syrup of ipecac induces vomiting through local irritation of the stomach and activation of the chemotactic trigger zone in the central nervous system. Ninety percent of patients begin to vomit within 30 minutes, and symptoms usually resolve by 2 hours. Due to a lack of evidence to support its effectiveness, and the potential risk of aspiration from persistent vomiting in a seizing or comatose patient, syrup of ipecac is no longer recommended for use in health care facilities. Based on recent AAPCC data, there are rare current indications for ipecac use, and its use at home has been discouraged by the American Academy of Pediatrics.

Gastric lavage involves placing a large plastic gastric tube (36–40 French in adults) through the mouth into the stomach. The stomach is emptied and lavaged with warm normal saline until the lavage effluent is clear of pill fragments and stomach contents. Activated charcoal is then placed down the tube into the stomach and the lavage tube is removed. Significant risks from this procedure include aspiration, esophageal perforation, and hypoxia. It is contraindicated following caustic or hydrocarbon ingestion, minor ingestions, or in patients who are unable to protect their own airway. Evidence suggesting a benefit from this procedure is limited. In an optimal scenario of small pill fragments, ingestion within an hour, and aggressive lavage, the best mean recoveries have been from 36–50% of ingested material. Balancing these significant risks with only minimal benefits, gastric lavage is usually recommended only in patients who present within 1 hour of life-threatening ingestions and have had their airway protected by endotracheal intubation. Even in highly selected patients, there is no evidence that it improves outcomes.

Activated charcoal has become the mainstay of GI decontamination in recent years. It is highly effective for the treatment of a variety of ingestions, and has a very good safety profile. Activated charcoal works by adsorbing the toxin, thereby making it less available for absorption from the gut into the bloodstream. It is most effective if given within 1 hour of ingestion, although it may be used in patients presenting up to 24 hours after ingestion if they remain symptomatic or have potentially delayed absorption from decreased intestinal motility, delayed-release products, or concretions. Activated charcoal is typically given in a dose of 1 g/kg, although larger doses are occasionally indicated. Patients can drink the activated charcoal through a straw, or it can be given via nasogastric (NG) or lavage tube. The first dose of charcoal is often given with sorbitol or magnesium citrate as a cathartic to reduce GI transit time. Multiple doses of activated charcoal are indicated for certain ingestions, and may be done in consultation with a PCC. Contraindications to activated charcoal include an inability to protect the airway; ingestion of caustic, hydrocarbons, or other agents not bound to charcoal; or planned endoscopy.

Whole bowel irrigation (WBI) involves placing an NG tube or having the patient drink a balanced solution of polyethylene glycol and electrolytes at a rate of 1–2 L/hour. The goal is to mechanically flush the GI tract, reducing the absorption of certain ingested agents. Treatment should be continued until the patient has clear rectal effluent and no further evidence of progressive toxicity. It can cause vomiting, diarrhea and abdominal pain, and should not be used in patients with intestinal obstruction or absent bowel sounds. WBI may be beneficial for decontamination in selected cases of drug bezoars, iron ingestions, sustained-release preparations of drugs such as calcium channel blockers, and ingested drug packets.

Enhanced elimination

There are two primary methods of enhancing the elimination of a toxin from the body: urinary alkalinization and hemodialysis. These techniques are useful only for a limited number of substances, and should be guided by advice from a PCC.

Urinary alkalinization works by "trapping" agents that are weak acids in the renal tubules and increasing their excretion in the urine. The urine can be alkalinized by administration of 1 meq/L of IV sodium bicarbonate. The patient is given sodium bicarbonate IV bolus followed by a continuous infusion. The goal is a urine pH of 7.5–8.0. A normal serum pH should be maintained during this process. The administration of adequate potassium may be necessary to prevent paradoxical aciduria from occurring due to hypokalemia. Urinary alkalinization may be indicated for symptomatic ingestions of salicylates, chlorpropamide and phenobarbital. Complications include volume overload, hypokalemia, and changes in serum pH.

Hemodialysis is effective at removing small, water-soluble substances with low protein binding and small V_d. Commonly dialyzable toxins include salicylates, theophylline, barbiturates, lithium, methanol, isopropanol and ethylene glycol. This invasive procedure requires placement of a large central venous access device. Hemodialysis can be lifesaving in a number of these overdoses, and should be considered with the guidance of a PCC and consulting nephrologist. The role of newer continuous arterial or venous filtration techniques in the treatment of toxic exposures has not been clearly defined.

Special patients

Pediatric

The approach to pediatric poisonings is similar to that in adults. Children make up a significant proportion of toxic exposures in the United States. In general, supportive care and gastric decontamination with activated charcoal are critical factors in treatment. Small doses (a single pill or taste) of TCAs, cardiotoxic agents, Lomotil, oral hypoglycemics, methylsalicylate, camphor, Visine, and methanol can be fatal in children. Children with serious or potentially serious ingestions should be managed in a pediatric intensive care unit (PICU) setting. For accidental exposures, parents and caregivers should be educated to prevent future incidents. Cases of suspected abuse or neglect should trigger mandatory referral to child protective services or police authorities.

Elderly

Geriatric patients are at high risk for complications of toxic exposure due to underlying cardiovascular, pulmonary, renal, hepatic and other systemic diseases. Patients on multiple medications are also at risk for adverse reactions from dosing errors, incorrect medications, or medication interactions. Medication complications such as chronic salicylism should be considered in geriatric patients presenting with hypotension, bradycardia, altered mental status, weakness, nausea or vomiting, and acidosis. Depression is also common in elderly patients; appropriate screening should occur if there is a suspicion of intentional overdose.

Immune compromised

Patients with human immunodeficiency virus (HIV), acquired immune deficiency syndrome (AIDS), or malignancy may be on a large number of toxic medications with a narrow therapeutic window. They should be carefully screened for medication-related causes of any presenting complaint. The patient's primary care provider or specialist, as well as the PCC, can provide guidance on the clinical presentation and treatment of toxicity from uncommon medications. An appropriate psychiatric history should be taken to look for signs or symptoms of depression and suicidal ideation.

Pregnancy

In general, appropriate treatment of the mother for her overdose will also benefit the fetus. Lifesaving therapy should not be withheld from the mother because of concerns for fetal toxicity. Depression is also common in this group, and pregnant women should be screened if an intentional overdose is suspected. CO poisoning, APAP and iron ingestions are examples of serious exposures for which the antidote given to the mother is safe and protects the fetus from toxicity. PCCs should be consulted as soon as possible for further instruction in such cases.

Disposition

Asymptomatic patients with potentially serious exposures should have appropriate diagnostic studies and be watched for at least 6 hours. In most cases, if they remain asymptomatic after 6 hours, they can be safely discharged. Every patient presenting following a suicidal gesture or attempt should receive an appropriate evaluation by a mental health professional. These patients require hospitalization for either their medical or psychiatric condition.

Certain medications with delayed onset of symptoms or prolonged clinical toxicity may require 12–24 hours of observation in either an emergency department (ED) or observation unit. Examples of such agents include methadone, Lomotil, oral hypoglycemics, lithium, sustained-release calcium channel blockers, and theophylline. In every case of toxic exposure, it is important to be familiar with the time course of expected symptoms.

Patients who have more than minor symptoms, develop toxicity during the 6-hour observation period, or who have evidence of toxicity on diagnostic testing should be admitted to the appropriate hospital setting.

PCCs are the single best source for information about diagnosis, management and disposition of patients with toxic exposures. They can provide consultation with a toxicologist, follow-up for patients discharged home, contact information and locations of medical centers that may accept patient transfers with special therapeutic needs, and assist with monitoring hospitalized patients.

Pearls, pitfalls and myths

- Poisoning is common in the United States. The most common serious exposures are from analgesics, antidepressants, cardiovascular drugs, CO and drugs of abuse.
- History from the patient may not be reliable and should be confirmed through different sources.
- The physical examination may lead to a diagnosis through classic signs and symptoms of common poisonings known as *toxidromes*.
- A fingerstick glucose and oxygen saturation should be checked, in addition to a complete set of vital signs, in every patient presenting to the ED following an ingestion with altered mental status. Consideration should be given to the immediate administration of naloxone and thiamine.
- Antidotes, although occasionally helpful, are secondary to general supportive care in the management of most toxic exposures.
- Gastric lavage is rarely performed, except on intubated and critically ill patients. There is essentially no role for syrup of ipecac in the hospital or home setting. The mainstay of gastric decontamination is activated charcoal.
- Pediatric patients are at high risk for accidental exposures, and may have serious side effects or death even from small doses or single tablets of certain medications or chemicals.
- Geriatric and immunocompromised patients are at high risk for drug toxicity from dosing errors, incorrect medications, medication interactions, renal or liver dysfunction, or dehydration. Intentional ingestions (i.e, suicide attempt or elder abuse) should be considered in the geriatric population.
- Asymptomatic patients should be observed in the ED for at least 6 hours. Patients with exposures to long-acting agents or agents with a delayed onset of action may need 12–24 hours observation.
- The PCC is the best source of information for the diagnosis and management of toxic exposures, and should be consulted on essentially every case presenting to a health care facility.

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42 Urinary-related complaints

Fred A. Severyn, MD

Scope of the problem

Urinary-related complaints are found in many patients presenting to the emergency department (ED). The variety of complaints can be staggering, overshadowed only by their cost to the health care system. Careful evaluation may uncover undiagnosed congenital abnormalities threatening future renal function, serious infections, or disease complications. Identification of urosepsis allows prompt treatment to prevent subsequent morbidity and mortality.

This chapter focuses on dysuria, hematuria, nephrolithiasis, urinary tract infection (UTI) and acute urinary retention (AUR). These complaints account for billions of health care dollars, and several million ED visits annually. It is therefore important for clinicians to understand urinary anatomy, and approaches to evaluation and treatment of these conditions.

Anatomic essentials

Urologic anatomy is essentially identical from renal unit to bladder in both sexes (Figure 42.1). It differs from bladder to urethral meatus in obvious ways. The renal unit and the gonads have a similar embryologic origin, so pain in one location is often referred to the other. The kidneys are retroperitoneal organs, relatively protected by the inferior ribs posteriorly.

After formation of urine in the glomerular unit, urine enters the renal calyces that merge to form the renal pelvis. This renal pelvis cones down to form the ureter. The ureter travels caudally and arises out of the posterior pelvic brim as it crosses over the iliac vessels, then inserts into the bladder through a small narrowed intramural portion. It is in these anatomic points of narrowing that renal calculi potentially lodge. A significant percentage of ureteral obstruction cases cause pain from distention and dilation of the upper ureter and renal pelvis (hydronephrosis).

Normal micturition is a complicated neuromotor process, with a control center located in the spinal cord at the S2–S4 level. Autonomic control of various muscle groups allows for coordinated voiding via a balance between parasympathetic and sympathetic innervation. Any interruption of this sacral reflex arc from a lower motor neuron lesion will cause a loss of the sensation of bladder fullness. This results in an atonic bladder that fails to empty. This may occur via traumatic mechanisms, as in lumbar vertebral fractures or disc herniation, or from atraumatic mechanisms, such as epidural abscess or tumor metastasis. Unfortunately, up to 5–10% of carcinomas (especially cancers of the breast, lung, prostate and kidney) show predilection for epidural metastasis. Upper motor neuron lesions above T11 may result in a spastic bladder that fails to allow adequate urine storage.

Normal urinary flow through the bladder with its subsequent bladder washout is the primary host defense mechanism against infection. Bacteria from the perineum can migrate up the urethra. Hence, the shorter female urethra predisposes females to more frequent UTIs, especially in infants, children, and sexually active women. Predominance of UTIs shifts towards males as they get older, when incomplete emptying of the bladder due to prostatic enlargement becomes problematic. Some authors comment on a "milking" action that occurs to the female urethra during sexual intercourse. This has led to the observation that prompt voiding following intercourse reduces the likelihood of UTI. Certain bacteria, however, have evolved efficient mechanisms to attach to the bladder's epithelial and mucosal surfaces, bypassing normal washout defense mechanisms.

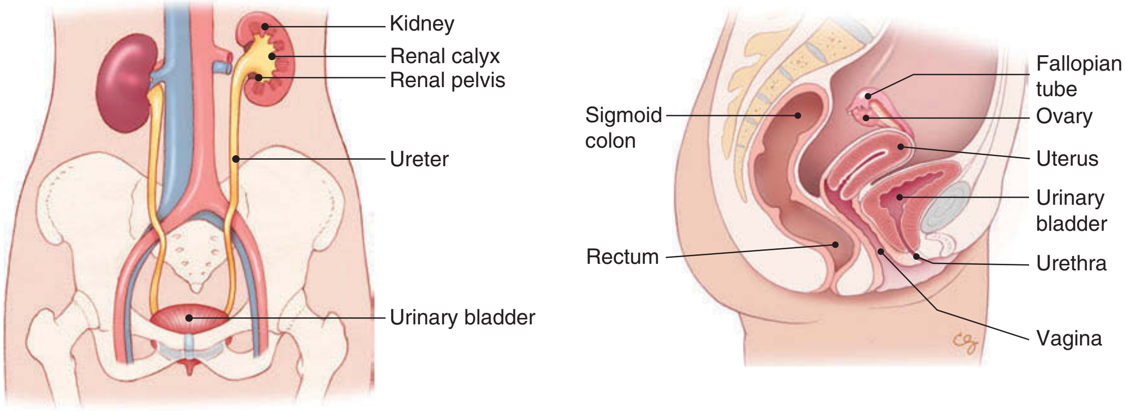

Figure 42.1
Female urinary tract anatomy: (a) anteroposterior (AP) view (b) sagittal view. © Chris Gralapp.

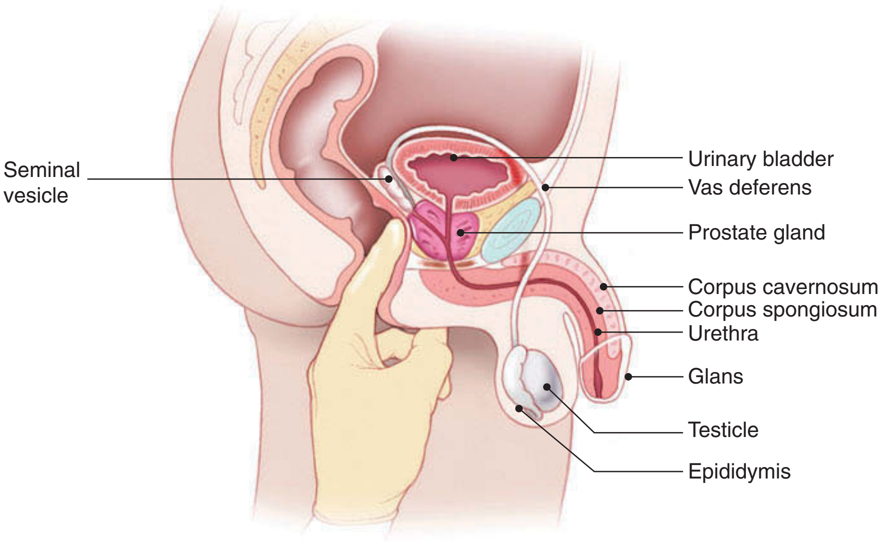

Figure 42.2
Male urinary tract anatomy: sagittal view. © Chris Gralapp.

From bladder to meatus, the anatomy differs greatly between genders. The female anatomy includes a short urethral distance to the actual meatus. However, the nearby vagina, cervix, uterus and parametrial structures complicate disease processes as well as evaluation.

The male urethra exits the bladder and is enveloped by the prostate gland (Figure 42.2). The male urethra is longer anatomically than the female urethra, and as such is more prone to strictures over its caliber and length. These may occur from prior instrumentation (indwelling catheter) or trauma, or as a sequelae of urethritis. The striated muscle in the urogenital diaphragm distal to the prostate forms the external sphincter. Benign prostatic hypertrophy or hyperplasia (BPH) is extremely common in older males; autopsy studies demonstrate that as many as 60% of men over 50 years of age have some element of BPH.

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 42.1).

History

The history in a patient with urinary complaints is extremely important, as specific external findings associated with causes of urinary complaints generally do not exist. A careful history also helps identify clues that may guide clinical investigation. One needs to determine whether the symptoms involve the urinary or reproductive tract, and investigate accordingly.

How did your symptoms begin?

A sudden change in normal voiding pattern usually brings patients to a physician. The onset of symptoms (acute or insidious), time of day when maximal, and alleviating or irritating factors should be investigated. Sudden onset of pain is often characteristic of acute obstruction. Progressive symptoms, such as dribbling and increased nocturia, frequently accompany enlargement of the prostate (e.g., BPH) that develops over time.

Can you describe the pain?

Many urologic presentations have some component of associated pain. This may be irritative pain during micturition, lower abdominal pain in the suprapubic region or perineum associated with the bladder or prostate, or flank pain associated with ureteral or renal disease. Renal colic is typically abrupt in onset, although some irritative symptoms may be noted preceding its onset. It may radiate from the flank or back towards the groin, testicles, or ovaries.

Are you unable to spontaneously void?

Inability to void is typically an acute presentation, and patients present with urgent need for relief. It is key to inquire about obstructive symptoms, such as slowly worsening hesitancy, decrease in stream force or caliber, and post-void dribbling. These symptoms suggest a slowly progressive obstruction with an acute end point, as opposed to an acute process with a different etiology. With acute presentations, think of infection, clot retention, medication, or toxicologic causes. Medications that may cause urinary retention or an inability to void completely include anticholinergic agents, sympathomimetic agents (often over-the-counter), and narcotic agents, as

Table 42.1 Urinary-related complaints red flags

History	Concerning diagnosis
Elderly flank pain	Ruptured AAA, pyelonephritis, perinephric abscess, renal vein thrombosis, renal malignancy
Febrile flank pain	Pyelonephritis, infected kidney stone, renal abscess
Syncope	Ruptured AAA, urosepsis, hematuria with anemia, acute painful condition related to the urinary tract
Crying child	UTI, penile tourniquet
Painless gross hematuria	GU malignancy
Fever, chills and dysuria	Pyelonephritis, complicated UTI
Bladder dysfunction (retention or incontinence) and back pain	Cauda equina syndrome
Inability to urinate or urinary incontinence	Acute urinary retention, stricture, obstruction, neurogenic bladder, malignancy, GU infection, phimosis/paraphimosis
Examination finding	**Concerning diagnosis**
Costovertebral angle tenderness	Pyelonephritis, nephrolithiasis, renal abscess, renal infarction or thrombosis
Perineal erythema, subcutaneous emphysema, fever	Fournier's gangrene
Prostatic tenderness	Prostatitis
Urethral erythema, discharge, lesions	Urethritis, herpes simplex or other sexually transmitted infections
Abnormal testicular lie	Testicular torsion
Saddle anesthesia, urinary retention	Cauda equina syndrome or spinal cord compression
Hypotension and flank pain	Vascular catastrophe (such as AAA), pyelonephritis with possible sepsis
Pneumaturia	Colovesical fistula (often due to malignancy)

AAA: abdominal aortic aneurysm; GU: genitourinary; UTI: urinary tract infection.

these affect detrusor contractility or smooth muscle tone. Bladder over-distention is often superimposed on chronic obstructive symptoms, and can occur with fluid challenges such as those occurring with alcohol ingestion and subsequent diuresis.

Is your urinary elimination normal?

Micturition-associated symptoms include frequency, urgency, dysuria, post-void fullness, incontinence and suprapubic pain. Careful questioning of the patient regarding the timing of these symptoms may localize the inflammatory process. Do the symptoms predominate before voiding, at the initiation of the voiding stream, during the mid-stream phase, or after voiding? These questions can help differentiate bladder from urethral symptoms. The presence of gas bubbles in association with the urinary stream (pneumaturia) can help identify an abnormal communication between the gastrointestinal (GI) and genitourinary (GU) tracts, often associated with colonic diverticula (colovesical fistula).

Is the urine discolored?

Not all discolored urine is secondary to hematuria. Gross hematuria and clot formation are easily identified, but abnormally pigmented urine may be from blood, abnormal chromogens (such as bile), nutritional sources, and dyes themselves. Cloudy urine usually occurs from protein or crystals, not from cells. One should also ask about the timing of hematuria in relation to the urinary stream. Bleeding from the anterior urethra to the trigone, the bladder, or the proximal structures can present with initial, terminal, or total stream hematuria, respectively. Purplish urine in a catheter bag has also been associated with bacteruria.

Are you having other abnormal symptoms associated with either elimination or the perineum?

Abnormalities of the micturition reflex arc tend to have other neurologic manifestations, such as bowel dysfunction with incontinence or retention. A patient may also complain of anesthesia in a saddle distribution if asked. A careful sexual history may be helpful, for the same autonomic innervation is important for both male and female sexual function. Abnormal pain, anesthesia, or inability to achieve an erection (impotence) may identify neurologic pathology.

Have you had previous genitourinary problems?

Many urologic conditions are chronic in nature, and patients may be able to relate symptoms to prior disease presentations. A history of kidney stones, urologic malignancies, prior surgical instrumentation of the urinary tract, or prior infections can help clinicians narrow the differential diagnosis. Abrupt flank pain in an elderly, hypertensive patient without a history of kidney stones could be related to nephrolithiasis, but acute aortic disease is a more concerning diagnostic consideration. Prior instrumentation of the urethra from indwelling catheters may lead to urethral strictures. A careful sexual history can also identify risk factors for sexually transmitted infections (STIs). Patients should be asked about a history of abnormal discharge, genital pain or lesions, and unprotected sexual activity with multiple partners. Colicky lower abdominal pain or acute urinary retention have been associated with ectopic pregnancy, so a detailed history and pregnancy testing are important in children and women of childbearing age.

Are you taking prescription or over-the-counter medications?

Recent antibiotic use raises concern for antibiotic resistance if an infection is present. Many medications have anticholinergic side effects that can cause bladder outlet obstruction and AUR. A common example is over-the-counter antihistamines used for a variety of unrelated URI symptoms. Other medications or foods can cause urinary discoloration, and at times burning or foul odor.

Associated symptoms

A history of fever or chills may serve as an indirect marker of infection, especially in an afebrile patient who may have taken nonsteroidal antiinflammatory drugs (NSAIDs) or acetaminophen for pain relief recently. Ureterolithiasis may cause intermittent pallor, nausea, diaphoresis and vomiting. Flank pain with shoulder radiation usually reflects a subdiaphragmatic process, such as hemorrhage or abscess. One should ask about cardiopulmonary symptoms such as cough, dyspnea and chest pain that may be referred to either flank. Renal disease may stimulate the celiac ganglion that serves the kidneys and stomach, producing nausea and vomiting. The inability to keep down oral fluids or medications impacts patient disposition, as such patients may require hospitalization or additional observation. Diarrhea in a female patient can colonize the urethra and bladder with coliform bacteria, causing a UTI. Gynecologic conditions such as menses, vaginal discharge and/or bleeding, abnormal pelvic pain, and contraception practices can help establish a differential diagnosis.

Physical examination

A focused physical examination may help differentiate upper tract disease from lower tract disease, but may also be normal. Clinicians must evaluate the entire patient, starting with the general appearance.

General appearance

The general appearance of a patient with urinary complaints may vary. Patients may present with minimal discomfort or severe pain; they may also have no pain whatsoever. Patients may appear pale, diaphoretic, cool and/or clammy. They may be actively vomiting and writhing in pain, or be very comfortable reading a magazine. Hydration status of the patient can also be gleaned at this time.

Renal colic tends to cause patients to writhe about, restless on the examination table, with neither relief nor exacerbation upon movement. Peritonitis, on the other hand, is classically much worse with patient movement; most patients with peritoneal pathology lie still to avoid exacerbating their pain.

Vital signs

Vital signs are a key component of every patient encounter. Hemodynamic instability can be inferred from tachycardia, hypotension, or signs of inadequate tissue perfusion, such as altered mental status. An accurate temperature is essential, especially in the context of possible infection. A relative bradycardia may be associated with nephrolithiasis.

Cardiopulmonary

Cardiopulmonary examination may identify comorbid conditions. Lower lobe pulmonary disease, such as infiltrates, abnormal pleural collections and pulmonary infarctions, can cause pain referred to either flank. Rales, decreased breath sounds, pleural rubs, or isolated wheezing can provide clues to these diseases. Cardiac auscultation can identify atrial fibrillation that may predispose a patient to the production of emboli. Abnormal valvular murmurs raise suspicion for infectious endocarditis, which may result in renal infarction.

Abdomen

On abdominal examination, the kidneys are generally not palpable. However, with polycystic disease or significant hydronephrosis, the kidneys may be palpated as deep structures in the upper abdomen. One must examine the patient for other masses, such as a pulsatile abdominal aortic aneurysm (AAA), pregnant uterus, or distended bladder. Auscultation of abnormal abdominal or renal bruits may suggest vascular disease.

Back

The kidneys are relatively protected in the bony confines of the costovertebral angles. Fist percussion may elicit flank pain; careful palpation separates this from midline musculoskeletal causes. It is recommended to start with the unaffected flank first, so the patient can develop a sense of trust and the clinician can develop a sense of the response to percussion. Renovascular disease may cause posteriorly located bruits.

Perineum

Examination of the external meatus may reveal irritation and/or discharge. Examine the labia and/or scrotum for additional pathology, such as subcutaneous air or cutaneous discoloration associated with necrotizing fasciitis (Fournier's disease). If these are identified and Fournier's disease is a possibility, prompt administration of intravenous (IV) antibiotics and timely surgical debridement in the operating room (OR) is critical. Lesions identified anywhere in or surrounding the perineum may be the cause of pain or suggest alternate etiologies.

Rectal

A careful rectal examination evaluates for both rectal pathology and integrity of the micturition reflex arc. Perianal sensation and "normal" sphincter tone may be subjective, but an intact *bulbocavernosus reflex* provides objective evidence that a complete reflex arc integrating the sensory nerves, spinal cord and motor fibers exists. The bulbocavernosus reflex is tested by inserting a finger in the rectum while gently tugging on an indwelling urinary catheter or lightly squeezing the glans or clitoris. This normally induces contraction of the anal sphincter and verifies the integrity of spinal cord segments S2–S4. The anal wink reflex (S2–S5) is tested by lightly touching the perianal skin and observing anal sphincter contraction.

Prostate

The digital examination of the prostate assesses for hypertrophy, tenderness and masses. A normal digital examination does not rule out internal urethral compression from asymmetric prostate lobe enlargement. Clinicians may identify a loss of the "normal" median sulcus with significant prostate enlargement, and the superior boundary of the prostate may be beyond reach of the examining digit. Abnormal pain or "bogginess" is a sign of prostatic infection. One must take care not to massage a tender or boggy prostate, as bacteremia may ensue and complicate a patient's course.

Pelvic

Often, the history alone cannot differentiate between a urologic or gynecologic cause of symptoms in female patients. A careful speculum and bimanual examination may identify gynecologic etiologies such as cervicitis, ovarian pathology, or urinary frequency associated with pregnancy. From a practical standpoint, a quick catheter sampling of urine may be obtained during the examination.

Differential diagnosis

Table 42.2 describes the differential diagnosis of urinary-related complaints.

Table 42.2 Differential diagnosis of urinary-related complaints

Dysuria
Bladder calculus
Cervicitis
Cystitis
Prostatitis
Urethral obstruction
• Stricture
• Foreign body
• Extrinsic compression
Urethritis
Urolithiasis
Vaginitis
Hematuria
Bleeding dyscrasias
Exercise
Infection – parasites, bacteria, renal tuberculosis
Glomerulonephritis
Myoglobinuria (positive urine dipstick, negative RBCs)
Neoplasia – from kidney, bladder, prostate to urethra
Polycystic kidney disease
Renal infarction – vascular disease, sickle cell disease
Trauma – from renal parenchyma to urethral orifice
Urolithiasis
Acute urinary retention
Benign prostatic hypertrophy
Clot retention – from trauma, neoplasia, renal origin
Myelitis
Paraplegia/cauda equina syndrome
Prostatitis
Urethral irritation – HSV infection, candida, chlamydia
Urethral or bladder neck calculus
Urethral stricture
Urethral trauma – iatrogenic vs. mechanical

HSV: herpes simplex virus; RBC: red blood cell.

Diagnostic testing

Laboratory studies

Urine collection

A clean sample of urine must be obtained from patients with urologic complaints. This is often problematic during an ED visit. Long waiting times often preclude adequate and timely sampling from patients able to produce an adequate specimen. The verbal presentation of diagnostic bladder catheterization to parents or patients is often met with fear.

In the pediatric population, a bag urine specimen is generally unacceptable for identification of UTI and submission for culture. Eighty-five percent or more of positive urine collections obtained via bag collection are false positives, prompting unnecessary and potentially harmful treatment and imaging studies that follow. Females and uncircumcised males need urine obtained by a one-time "in-and-out" bladder catheterization, which has less than a 1% chance of inducing a UTI. Some literature argues for the usefulness of a spontaneously voided mid-stream sample in a circumcised pediatric male, but waiting with a cup next to an uncomfortable patient is challenging at best, and futile in practice.

Adult males for the most part can provide a clean catch, mid-stream urine collection. This may be impossible in a debilitated patient unable to comply with instructions. Proper cleansing of the external meatus is important, as well as wasting the initial portion of urine into the toilet before collecting the sample.

Urine collection in sexually active adult females relies on the absence of vaginal discharge that can contaminate the urinary stream. History of vaginal discharge may not be accurate, making a pelvic examination important (especially in an adolescent female). The absence of vaginal discharge coupled with historical complaints of dysuria and frequency has nearly a 90% positive predictive value for cystitis. If there are no gynecologic complaints or discharge, a correctly obtained clean-catch urine sampling should suffice. Have the patient sit backwards on the toilet, cleanse her external genitalia, void a small amount into the toilet, and then collect the sample in a sterile container.

Urinalysis

Once an adequate urine sample is obtained, either point-of-care dipstick testing or formal laboratory urinalysis (UA) can be performed. Time of testing, cost and local practice trends contribute to widespread variation in testing.

Urine dipstick testing is quick, relatively inexpensive, and can be performed at the bedside, but is not without limitations. Up to 5% of patients with normal dipsticks have abnormal microscopy. Leukocyte esterase will pick up white blood cells (WBCs) associated with infection, but will also identify WBCs associated with any inflammation. Nitrite examination has a high positive predictive value (96%) for UTI, but a low negative predictive value. Nitrate is converted to nitrite via bacterial metabolism; however, bladder incubation is a prerequisite. Nitrite testing is best performed on the first void of the day, after overnight incubation in the bladder has allowed nitrite production to occur. Nitrites will not be detected for all enterococci or staphylococci species, as some do not reduce nitrates to nitrites. Bacterial counts double every hour at room temperature, and double more quickly at body temperature. Once obtained, a dipstick analysis should be performed promptly or urine should be sent to the laboratory. A centralized hospital lab may take over an hour to process the specimen and report results. Delayed evaluation of a urine specimen (e.g., urine obtained from the patient in the waiting room and left sitting before going to the laboratory) may lead to false-positive results.

The urine dipstick for blood looks for hemoglobin-like compounds, but does not differentiate between hemoglobin from red blood cells (RBCs) and myoglobin from skeletal muscle breakdown. Positive dipsticks should have confirmatory microscopy to quantitate the degree of hematuria. There is no correlation between red cells on microscopy and the degree of obstruction seen with renal calculi. Occasionally, a patient may have complete obstruction of the ureter without RBCs on initial UA; subsequent UAs may reveal hematuria, however. The presence of red cell casts suggests a renal etiology of hematuria.

Urinary pH is obtained via dipstick, and helps differentiate infections from intrinsic metabolic conditions. Alkaline urine (pH > 7.5) typically results from urea-splitting bacterial infections, such as those caused by *Proteus*, *Klebsiella* or *Pseudomonas*. Acidic urine (pH < 5.5) effectively rules out renal tubular acidosis (RTA) as a cause for nephrolithiasis; it does favor uric acid stone formation.

If preliminary testing is suggestive of UTI, the next question is "Does the patient need a urine culture?" Multiple studies have shown that urine cultures are unnecessary for the management of simple, uncomplicated patients. High-risk patients who require urine cultures include immunocompromised, age extremes, pregnant, obstructed, and treatment failures. It is important to carefully consider whether or not a urine culture will help with subsequent care, and consider whether systems are in place to ensure timely and accurate follow-up of culture results.

Additional studies

If a pelvic examination has been performed, standard practice is to send cultures for STIs such as gonorrhea and chlamydia. The actual test may vary between institutions, but often identifies asymptomatic STIs. The female patient of childbearing age should have a urine pregnancy test performed.

Gross hematuria may also prompt hematologic testing. A baseline hematocrit can identify anemia, but is often unnecessary with simple hematuria. Laboratory investigation of coagulation profiles may be helpful in the patient with a coagulation disorder or currently taking warfarin, but there is no indication for routine testing in a patient with hematuria. An international normalized ratio (INR) may uncover drug interactions between oral anticoagulants and antibiotics that prolong the INR (e.g., sulfonamides or quinolones), even if there has not been a change to a patient's anticoagulation dose or diet.

Routine investigation of renal function is common prior to IV administration of nephrotoxic radiocontrast material. Serum creatinine levels should also be measured in patients with a solitary kidney, a transplanted kidney, or a history of renal insufficiency. Prolonged ureteral obstruction can cause irreversible renal damage and creatinine elevation; however, not every patient with a kidney stone needs a baseline creatinine. Practice variations occur, but most urologists request a baseline creatinine for any stone > 5 mm in size, as these stones may not pass spontaneously.

Radiologic studies

The goal of radiologic imaging for urinary complaints is to identify complicated infections, anatomic abnormalities, or obstruction. Options include ultrasound (US), plain films, intravenous pyelogram (IVP) and computed tomography (CT).

US may identify bladder distention, and a carefully performed study can demonstrate fluid–fluid interfaces between urine and retained clot. Renal US may show hydronephrosis from obstruction. Though US may reveal shadowing from renal parenchymal stones, ureteral stones are typically not visualized.

The kidney–ureter–bladder (KUB) film, if properly performed, can identify radiopaque structures such as ureteral or bladder calculi. A urologist can identify stone location and follow progress towards spontaneous elimination. Calculi secondary to protease inhibitors are often not radiopaque, and may be missed by noncontrast imaging. A KUB X-ray may also diagnose emphysematous pyelonephritis from gas-forming organisms.

IVP images the entire renal unit via excretion of dye into the collecting system and transit to the bladder. This procedure places the patient at risk for ionic contrast medium reactions including increased pain, renal failure and anaphylaxis (which can be fatal). If positive for high-grade obstruction, a properly performed IVP requires delayed films that may take hours. IVPs can, however, generate valuable anatomic and functional information, and are still preferred by many urologists.

A recent advance in urinary imaging, especially for calculus disease, is noncontrast CT. This study may reveal the obstructing calculus as well as the degree of resulting hydronephrosis (Figure 42.3). Perhaps more importantly, the scan can identify an alternative etiology for a patient's symptoms in up to one-third of cases. If the CT is positive for obstruction, a urologist may request a confirmatory KUB film to identify the stone's location in two dimensions in relation to anatomic structures, such as a transverse process or the pelvic brim. This is a more cost-effective approach to following a stone's progress through the collecting system, and reduces potential morbidity associated with increased radiation exposure.

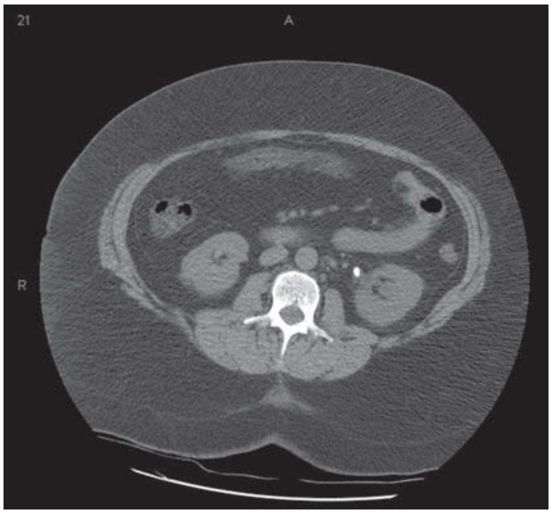

Figure 42.3
Kidney stone. Axial CT scan demonstrating a calcific opacity adjacent to the left kidney, consistent with a calculus in the proximal ureter. Courtesy: Gus M. Garmel, MD.

Calculous disease is often diagnosed clinically, and confirmed with radiologic imaging or eventual stone passage. Radiologic studies allow the clinician to accurately measure the size and precise anatomic location of the obstruction. Calculi sizing is very important in prognosis; a stone < 4–5 mm will pass spontaneously greater than 90% of the time. This rate of spontaneous passage falls dramatically with increasing stone size; only 10% of 8-mm calculi will pass spontaneously.

The time from diagnosis to actual stone passage also varies with stone size and initial location, among other things. Gravel-sized calculi up to 2 mm typically pass in 5–8 days. Calculi as large as 4 mm often require 7–14 days for spontaneous passage. These are rough estimations at best, and individual time to passage varies with the clinical situation, the stone's location and composition (especially shape), and the patient's anatomy.

In general, distal calculi pass more quickly than calculi causing proximal obstruction. Right-sided calculi are more likely to pass spontaneously compared with left-sided calculi. The three most common sites for ureteral obstruction include the junction of the ureter with the renal pelvis, the deflection of the ureter over the iliac vessels, and the ureterovesical junction (UVJ). The UVJ is the most common site of obstruction.

General treatment principles

Specific treatment of urologic disorders encountered in the ED varies by etiology. It is easiest to divide these into infection treatment, obstruction relief and pain control. The majority of urinary tract complaints have a combination of issues for clinicians to address.

Infection treatment

Infectious disorders need antimicrobial therapy directed against typical uropathogens. Most causative organisms are from colonization of the perineum, which helps direct therapy. There are many effective drugs for the typical uropathogen. A variety of past studies have compared short course (3 days) with long course (7–10 days) treatment for simple cystitis. For most drugs except nitrofurantoin, a 3-day course is preferred. The potential benefits of short-duration therapy include lower cost, improved compliance, and lower incidence of both patient-associated adverse outcomes and bacterial resistance to the agent selected.

Recommended therapy for UTIs, both complicated and simple, is not without controversy. Advocates for all types of therapy try to balance cost of therapy with local bacterial resistance patterns in order to come up with specific treatment recommendations. Antimicrobial resistance is often difficult to characterize because empiric therapy without urine culture is commonplace. While some locales are demonstrating increased bacterial resistance to all antimicrobials, there has also been a recent reversal in antibiotic resistance noted. For example, quinolone resistance seems to be increasing in many regions while trimethoprim resistance is actually decreasing, presumed secondary to practice patterns of the last decade.

Bacterial resistance to drugs and cost recommendations specific to a hospital's formulary are best handled by referring to your particular hospital or region's bacterial sensitivity patterns. Some regions have abnormally high bacterial resistance to cephalosporins or sulfonamide drugs. In these regions, quinolone therapy may be a more practical choice, despite the increased expense. Caution should also be exercised in older individuals due to a slightly increased risk of tendon rupture. The potential for quinolone-directed osseous and cartilage damage prevents its use in the pediatric population. Sulfa-based medicines have worked well in the pediatric population in the past, attacking bacteria by inhibiting folate metabolism. This class of drugs poses special concerns to pregnant patients and the fetus in the first trimester, when folate is crucial to organogenesis. Third trimester use of sulfonamides may also lead to *kernicterus*, the deposition of bilirubin pigments in the fetal brain. Recent studies have questioned the safety of nitrofurantoin use in pregnancy. It is no longer recommended in patients over 65 years if age, and it is contraindicated in children less than 1 month of age or in individuals with G6PD deficiency.

For uncomplicated UTIs, a 3-day course of trimethoprim–sulfamethoxazole (TMP–SMX) results in a bacteriologic cure within 7 days in 94% of women (Table 42.3). Even where bacterial resistance rates are as high as 30%, cure rates of 80–85% in women are still possible. Concern over TMP-SMX resistance has prompted many experts to recommend fluoroquinolones for the treatment of UTIs. Fluoroquinolones are highly bioavailable, allowing for single daily dosing and higher patient compliance. However, they are class C agents with regard to pregnancy, and are more expensive. The Infectious Diseases Society of America recommends initial use of TMP–SMX except in areas where bacterial resistance exceeds 10–20%. Care must be exercised with some of the newer quinolones on the market, as they have little or no renal penetration, which limits their effectiveness against urinary pathogens.

Differentiating between simple and complicated UTIs can be difficult. Thirty to fifty percent of women with isolated lower tract symptoms have subclinical pyelonephritis (an upper tract urinary infection, which, by definition, is not simple). Prior treatment, microabscess formation, retained calculi, indwelling catheters, and urine obstruction and stasis all create complicated infections in otherwise immunocompetent hosts. Altered anatomy, such as progesterone-induced ureteral dilation and bladder stasis of urine found in pregnant patients, warrants prolonged treatment for 7–14 days (Table 42.4). As many pediatric UTIs are accompanied by anatomic abnormalities, a longer treatment regimen with continued prophylaxis until the child has had urologic imaging is generally recommended (Table 42.5). Patients at age extremes commonly suffer from transient bacteremia, making aggressive therapy important. Any patient discharged from the ED with a complicated UTI should be referred to primary care for a prompt recheck and close follow-up. If primary care resources are not immediately available in the community, these patients should return to the ED to be rechecked within 2 days. At the recheck visit, response to therapy should be noted and urine cultures should be checked for antibiotic sensitivities. If the patient is not improving, or the sensitivities are marginal, the duration of antibiotic therapy can be extended to 14–21 days.

Other clinical factors to consider include the patient's hydration status and his or her ability to maintain hydration following discharge. Failure to keep even the most powerful and expensive antibiotic in the GI system renders it ineffective. Discharge instructions for the patient with a simple UTI often encourage "forcing fluids" and acidifying the urine. Forcing fluids should ensure adequate hydration, and contributes to bladder washout. However, large volumes of dilute urine will decrease the antibiotic's concentration in the urine. It is therefore probably more important to emphasize frequent and complete bladder emptying. Urine acidification,

Table 42.3 Uncomplicated UTIs

Agent	Dosing
TMP–SMX DS	160/800 mg PO BID for 3 days
Ciprofloxacin	250 mg PO BID for 3 days
Nitrofurantoin	100 mg PO BID for 10 days
Nitrofurantoin monohydrate	100 mg PO BID for 7 days
Levofloxacin	250 mg PO QD for 3 days
Cefpodoxime	100 mg PO BID for 3 days

BID: twice a day; DS: double strength; PO: per os; QD: once a day; TMP–SMX: trimethoprim–sulfamethoxazole.

Table 42.4 Complicated UTIs

Agent	Dosing
Initial therapy – fluoroquinolone	
Levofloxacin	250 mg PO QD for 10–14 days
Ciprofloxacin XR	500 mg PO QD for 10–14 days
Second-line therapy	
TMP–SMX DS	160/800 mg PO BID for 10–14 days
Amoxicillin/clavulanate	500 mg PO TID *or* 875 mg PO BID for 10–14 days
Ceftriaxone–parenteral	1–2 g IV QD
Gentamicin–parenteral	3–5 mg/kg/day IV divided TID (according to renal function)
Ampicillin/sulbactam–parenteral	3 g IV Q 6 hrs
Aztreonam – parenteral (if contraindications to quinolones or beta–lactam agents)	1 g IV Q 8–12 hrs

BID: twice a day; DS: double strength; IV: intravenous; PO: per os; Q: every; QD: once a day; TID: three times a day; TMP–SMX: trimethoprim–sulfamethoxazole; XR: extended release.

Table 42.5 Antimicrobial treatment of pediatric UTI

Agent	Dosing
Oral drugs	
Penicillin	
Ampicillin	50–100 mg/kg/day Q 6 hrs
Amoxicillin	20–40 mg/kg/day Q 8 hrs
Augmentin	20–40 mg/kg/day Q 8 hrs
Sulfonamide	
Trimethoprim–sulfamethoxazole	8 mg/kg/day Q 6 hrs[a]
Cephalosporin	
Cephalexin	25–50 mg/kg/day Q 6 hrs
Cefaclor	20 mg/kg/day Q 8 hrs
Cefixime	8 mg/kg/day Q 12–24 hrs
Cefadroxil	30 mg/kg/day Q 12–24 hrs[a]
Fluoroquinolone	
Ciprofloxacin	20–40 mg/kg/day Q 12 hrs[a]
Nalidixic acid	55 mg/kg/day Q 6 hrs
Other	
Nitrofurantoin	5–7 mg/kg/day Q 6 hrs
Parenteral drugs	
Aminoglycoside	
Gentamicin	7.5 mg/kg/day Q 8 hrs[a]
Tobramycin	7.5 mg/kg/day Q 8 hrs[a]
Penicillin	
Ampicillin	50–100 mg/kg/day Q 6 hrs
Ticarcillin	50–200 mg/kg/day Q 4–8 hrs
Cephalosporin	
Cefazolin	25–50 mg/kg/day Q 6–8 hrs[a]
Cefotaxime	50–180 mg/kg/day Q 4–6 hrs[a]
Ceftriaxone	50–75 mg/kg/day Q 12–24 hrs
Cetriazidime	90–150 mg/kg/day Q 8–12 hrs[a]
Cefepime	100 mg/kg/day Q 12 hrs
Fluoroquinolone	
Ciprofloxacin	18–30 mg/kg/day Q 8 hrs[a]

[a] Dose adjustment required with azotemia.

Adapted from Chang SL, Shortliffe LD. Pediatric urinary tract infections. *Pediatr Clin North Am* 2006;**53**:379–400.

often accomplished by drinking cranberry juice, can result in a relative reduction of bacterial loads in infected urine. Some of this may be related to interference with bacterial adherence to uroepithelial cells via an unknown mechanism.

If a patient has a UTI in the setting of simultaneous ureteral obstruction, a renal abscess should be considered. Antibiotics should be initiated immediately in the ED, but prompt emptying of the renal pelvis via ureteral stent or percutaneous nephrostomy drainage will be required in most cases. Urologic or interventional radiology consultation is important in these patients.

Obstruction relief

Bladder obstruction requires urinary drainage or diversion. Any mechanical instrumentation to relieve such obstruction poses iatrogenic risk to the patient, either via transient bacteremia or creation of a false urethral passage. Lubrication, topical anesthetics, patience and practice help prevent such complications. The most common approach to bladder obstruction is placement of a Foley catheter. Another specialized urinary catheter regularly used in the ED setting is the Coudé catheter, a slightly stiffer catheter with a relatively fixed curve at the distal portion. This feature allows the catheter to deflect off an enlarged prostate lobe at the bladder neck and pass through the prostatic urethra. If the patient has had a prior transurethral resection of the prostate (TURP), one may consider using a smaller Coudé catheter.

Another option for urinary diversion is the placement of a suprapubic catheter, bypassing the prostate and urethra completely. This temporizing measure of bladder decompression via needle and syringe may buy time in an extremely uncomfortable patient awaiting the arrival of a urologist. A urologist may attempt to instrument the urethra through the use of urethral sounds, filiforms and followers, or fiberoptic instruments and Seldinger wire passage. These techniques are beyond the scope of emergency medicine practice.

Following bladder catheterization, if greater than 800 mL of urine is obtained, it is prudent to leave the catheter in place and remove it in 1–2 weeks. Otherwise, bladder atony will likely lead to a subsequent episode of urinary retention, as will continued obstruction secondary to prostatic enlargement. Up to 56% of males will have recurrence of AUR in the week following catheter removal, an important consideration when presenting treatment options to the patient. Outpatient initiation of alpha-blocking agents (tamsulosin) relax smooth muscle at the bladder neck and prostatic capsule, potentially limiting further episodes of AUR.

The clinician may also prescribe therapy to facilitate ureteral calculi migration to the bladder via “medical expulsive therapy.” Stone passage is 44% more likely to occur with alpha-blocker therapy, which has become standard practice by most emergency physicians. There is also evidence that calcium channel blockers may effect smooth muscle relaxation of the ureters as well, perhaps a less expensive option in a patient with limited resources.

Pain control

Pain management for patients presenting with urinary complaints is directed at either irritative or obstructive symptoms. Irritative symptoms may require only acetaminophen or NSAIDs, with the occasional use of urinary anesthetics (phenazopyridine [Pyridium]). Only a few days of therapy are required to relieve irritative symptoms. Phenazopyridine, in particular, should not be used longer than 2 days. Side effects of phenazopyridine include urine discoloration and the potential for hemolysis and methemoglobinemia, especially in susceptible patients with glucose-6-dehydrogenase (G6PD) deficiency who are also treated with sulfonamides.

Ureteral obstruction from calculi creates intermittent episodes of complete obstruction, hydronephrosis and pain. Many clinicians use either spasmolytic or anticholinergic drugs to reduce the pain and frequency of these colicky episodes, although the medical literature does not support this practice. Newer investigations have revealed that much of the pain is mediated by ureter-released prostaglandins in response to obstruction. These prostaglandins increase peristalsis, which is an attempt by the ureter to move the stone down for spontaneous passage. NSAIDs (ibuprofen or ketorolac) are effective in relieving the pain, nausea and vomiting associated with renal colic.

Narcotics are also effective at blunting pain in the ED. This class of agents is by far the most commonly used for the treatment of acute pain from urinary complaints such as renal colic or nephrolithiasis. Incremental IV dosing allows better titration and pain reduction than intramuscular (IM) injections. A short course of narcotics and NSAIDs are commonly prescribed at discharge to control pain.

Special patients

Male

AUR is almost exclusively a male disorder secondary to BPH. UTIs in the previously healthy young male are uncommon, and are considered complicated as they are often related to underlying, often undiagnosed urologic anomalies. Prostatitis is also considered a complicated infection, with retrograde inoculation of Gram-negative organisms or Chlamydia from the urethra. Chronic prostatitis may require 4–6 weeks of antibiotic duration to sterilize the glandular prostatic tissue .

Elderly

Elderly patients are likely to have significant comorbidities, but may not be able to provide this information. It is not uncommon for UTIs to cause confusion or altered levels of consciousness in elderly patients; therefore, UTI must be considered as a possible etiology for behavioral changes. Nursing home patients present several unique challenges; not only do these patients tend to be colonized with antibiotic-resistant organisms, but indwelling catheters predispose to bladder colonization and ascending infections. Catheter-associated infections can develop from urine reflux from an infected or colonized catheter collection bag, or from bacterial migration of exudates along the catheter itself. Consider removing the old catheter and sampling urine through a new catheter, or consider other forms of urinary collection for the long term. Condom catheters for males without bladder outlet obstruction remain a viable means of urinary collection, although skin breakdown and infection of the glans is common. Catheter-associated infections are best treated for 10–14 days duration, as these are complicated UTIs by their very nature. Emergency physicians should coordinate care with primary care physicians, as the unwarranted or extended use of indwelling bladder catheters is a common problem. Approximately 5% of these patients will develop bacteriuria each day, even with optimal catheter and bladder care, which adds risk to this older population. A unique problem in the elderly male patient is extension of organisms into the prostate itself, with subsequent obstruction of prostatic drainage from an indwelling catheter. Such a patient needs prompt urologic consultation and likely suprapubic urinary diversion. Another problem unique to a male patient with an indwelling catheter is the development of a paraphimosis, which occurs when the foreskin is not reduced over the glans. This creates the potential for ischemic complications of the glans.

Pregnant

Symptoms related to the urinary tract are relatively common throughout pregnancy. Conditions ranging from asymptomatic bacteriuria to complicated upper tract disease (pyelonephritis) can have adverse effects on both mother and fetus, and are important for clinicians to consider and identify. Hormone-induced changes of the renal collecting system, primarily from elevated progesterone, result in decreased ureteral and bladder tone and physiologic hydroureter of pregnancy. Pregnant women also do not completely empty their bladders; urinary stasis predisposes their bladders to bacterial overgrowth and proliferation.

Asymptomatic bacteriuria is a relatively common entity, identified in up to 9% of first-trimester gestations. Untreated disease can progress to pyelonephritis in 20–40% of patients in some studies, with associated risks of sepsis, preterm delivery, and potential intrauterine fetal growth retardation. Symptomatic cystitis in pregnancy deserves empiric treatment, with the additional caveat that urine culture be performed. Up to 15% of pregnant patients with cystitis will develop relapses during their pregnancy, thus the culture and sensitivity helps guide follow-up therapy. Ascending upper tract infection (pyelonephritis) poses significant risk to pregnant women, both from a medical as well as an obstetrical standpoint. Up to 20% of such patients have been shown to develop severe complications, such as urosepsis or preterm delivery.

Outpatient management of asymptomatic bacteriuria and uncomplicated cystitis in pregnancy is directed at the typical uropathogens, with *Escherichia coli* responsible for up to two-thirds of all cases. Cephalosporins, amoxicillin, or sulfonamide therapy for a 3-day course is appropriate (Table 42.6). Use of sulfonamides in first-trimester pregnant women, at term, or for mothers nursing infants younger than 2 months of age is not advised, as sulfonamides may be teratogenic and likely promote displacement of bilirubin from plasma proteins, leading to kernicterus. Fluoroquinolones should never be used in the pregnant patient due to their actions on growing cartilage. Nitrofurantoin for a 7-day course of therapy is an effective agent for uncomplicated disease, although recent reports have recommended increased scrutiny with its use in pregnancy.

Table 42.6 Treatment of UTI during pregnancy

Agent	Dosing
Amoxicillin	500 mg PO TID
Cephalexin	500 mg PO QID
TMP–SMX DS (2nd trimester only)	160/800 mg PO BID
Nitrofurantoin monohydrate	100 mg PO BID
Ceftriaxone–parenteral therapy	1–2 g IV QD

BID: twice a day; DS: double strength; IV: intravenous; PO: per os; QD: once a day; QID: four times a day; TID: three times a day; TMP–SMX: trimethoprim–sulfamethoxazole.

Pyelonephritis may complicate up to 2% of all pregnancies. The nonpregnant patient often responds to outpatient therapy, but the pregnant patient may require admission by the obstetrical service for IV hydration and antibiotics. Cephalosporins are typically chosen for therapy, but local resistance patterns as well as obstetrician preferences should be considered. It is prudent to involve the obstetrical service in the care plan. If discharge is appropriate, a 10–14-day course of antibiotic therapy is typical, with close follow-up essential.

Pediatric

Pediatric patients, like the elderly, are often unable to localize pathology via an adequate history. One must consider a urinary source of infection in all febrile infants. When obtaining urine samples via bladder instrumentation, it is advisable to send off a culture to ensure appropriate treatment is continued during follow-up. After completion of therapy for a UTI in a high-risk pediatric patient, many authors recommend outpatient urologic imaging to identify possible anatomic anomalies. Therefore, prompt and close outpatient communication and follow-up is crucial.

Immune compromised

There are several important points to remember when clinicians encounter an immunocompromised patient. Often, the organisms causing the infection are atypical, so urine culture is necessary to identify the organism and antibiotic sensitivities. Clinicians must lower their threshold to hospitalize an immunocompromised patient with a urinary infection. If the patient has had a renal transplant, it is extremely important to carefully examine the transplanted kidney during the abdominal examination. The transplanted organ is typically located in the right lower quadrant, and may mimic appendicitis. Excessive pain on palpation can signify either rejection or complicated pyelonephritis. In such cases, urgent consultation with the patient's nephrologist or transplant surgeon is required.

Human immunodeficiency virus (HIV) protease inhibitor use (primarily indinavir) can lead to calculi that are not radiopaque, even with CT-KUB studies. Formal renal imaging with IV contrast using IVP or CT-KUB can delineate function and obstruction location, and might be needed in this patient population. If the patient practices ano-insertive intercourse, enteric organisms such as enterobacter species must also be considered as causative agents, and the therapy altered appropriately.

Disposition

Patient disposition depends on several factors, including disease process, comorbidities, and ability to follow directed therapy. Urinary obstruction with concomitant infection predisposes to renal abscess formation; therefore, admission with urinary drainage to bypass the obstruction is mandatory. Pain that is not well controlled by oral agents, or persistent nausea and vomiting that prevent adequate hydration should prompt strong consideration for hospital admission. An immunocompromised host, a problem with a solitary kidney, and a painful high-grade obstruction are relative indications for hospital admission, as are pregnancy and complicated social situations.

Patients with intermittent obstruction secondary to calculi need proper follow-up arranged. One pitfall in management is the erroneous conclusion that the absence of pain means the absence of obstruction. Irreversible renal damage can be seen in as little as 2 weeks, so prompt patient follow-up is imperative. Confirmation of calculi passage is the gold standard. Patients should be instructed to strain their urine in the ED and following discharge. This is much easier for the male patient to accomplish, using either a strainer or urinating through coffee filters. Following passage, patients should bring the stone(s) to their primary care provider. Crystallographic analysis of the stone is not always necessary, as it is often difficult to arrange, costly to the patient, and helpful only in genetic- or metabolic-induced nephrolithiasis.

Pearls, pitfalls and myths

- UA is the cornerstone for diagnosing patients with urologic disease, yet false negative results do occur. Negative dipstick UA carries a 5% chance of abnormal urinary sediment on microscopy. Nephrolithiasis with complete ureteral obstruction may have a completely normal UA due to urinary flow through the unaffected ureter.
- Although nephrolithiasis is a common clinical diagnosis, resist the temptation to discharge the patient without first obtaining a UA. UTI in the presence of obstruction is a true urologic emergency, often referred to as “pus under pressure.”
- Cost-effective emergency care should be a goal of all clinicians, but the total elimination of urine cultures is not part of this goal. Culture the high-risk patient (immunocompromised, elderly, infants, pregnant), as well as patients with prior treatment failures.

- Although quinolones are increasingly utilized as monotherapy in urologic infections, clinicians must consider quinolone-related resistance, drug interactions, or adverse effects, especially when prescribing to the elderly. Always investigate potential drug–drug interactions to prevent iatrogenic complications, such as bleeding or INR elevations in patients taking anticoagulants.

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43 Vaginal bleeding

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Scope of the problem

A common emergency department (ED) complaint is vaginal bleeding, accounting for up to 10% of ED visits in many centers. The differential diagnosis for vaginal bleeding is relatively short, with the most serious condition being ectopic pregnancy. The most common conditions include threatened miscarriage in the pregnant patient and abnormal uterine bleeding (AUB) in the non-pregnant patient. According to the Centers for Disease Control and Prevention (CDC), there were 108,800 ectopic pregnancies reported in the United States in 1992. Variable reporting standards and improved diagnostic testing have made subsequent statistics incomparable. Currently, ectopic pregnancies account for approximately 2% of reported pregnancies. Threatened miscarriage (formerly referred to in the medical literature as threatened abortion) occurs in 20–30% of all pregnancies; as many as 50% of those threatened will go on to spontaneously miscarry. Septic miscarriage and gestational trophoblastic disease must also be considered in the differential diagnosis of vaginal bleeding in a pregnant woman, accounting for 0.4–0.6 per 100,000 spontaneous miscarriages and 0.1–1% of pregnancies, respectively. Understanding the definitions of different classifications and management options for nonviable gestations is important to effectively manage patients and communicate with consultants in the ED. Second- and third-trimester bleeding and postpartum vaginal bleeding are not discussed in this chapter, because these patients are routinely evaluated in Labor and Delivery and infrequently managed by emergency physicians.

Not all women with vaginal bleeding are pregnant. During the reproductive years, AUB is defined as bleeding from the uterus that is irregular in amount, timing, or duration. Prior to menarche or following menopause, any uterine bleeding is considered abnormal. Dysfunctional uterine bleeding (DUB) is a subset of AUB; it is defined as abnormal bleeding unrelated to pregnancy, exogenous gonadal steroids (e.g., contraceptives and hormone replacement), intrauterine contraceptive devices (IUDs), other medical conditions, or structural uterine pathology. DUB is estimated to affect up to 20% of reproductive-aged women. Although the diagnosis and ultimate treatment may be left to obstetrician-gynecologists, acute care practitioners should understand the various causes of AUB so that appropriate acute intervention can be provided.

Anatomic essentials

Physiology of menstruation

Normal menstrual bleeding results from the cyclical withdrawal of estrogen and progesterone that occurs, on average, 14 days following ovulation in the absence of pregnancy (Figure 43.1). Menses typically last 4 or 5 days, with a normal range of 2–7 days. Flow is usually heaviest in the first 2–3 days, and tapers to spotting in the last 2–3 days. The normal blood volume lost during menses averages 30–40 mL; an amount greater than 80 mL is considered excessive. For the typical woman of reproductive age, iron stores can be depleted when monthly blood loss exceeds 60 mL. The term *menorrhagia* means excessive menstrual blood flow. Menses are normally predictable, with a cycle (first day of one menses to the first day of the next) ranging from 21 to 35 days.

The regulation of menses depends on a number of hormonal factors that result in ovulation. If ovulation does not occur, as in anovulation associated with polycystic ovarian syndrome, the endometrium is exposed solely to the proliferative effects of unopposed estrogen. This endometrium will eventually build to the point of instability and result in heavy, asynchronous bleeding termed *metrorrhagia*. Following ovulation, the Graafian follicle that expelled the ovum becomes the corpus luteum (CL), which produces abundant estradiol and progesterone. These hormones exert profound effects on the endometrium, causing it to be receptive to implantation for a short period of time. In the absence of pregnancy, the CL involutes, resulting in withdrawal of the effects of estradiol and progesterone on the endometrium. The culmination of this is sloughing and bleeding that comprises menstrual flow.

The next steps in the menstrual process are all directed at establishing hemostasis and subsequent regeneration of the endometrium's superficial layer. The endometrium is unique in that platelets have relatively little role in the process of hemostasis. Instead, endometrial hemostasis relies first on the presence of local vasoconstrictors, then on factors of the extrinsic and intrinsic clotting cascades that result in the formation of a vascular clot. Once hemostasis has been achieved, the processes of endometrial angiogenesis and re-epithelialization rebuild the endometrium, preparing it for implantation should fertilization occur during the next cycle. The factors involved in initiating angiogenesis are unclear, but appear to be associated with rising estrogen levels.

Fertilization, implantation and embryology

For pregnancy to occur, fertilization must take place after ovulation. This generally occurs in the ampullary region of the fallopian tube. The ovum, fertilized or not, then moves down the tube towards the uterine endometrial cavity by ciliary transport. Implantation of the fertilized ovum in the endometrium occurs about 7 days post-fertilization, approximately 3 weeks after the last menstrual period (LMP). The developing embryo is surrounded by the

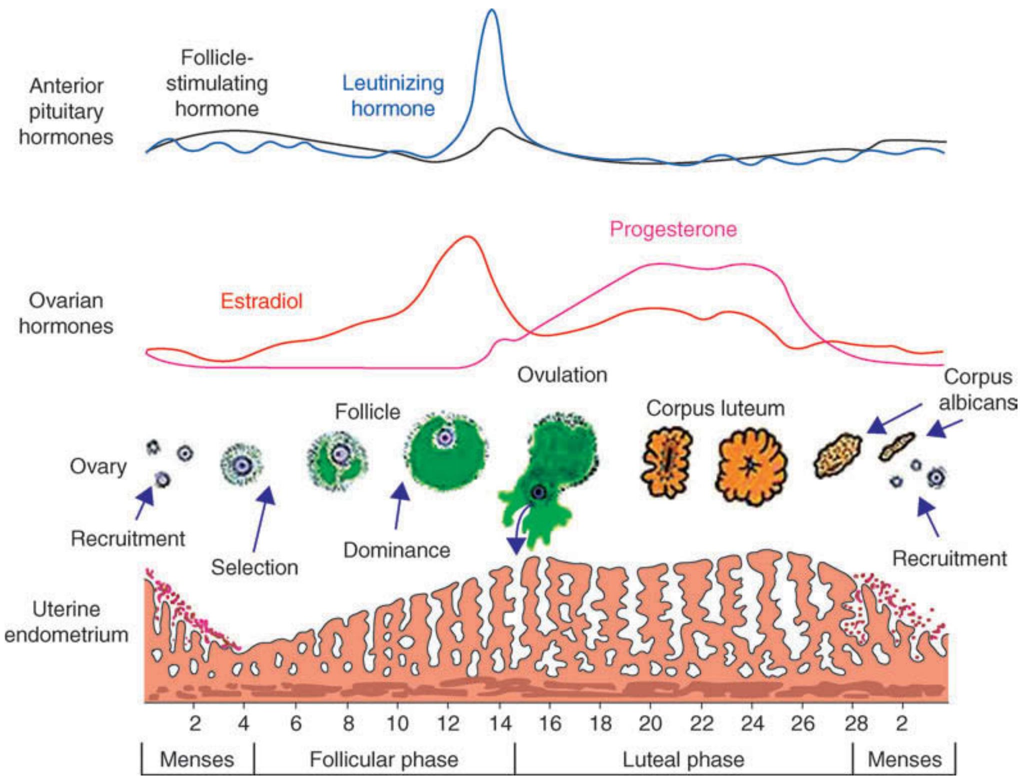
Anterior pituitary hormones
Follicle-stimulating hormone
Leutinizing hormone
Ovarian hormones
Estradiol
Progesterone
Ovary
Follicle
Ovulation
Corpus luteum
Corpus albicans
Recruitment
Selection
Dominance
Recruitment
Uterine endometrium
2 4 6 8 10 12 14 16 18 20 22 24 26 28 2
Menses
Follicular phase
Luteal phase
Menses

Figure 43.1
Normal menstrual cycle, including ovulation and menses. Used with permission, John Parrish, University of Wisconsin-Madison.

trophoblast, which gives rise to the placenta and secretes human chorionic gonadotropin (hCG). It is hCG that prevents the degeneration of the CL, which continues to secrete adequate progesterone to maintain the developing gestation.

Ectopic pregnancy results from implantation of the fertilized ovum in a location other than the endometrium. Greater than 95% of ectopic pregnancies implant in the fallopian tube. Of these, 80% implant in the ampullary portion, 12% in the isthmus, 5% in the fimbriated end of the tube, and 2% at the junction of the fallopian tube and uterus. The latter site of implantation is often referred to as an interstitial or cornual ectopic pregnancy. Interstitial ectopic pregnancies deserve special consideration because they are both rare (accounting for only 2–3% of ectopic pregnancies) and dangerous (mortality rate more than twice that of other tubal pregnancies, 2.2% vs. <1%). Additional sites of ectopic implantation include the abdomen, cervix and ovary. Once tubal implantation has occurred, there are four potential outcomes: the ectopic pregnancy may erode through the tube, leading to tubal rupture with associated intra-abdominal hemorrhage; it may persist within an intact tube with or without associated tubal hematoma and/or intra-abdominal hemorrhage; it may abort out the fimbriated end of the fallopian tube; or it may spontaneously involute.

Red flags

Emergency clinicians must be adept at recognizing “red flags” (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 43.1).

History

Are you pregnant?

This is the key question, as the entire thought process for diagnosing and treating patients with vaginal bleeding is contingent on whether or not the patient is pregnant. Although many patients know they are pregnant, many do not. Sixty-three percent of the time, a woman who thinks she may be pregnant is correct. However, about 11% of ED patients who deny pregnancy are pregnant, and approximately 7% who report a normal menstrual history and deny sexual contact are pregnant. Therefore, a negative history for pregnancy should be considered unreliable. All women of childbearing age (12–55 years old) with vaginal bleeding should have a pregnancy test, regardless of menstrual history.

Table 43.1 Vaginal bleeding red flags

History	Concerning diagnosis
VB + fever and/or foul discharge	Septic miscarriage, PID
VB + syncope	Ectopic pregnancy, severe anemia
VB + abdominal pain	Ectopic pregnancy, miscarriage, appendicitis, endometritis, pyelonephritis
VB + trauma	Pelvic fracture, placental abruption, uterine rupture, vaginal trauma
VB + ovulation induction (fertility) agents	Heterotopic pregnancy
Examination finding	**Concerning diagnosis**
VB + cervical motion tenderness	Ectopic pregnancy, PID, appendicitis
VB + asymmetric adnexal tenderness	Ectopic pregnancy, tubo-ovarian abscess, adnexal torsion
VB + open internal cervical os	Miscarriage in progress (inevitable miscarriage)
VB + enlarged tender uterus and/or foul discharge	Septic miscarriage, PID, salpingitis, endometritis, retained products of conception if recently postpartum

PID: pelvic inflammatory disease; VB: vaginal bleeding.

When was your last normal menstrual period, and are your periods usually regular?

Despite the above discussion, it is useful to identify the timing of the patient's LMP in order to date the pregnancy (should it be confirmed). The most common reason for amenorrhea (cessation of menses) is pregnancy. The patient's LMP is also important in a non-pregnant vaginal bleeding patient, as the pattern of her periods and current bleeding history can establish whether her abnormal bleeding is ovulatory (cyclic and predictable bleeding) or anovulatory (bleeding that is neither cyclic nor predictable).

When did the bleeding start, is the quantity of bleeding more than a usual period, is the blood clotted or not, and are you dizzy with position change?

These questions provide a crude estimate for how much blood has been lost. The recent onset of bleeding may be more consistent with potentially life-threatening conditions such as ectopic pregnancy and miscarriage, whereas prolonged or insidious bleeding is more suggestive of hormonal dysfunction. The actual amount of blood lost is usually difficult for a patient to quantify specifically, so comparing the amount of blood lost to the woman's usual menstrual flow may be helpful. Menstrual blood does not usually clot because of fibrinolysis that occurs in the endometrium. However, clots in the menstrual flow indicate relatively heavy bleeding, because the blood does not remain in the uterus long enough for fibrinolysis to occur. The clinical significance of the bleeding (such as symptomatic anemia) is important since this helps guide ED management and resuscitation, if necessary.

Have you passed any tissue?

The passage of fetal tissue may be helpful in excluding ectopic pregnancy in the correct clinical context. However, without a clinician's ability to identify and confirm products of conception (POC), the information is too unreliable to be of much value. Additionally, large clots or a decidual cast from the endometrium may be passed and mimic POC in ectopic pregnancies, which may confuse the clinical scenario.

Are you on hormone replacement therapy or using any form of birth control?

Exogenous gonadal steroids (estrogens and progesterones) can be associated with AUB. These hormones may be used for contraception or hormone replacement therapy (HRT), depending on the woman's age. Nonhormonal IUDs for contraception have also been associated with heavy menses and AUB secondary to changes in the prostaglandin milieu of the endometrium. Pregnancy despite the presence of an IUD is an ectopic pregnancy until proven otherwise.

Any history of trauma?

Vaginal bleeding in the setting of abdominal or pelvic trauma expands the differential diagnosis to include uterine rupture and placental abruption in a pregnant patient. The trauma patient will generally be evaluated and resuscitated more urgently because of the potential for rapid hemodynamic compromise. Pregnant women are at increased risk for nonaccidental abdominal and pelvic trauma from current or prior partners, which must be considered during patient evaluation.

Associated symptoms

Nausea and vomiting

Gastrointestinal (GI) upset or distress may be the first symptom of early pregnancy in a woman who is having AUB. Thus it is important to rule in or rule out pregnancy at the beginning of an evaluation of all female patients with nausea or vomiting. Pyelonephritis also commonly presents with nausea and vomiting; management of the pregnant patient with pyelonephritis may differ from the nonpregnant patient.

Fever or "flu" symptoms

Pelvic inflammatory disease (PID) may present with AUB secondary to concomitant endometritis. Septic miscarriage accompanied by infection, spontaneous miscarriage, or induced abortion may also present with these symptoms.

Urinary symptoms

Dysuria is a common complaint in women with gynecologic (GYN) pathology. Hematuria from a urinary tract infection (UTI) or pyelonephritis may be mistaken as vaginal bleeding with or without urinary symptoms.

Dizziness, lightheadedness, syncope or near-syncope

Does the patient have symptomatic anemia? Has the patient lost enough blood volume such that she has orthostatic hypotension? Symptoms such as dizziness, lightheadedness, syncope or near-syncope suggest immediate fluid resuscitation is necessary. Clinicians must decide upon blood product replacement, and must closely monitor vital sign trends. Complaints of chest pain and shortness of breath should be taken seriously in the setting of vaginal bleeding, as anemia may precipitate coronary ischemia.

Past medical

Obstetric

By convention, the number of pregnancies, regardless of their result, is termed *gravida*, the number of live births is termed *para*, and the number of pregnancies that did not result in a live birth is termed *abortions*. The nomenclature $G_{\#}$–$P_{\#}$–$Ab_{\#}$ is used, such that a woman who has been pregnant five times with three live births, one miscarriage and one therapeutic abortion would have her obstetrical history summarized as $G_5P_3Ab_{1,1}$.

Prior sexually transmitted infection, pelvic inflammatory disease, previous ectopic pregnancy, tubal surgery, smoking, frequent (daily) douching, current intrauterine contraceptive device, or infertility with or without treatment

The most common cause of ectopic pregnancy is damage to the mucosa of the fallopian tube, which prevents transport of the fertilized ovum to the endometrial cavity. Mucosal damage is most often a result of tubal infection and scarring. Tubal surgery and diethylstilbestrol (DES) exposure have been demonstrated to play a role in other causes of ectopic pregnancy, as well as defects in tubal motility. Defects in the fertilized ovum itself may increase ectopic pregnancy risk due to impaired implantation prior to arrival in the endometrial cavity. Hormonal factors have also been associated with an increased risk of ectopic pregnancy. Supraphysiologic levels of estradiol or progesterone have been shown to inhibit tubal migration, which may account for the increased incidence of ectopic pregnancies in patients on ovulation-induction agents (e.g., clomiphene [Clomid] or other fertility agents). There has been an association of smoking and daily douching with ectopic pregnancy as well, assumed due to abnormal ciliary transport.

Risk factors for ectopic pregnancy are important to elicit, as 50% of women with ectopic pregnancy will have one or more of these risk factors (Table 43.2). However, depending on the population, 25% of women presenting with threatened miscarriage will have one or more risk factors for ectopic pregnancy. The presence of one or more risk factors for ectopic pregnancy is helpful, but the diagnosis remains possible despite their absence.

Table 43.2 Ectopic pregnancy risk factors

- Previous ectopic pregnancy
- Tubal infection
- Tubal or pelvic surgery
- Intrauterine contraceptive device use
- Infertility for > 2 years, treated or untreated
- Diethylstilbestrol exposure
- Smoking
- Frequent (daily) douching

Previous treatment for abnormal uterine bleeding, oligomenorrhea, or amenorrhea

Oligomenorrhea is a decreased frequency of menstrual periods; amenorrhea is the cessation of menses. A history of either should increase a clinician's suspicion for anovulation and possibly DUB as the cause for abnormal vaginal bleeding.

Hirsutism, obesity, acne, heat or cold intolerance, diarrhea, constipation, weight loss or gain, or galactorrhea

Signs of androgen excess commonly associated with polycystic ovarian syndrome may suggest a cause of AUB. Endocrinopathies such as thyroid dysfunction (mainly hypothyroidism) and hyperprolactinemia can cause AUB, and are relevant to the diagnostic evaluation.

Easy bruising, excessive bleeding, or familial bleeding tendencies

Vaginal bleeding might be the presenting sign of a bleeding disorder or coagulopathy, such as idiopathic thrombocytopenic purpura (ITP) or von Willebrand's disease. A family history of bleeding disorders in a female with AUB may warrant investigation for coagulopathy or bleeding diathesis.

Medications

Use of platelet inhibitors such as aspirin or clopidogrel, or other anticoagulants like warfarin or low-molecular-weight heparin would be important to elicit, because vaginal bleeding might be iatrogenic.

Knowledge of the use of ovulation-induction (fertility) agents is crucial because of the increased risk for ectopic pregnancy and multiple gestations. Additionally, concomitant (simultaneous) intrauterine and ectopic pregnancies, termed *heterotopic pregnancy*, can occur with significantly increased frequency in women using these agents. This impacts the diagnostic accuracy of conventional tests used for excluding ectopic pregnancy, including pelvic ultrasound and quantitative β-hCG levels.

Physical examination

General appearance

Clinicians should immediately assess whether the patient appears to be in distress secondary to pelvic or abdominal pain. A pale, diaphoretic patient who presents with vaginal bleeding and abdominal or pelvic pain is concerning for hemoperitoneum, and requires more urgent and aggressive intervention than a patient who appears well or minimally uncomfortable.

Vital signs

Blood pressure and pulse are key vital signs. However, young healthy patients may become symptomatic from significant blood loss (dizzy or near-syncopal) before their vital signs become abnormal. Fever may imply an infectious etiology for the patient's symptoms.

Abdomen

The abdomen should be evaluated thoroughly for signs of hemoperitoneum. Distention, rigidity, rebound, guarding and pain localization should be assessed.

Pelvic

Speculum examination

A speculum examination is an essential part of the assessment of AUB due to any etiology. However, information can be obtained by visual inspection of the external genitalia prior to the insertion of the speculum. Assess the vulva, anus and urethra for non-uterine sources of bleeding and areas of infection or inflammation. Upon speculum insertion, cervical gonorrhea and chlamydia cultures and a vaginal wet mount should be obtained. While these are collected, evaluate for foreign bodies, masses, vaginal synechiae, or lacerations, and note the quality and quantity of blood or clots in the vaginal vault. Bright red blood implies ongoing bleeding, whereas brownish discharge is more consistent with bleeding that has already stopped. The cervix should be inspected for evidence of trauma, active bleeding, expulsion of tissue, or masses. If the cervix is difficult to visualize due to bleeding or discharge, a ringed forceps with a small piece of gauze may be gently inserted to clean the area.

Bimanual examination

A careful bimanual examination is part of the evaluation of a patient presenting with vaginal bleeding. It is performed to assess the uterus and adnexa for size and tenderness, as well as the status of the internal cervical os in a pregnant patient. The external cervical os may remain slightly open to fingertip in any woman who has delivered a baby. However, the internal cervical os only opens (dilates) when uterine contents are in the process of being expelled. The internal os is examined by gently inserting the gloved index finger through the external os. If there is the sensation that the finger enters the opening without resistance, the internal os is open. If the finger is stopped by closed tissue, the internal os is closed. After assessing for patency, the cervix should be held between the index and middle fingers of the examining hand and gently moved from side to side to assess for cervical motion tenderness (CMT). This may be found in conditions that involve peritoneal or pelvic floor inflammation or irritation, although it is nonspecific. Next, the uterus is palpated between the examiner's intravaginal fingers and the hand on the patient's suprapubic area. The uterine size, shape, position and contour are assessed, as well as its degree of tenderness. Uterine size may be estimated by comparison to common standard-sized objects, such as a golf ball (normal, non-pregnant), baseball (4–6 weeks gestational age), or softball (6–8 weeks gestational age). Lastly, the adnexa are palpated to assess for ovarian size, tenderness and masses. The ovaries are normally non-palpable in postmenopausal women, and are the size of walnuts in premenopausal women.

In order to maximize the yield from the pelvic examination, the patient should be as comfortable as possible. If the patient is having significant abdominal or pelvic pain, it is prudent to medicate the patient with appropriate analgesia before performing this examination. The choice of analgesic agent should take into consideration whether the patient is pregnant or lactating. Agents that alter clotting function (aspirin and nonsteroidal antiinflammatory agents, such as ketorolac and ibuprofen) should be avoided. Although no studies specifically address the safety and efficacy of premedicating a woman in distress before pelvic examination, literature clearly supports this practice in patients with abdominal pain before abdominal examination. Thus, appropriate analgesia should not be withheld. Additionally, although a bimanual examination is routine in this setting, data suggest poor inter-examiner reliability of the bimanual pelvic examination, questioning the utility of this portion of the examination in a non-pregnant female.

Auscultation for fetal heart tones

Fetal heart tones (FHTs) should be heard using a hand-held Doppler (placed over the suprapubic area) if the fetus is at least 12 weeks estimated gestational age (EGA). FHTs may be audible at 10 weeks EGA, but no cause for concern should arise unless they are not appreciated at 12 weeks EGA, provided a woman's dates are accurate. A normal fetal heart rate is between 120 and 160 beats per minute (bpm). It is important to document the presence, rate and location of the FHTs if the patient is > 12 weeks EGA, which verifies both the gestation's dates and viability at that point in time.

Differential diagnosis

Table 43.3 Differential diagnosis of vaginal bleeding in first-trimester pregnancy

Diagnosis	Symptoms	Signs	Work-up
Blighted ovum (anembryonic gestation, embryonic failure)	Varied	Internal cervical os open or closed	Serum β-hCG (declining or plateau, if there is a previous level for comparison), CBC, EVUS (gestational sac too big not to have embryo [i.e., >20 mm]), type and Rh, UA
Completed miscarriage	Vaginal bleeding ± abdominal pain	Benign examination, internal cervical os closed	Serum β-hCG (declining, if there is previous level for comparison), CBC, EVUS (empty uterus), type and Rh, UA
Ectopic pregnancy	Varied: vaginal spotting or bleeding, and/or abdominal pain, syncope or near-syncope possible	Varied: benign pelvic and abdominal examination, or tachycardia, hypotension, abdominal rigidity, rebound and/or guarding, adnexal mass and/or tenderness	Serum β-hCG (level correlates with EVUS findings), CBC, EVUS (varied: normal with empty uterus, or adnexal or intrauterine findings consistent with ectopic pregnancy), type and Rh
Embryonic demise (missed miscarriage)	Varied	Internal cervical os closed	Serum β-hCG (declining or plateau, if there is a previous level for comparison), CBC, EVUS (embryo lacking cardiac activity with CRL >5 mm), type and Rh, UA
Gestational trophoblastic disease (molar pregnancy)	Vaginal bleeding or spotting, nausea/vomiting	Tremor, tachycardia	Serum β-hCG (may be greater than expected for EGA), serum TSH, EVUS ("snowstorm" appearance)
Implantation bleeding	Minimal vaginal spotting near the time of a missed period	Benign examination	Serum β-hCG, CBC, EVUS (empty uterus seen if EGA 3–5 weeks), type and Rh, UA
Incomplete miscarriage	Vaginal bleeding or spotting and/or abdominal pain	Benign or tender pelvic examination, internal cervical os open or closed	Serum β-hCG (declining or plateau, if there is a previous level for comparison), CBC, EVUS (thickened, irregular endometrium >5 mm double stripe), type and Rh, UA
Inevitable miscarriage (miscarriage in progress)	Vaginal bleeding or spotting and/or abdominal pain	Benign or tender pelvic examination, internal cervical os open	Serum β-hCG (declining or plateau, if there is a previous level for comparison), CBC, EVUS (retained POC), type and Rh, UA
Septic miscarriage	Varied: vaginal bleeding or spotting, fever, abdominal pain, foul vaginal discharge	Internal cervical os open or closed, abdominal tenderness, peritoneal signs, CMT, foul cervical discharge	Serum β-hCG (declining or plateau, if there is a previous level for comparison), CBC, blood and cervical cultures, EVUS (thickened, irregular endometrium >5 mm double stripe), type and Rh, UA
Threatened miscarriage	Vaginal bleeding or spotting and/or abdominal pain	Internal cervical os closed, pelvic and abdominal examination benign or mild-moderately tender	Serum β-hCG, CBC, EVUS (empty uterus [3–5 weeks], embryo with cardiac activity or empty gestational sac [5–6.5 weeks], or subchorionic hemorrhage with any of the above), type and Rh, UA

CBC: complete blood count; CMT: cervical motion tenderness; CRL: crown-rump length; EGA: estimated gestational age; EVUS: endovaginal ultrasonography; hCG: human chorionic gonadotropin; POC: products of conception; TSH: thyroid-stimulating hormone; UA: urinalysis.

Table 43.4 Differential diagnosis of vaginal bleeding in non-pregnant female

Diagnosis	Symptoms	Signs	Work-up
Abnormal uterine bleeding due to exogenous gonadal steroids	Breakthrough bleeding with first three cycles of using OCP preparations Post-coital emergency contraception Irregular bleeding or delayed menses Menorrhagia Metrorrhagia Oligomenorrhea	Benign examination, although orthostatic hypotension or symptomatic anemia may result if bleeding is prolonged or heavy	Urine qualitative pregnancy test, CBC (if clinically indicated)
Abnormal uterine bleeding due to IUD	Menorrhagia Metrorrhagia Oligomenorrhea (if progestin-impregnated IUD)	Benign examination, although orthostatic hypotension or symptomatic anemia may result if bleeding is prolonged or heavy	Urine qualitative pregnancy test, CBC (if clinically indicated)

(continued)

Table 43.4 Differential diagnosis of vaginal bleeding in non-pregnant female *(cont.)*

Diagnosis	Symptoms	Signs	Work-up
Dysfunctional uterine bleeding (anovulatory)	Irregular vaginal bleeding in timing and in quantity, including oligomenorrhea or amenorrhea (no menses for 6 months), metrorrhagia, history of psychologic stress, weight gain, weight loss	Acne, obesity, hirsutism	Urine qualitative pregnancy test, CBC, estrogen level, 17-OH progesterone level, free testosterone level, prolactin level, TSH
Dysfunctional uterine bleeding (ovulatory)	Cyclic and predictable heavy menstrual bleeding (menorrhagia), fatigue	Anemia	Urine qualitative pregnancy test, CBC
Endometrial hyperplasia and carcinoma of the vulva, vagina, cervix and endometrium	Menorrhagia Metrorrhagia Oligomenorrhea Constitutional symptoms (if carcinoma is etiology)	Benign examination or mild pelvic tenderness	Urine qualitative pregnancy test, CBC (if clinically indicated), EVUS, endometrial sampling
Uterine leiomyoma	Menorrhagia Metrorrhagia	Benign examination or mild pelvic tenderness; orthostatic hypotension or symptomatic anemia if bleeding excessive or prolonged	Urine qualitative pregnancy test, CBC (if clinically indicated), EVUS
Vaginal trauma	Vaginal bleeding, pain, spotting, discharge	Vaginal laceration or foreign body	Urine qualitative pregnancy test, CBC (if clinically indicated)
Vaginitis/cervicitis	Vaginal bleeding or spotting, especially after intercourse, insertion of diaphragm, or pelvic examination; discharge	Cervical or vaginal wall friability	Urine qualitative pregnancy test, cervical cultures, wet mount

CBC: complete blood count; EVUS: endovaginal ultrasonography; IUD: intrauterine contraceptive device; OCP: oral contraceptive pills; TSH: thyroid-stimulating hormone.

Diagnostic testing

Laboratory studies

Urine pregnancy test

A urine qualitative pregnancy test is absolutely necessary in the evaluation of *any* woman of reproductive age with abnormal vaginal bleeding. The sensitivity for diagnosing pregnancy is 99.4%, so it is extremely useful for ruling out pregnancy by approximately the same date or possibly a few days before a woman misses her period (when the serum β-hCG is > 25 mIU/mL). False-negative tests occur when the serum β-hCG is between 10 and 50 mIU/mL, and also when the urine is dilute (specific gravity < 1.015). This may be overcome by using 20 drops of urine instead of the usual 5 drops to super-concentrate the hormone on the test diaphragm. The urine pregnancy test establishes the patient as pregnant, and should be ordered for all patients with vaginal bleeding, regardless of the patient's menstrual history or denial of sexual activity.

Urinalysis

A urinalysis with urine culture per protocol should be ordered in all pregnant patients with vaginal bleeding to diagnose UTI, regardless of symptoms. Generally, a catheterized specimen is best in this setting given the difficulty in obtaining a true "clean catch" in a woman with vaginal bleeding. UTIs are an etiologic risk factor for miscarriage. Also, asymptomatic bacteriuria and pyuria are relatively common in pregnancy, occurring in 2–11% of pregnant women. Up to one-fourth of these asymptomatic women will go on to develop upper tract urinary infections. Therefore, UTIs in pregnant patients should be identified and treated to potentially prevent miscarriage.

Complete blood count

A complete blood count (CBC) should be obtained routinely in patients presenting with vaginal bleeding in order to estimate how much they have bled prior to arrival in the ED. This also serves as a baseline hematocrit (HCT) for comparison if serial HCTs are obtained during the patient's course, especially if changes in hemodynamic status occur.

Serum qualitative beta-human chorionic gonadotropin

A serum qualitative β-hCG provides a "yes or no" answer to whether or not the patient is pregnant. Because the urine qualitative β-hCG test is often a point-of-care test and is extremely sensitive, the serum qualitative pregnancy test has a limited role. Consider this in a patient unable to provide a urine specimen, or as an efficiency measure in some EDs if blood is being drawn for other purposes.

Serum quantitative beta-human chorionic gonadotropin

The serum quantitative β-hCG is a measure of trophoblastic tissue activity, a marker for the volume of living trophoblastic tissue. Both ectopic and intrauterine pregnancies (IUPs) produce β-hCG, though they usually differ in the rate at which quantitative β-hCG increases. Patients with ectopic pregnancy tend to have a lower quantitative β-hCG than those with viable IUPs at the same gestational age. Abnormal IUPs may also have lower β-hCGs than normal IUPs.

Due to the large range of acceptable β-hCG levels for each stage of embryonic development, a single value of β-hCG is not useful for differentiating between normal IUP, abnormal IUP, and ectopic pregnancy. Variation in the expected rate of rise of the β-hCG level can be helpful to clinicians. For levels < 10,000 mIU/mL, the β-hCG normally doubles in 1.9 ± 0.5 days. Also, an increase of ≥ 66% over 48 hours is seen in 85% of normal IUPs. An abnormal increase in β-hCG, < 66% over 48 hours, is 75% sensitive and 93% specific for an abnormal gestation of some variety. Additionally, 85% of ectopic pregnancies and 15% of normal IUPs have an abnormal rate of rise of β-hCG. Declining β-hCG levels indicate a non-viable fetus, either ectopic or intrauterine. The rate of fall of the β-hCG has been found to differ significantly between these two entities. The half-life of the β-hCG is > 7 days in ectopic pregnancy, whereas it is < 1.4 days in failing IUPs. A falling β-hCG does not exclude the possibility of tubal rupture, and there is no minimum value of β-hCG that precludes rupture. The quantitative serum β-hCG is also useful in assisting with ultrasound interpretation (Table 43.5), and with monitoring response to medical management with methotrexate.

Table 43.5 Sonographic findings in early pregnancy

EVUS landmarks of early pregnancy	EGA (weeks)	Serum quantitative β-hCG (mIU/mL)
Gestational sac	4.5	1,500
Yolk sac	5.5	1,000–7,500
Embryo with cardiac activity	6.5	7,000–23,000

EGA: estimated gestational age; EVUS: endovaginal ultrasonography; hCG: human chorionic gonadotropin.

Rh type

Routine screening for Rh status in a pregnant vaginal bleeding patient is controversial. It has been established that completed miscarriage, ectopic pregnancy, antepartum hemorrhage and trauma are associated with possible feto-maternal transfusion, and thus potential for Rh isoimmunization if the mother is Rh-negative and the fetus is Rh-positive. Evidence for the same concept in threatened miscarriage is equivocal. However, because ED patients may not have access to or seek follow-up if they complete their miscarriage, it is prudent to administer Rh immune globulin to prevent isoimmunization in an Rh-negative patient during the ED visit. It is therefore the standard of care to give Rh immune prophylaxis to Rh-negative, pregnant women with vaginal bleeding. If the gestational age is less than 12 weeks, an intramuscular dose of 50 mcg RhoGAM is sufficient. However, as pregnancy dating is difficult and often inaccurate, it is recommended that all unsensitized Rh-negative women with vaginal bleeding receive 300 mcg of Rh immune globulin in the first or second trimester. This should be given before the patient leaves the ED, although protection occurs if RhoGAM is administered within 72 hours of bleeding. It is not necessary to repeat the dosage at subsequent ED or clinic visits for continued or repeat bleeding before 20 weeks gestation. A subsequent 300 mcg dose should be administered in the third trimester or prior to delivery.

Radiologic studies

Ultrasound

The value of pelvic endovaginal ultrasonography (EVUS), also known as transvaginal ultrasound, in the evaluation of a pregnant vaginal bleeding patient is to confirm the presence of an IUP, which ostensibly excludes the diagnosis of ectopic pregnancy. An understanding of what is necessary to make the sonographic diagnosis of an IUP is important for the clinician to optimally use the information. The hormones of pregnancy cause an early uterine decidual reaction that may be seen by ultrasound shortly after a missed menses. However, this finding is nonspecific and occurs with both IUPs and ectopic pregnancies. The earliest sonographic landmark consistent with an IUP is the gestational sac. With EVUS, this can be visualized as early as 4.5 weeks after the LMP (reliably by 5 weeks). The gestational sac lies eccentrically within the decidua of the endometrium, and has two distinct sonographic layers: the decidua capsularis and decidua parietalis. These two layers give a sonographic appearance of two rings, called the *double ring sign*, that is diagnostic of an intrauterine gestational sac (Figure 43.2). The yolk sac seen within

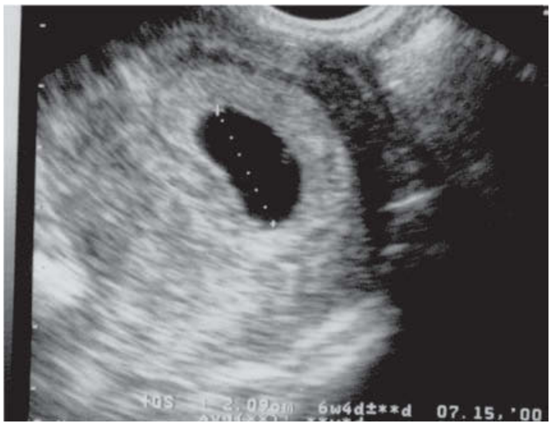

Figure 43.2
Double ring sign seen with EVUS in early first-trimester pregnancy.

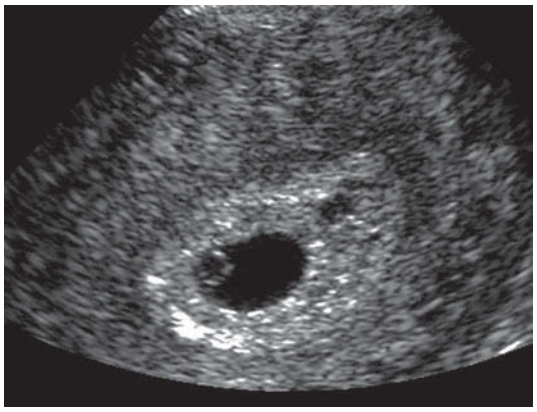

Figure 43.3
Normal intrauterine pregnancy. Sagittal endovaginal US of a normal first-trimester pregnancy. Note echogenic gestational sac within the uterus containing a yolk sac.

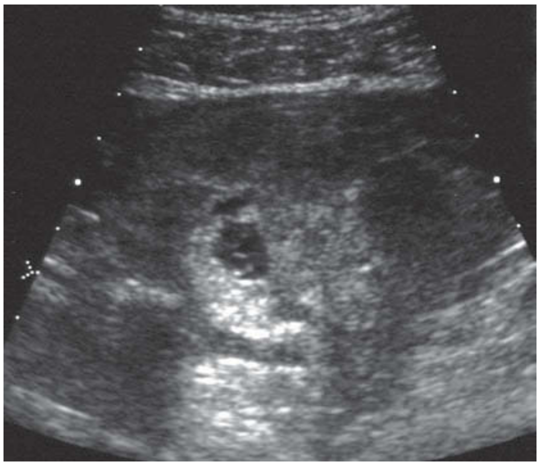

Figure 43.4
Ectopic pregnancy. Transverse scan of right adnexa demonstrating echogenic gestational sac containing a yolk sac within an ectopic pregnancy. Courtesy: R. Brooke Jeffrey, MD.

the gestational sac is the next sonographic landmark of developing pregnancy, seen reliably by the end of the fifth week (Figure 43.3). The embryo and cardiac activity are seen concurrently and reliably adjacent to the yolk sac by 6.5 weeks gestation using EVUS. Table 43.5 lists sonographic findings of early pregnancy development with their corresponding gestational ages and discriminatory levels of β-hCG.

The sonographic finding that is most reassuring for a favorable prognosis is the presence of embryonic cardiac activity. For women under 35 years of age at 8 weeks EGA, the presence of sonographic cardiac activity suggests a rate of spontaneous miscarriage of only 3–5% overall. This increases to about 8% for women over 35 years of age. Sonographic findings that foreshadow a poor outcome include a slow embryonic heart rate (<90 bpm), small gestational sac for the size of the embryo, and large yolk sac (>6 mm).

Differentiation between complete and incomplete miscarriage can be challenging if the cervical os is closed, bleeding is not heavy, and the patient is not appreciably tender on examination. In this setting, EVUS is a useful adjunct to make the diagnosis of completed miscarriage based on the presence of an empty uterus.

Specific findings suggestive or diagnostic of ectopic pregnancy can be identified by EVUS in up to 79% of ED cases. Intrauterine findings suggestive of ectopic pregnancy include the intrauterine decidual reaction. This can be problematic, because 10–20% of the time the intrauterine decidual reaction forms a cystic shape resembling a sac. This is referred to as a *pseudogestational sac*, thought to represent blood surrounded by decidual cast. An empty uterus found in a pregnant woman is present in up to 20% of cases of ectopic pregnancy. Extrauterine findings on EVUS may also be consistent with ectopic pregnancy. The most common finding is a complex adnexal mass (Figure 43.4), seen in 60–90% of cases. Other findings include free fluid in the cul-de-sac (20–40%) and an ectopic embryo (25–35%). A completely normal pelvic ultrasound has been reported in approximately 20% of patients with proven ectopic pregnancies.

When EVUS is neither diagnostic of an IUP nor suggestive of an ectopic pregnancy, it is characterized as indeterminate. Interpretation of this result requires consideration of the clinical context. Depending on their clinical status and β-hCG level, these patients need to be followed closely by an obstetrician-gynecologist in order to reassess their clinical status and recheck their β-hCG level within 48 hours.

In the non-pregnant woman with vaginal bleeding, an EVUS is a useful adjunct to physical examination, particularly if an adequate pelvic examination cannot be performed or the patient has an abnormal pelvic examination (i.e., adnexal mass, enlarged uterus).

In postmenopausal women who are not taking HRT and have AUB, EVUS can be used to measure endometrial thickness. An endometrial stripe thickness of <4 mm reliably excludes endometrial neoplasm as the etiology of bleeding, eliminating the need for endometrial biopsy.

General treatment principles

As with all ED patients, treatment begins with a general assessment of the patient's hemodynamic status. Patients with vaginal bleeding may present in various clinical states that may change during their ED evaluation. Pregnant vaginal bleeding patients have extremely high potential for rapid change in hemodynamic status; therefore, frequent reassessment of these patients is prudent.

Volume replacement and antibiotics

Not all patients with vaginal bleeding require intravenous (IV) access or IV fluid replacement. However, most first-trimester pregnant patients with vaginal bleeding will undergo evaluation for ectopic pregnancy, which may become life-threatening due to exsanguination.

Therefore, patients should have an IV placed on initial assessment. The degree of volume replacement required varies depending on the patient's clinical status. Crystalloids (normal saline) are appropriate as initial resuscitation fluids, adding packed red blood cell transfusion if the patient's clinical status deteriorates despite aggressive volume replacement. As most of these patients are young and otherwise healthy, they should be able to tolerate significant anemia (i.e., hematocrit to 20%) before blood transfusion is needed. For women with history of or risk factors for cardiac ischemia, significant anemia is akin to a cardiac stress test and may not be well tolerated. Thus a different standard for transfusion in such patients is prudent.

Patients diagnosed with or suspected of having septic miscarriage should receive IV broad-spectrum antibiotics as soon as possible, with allergies and drug-drug interactions in mind.

Ectopic pregnancy

A thorough understanding of non-surgical management options for patients diagnosed with ectopic pregnancy is important for emergency physicians, because these options include discharging a patient from the ED with a potentially life-threatening surgical emergency. Medical management of stable patients with ectopic pregnancy is achieved with methotrexate, a folic acid antagonist that prevents the synthesis of amino acids, ribonucleic acid (RNA) and deoxyribonucleic acid (DNA). Methotrexate therapy thereby eradicates rapidly developing trophoblastic tissue. Both the decision to use and administer methotrexate should be made by the obstetrics/gynecology service, since this is not currently within the scope of emergency medicine practice.

Evidence-based criteria for methotrexate therapy:

- Hemodynamic stability
- EVUS showing an unruptured ectopic pregnancy with greatest diameter < 4 cm (<3.5 cm if cardiac activity present)
- No active bleeding or free fluid on ultrasound
- Stable or rising β-hCG titer < 5,000 mIU/mL
- β-hCG increasing after curettage
- Desired future fertility
- Patient does not desire surgical therapy
- Patient willingness and ability to return for serial follow-up assessments and β-hCG measurement
- Certain cases of cervical and cornual pregnancy

Contraindications to methotrexate therapy:

- Hepatic or renal dysfunction
- Active peptic ulcer disease
- Blood dyscrasias

The protocol for management with methotrexate involves a single intramuscular (IM) injection of 50 mcg/m^2 body surface area, with close obstetric-gynecologic follow-up and serial β-hCG levels. The success rate for resolving the ectopic pregnancy without surgical intervention is 85–100% using this protocol. However, it takes between 20 and 44 days for the β-hCG levels to become undetectable. The main complication of methotrexate therapy is tubal rupture, which occurs in about 4% of cases. Methotrexate has a variety of side effects, the most common of which is increased abdominal pain (in up to 60% of patients). This is problematic, as this symptom may be indistinguishable from tubal rupture. Patients who have been treated for ectopic pregnancy with methotrexate and complain of increasing abdominal pain should have an immediate ultrasound and obstetric-gynecologic consultation to evaluate for tubal rupture. Other side effects of methotrexate include nausea, vomiting and diarrhea, which occur in as high as 20% of cases.

Surgical management of ectopic pregnancy is reserved for patients who do not fall into the medical management group described above. This is generally done either by tube-sparing laparoscopic salpingotomy, salpingectomy, or through open laparotomy, which may be required for patients in extremis or with significant hemoperitoneum.

Missed and incomplete miscarriage

If tissue or blood clots are found in the cervical os, they should be gently removed with ring forceps to facilitate uterine contractions and hemostasis. Care should be employed to avoid trauma to vaginal and cervical tissue. For the same reason, oxytocin (10–20 mcg/L of normal saline, given wide open) may be used in cases of severe bleeding. Consider hemotransfusion for severe bleeding and/or hemodynamic instability if resuscitation with crystalloid is insufficient. Management of stable patients diagnosed with incomplete and missed miscarriages varies depending on the preferences of the patient and/or provider. Options include medical management with misoprostol to facilitate completion of tissue passage, surgical management with dilatation and curettage (D&C), or expectant management with close follow-up in a compliant patient. All three management options are medically acceptable and generally well tolerated by patients. Table 43.6 compares the three management options.

Abnormal uterine bleeding

Gonadal steroids are the first-line therapeutic option for vaginal bleeding in a non-pregnant patient. Immediate therapy for severe symptomatic bleeding in hemodynamically compromised patients requiring hospital admission may consist of high-dose conjugated estrogens. Starting doses of 25 mg IV every 4 hours for up to 48 hours usually results in the desired response within 5 hours. For stable patients, oral treatment with medroxy-progesterone 60–120 mg followed by 20 mg daily for 10 days is effective in halting AUB. Another alternative, especially for premenopausal patients, is combined estrogen and progestin oral contraceptive pills (OCP). A monophasic pill preparation (constant dosage throughout the pill-pack month) with at least 35 mcg of ethinyl estradiol is prescribed in the following

Table 43.6 Management options for first-trimester missed and incomplete miscarriage

Surgical curettage
- Bleeding stops in average of 9 days (missed miscarriage) and 7 days (incomplete miscarriage)
- May require admission or observation

Medical
- Misoprostol 800 mcg intravaginally or by mouth (PO)
- Repeat the dose in 1 day if not complete
- No proven benefit to "priming" with mifepristone (RU486)
- Outpatient
- Success rate 87% (missed miscarriage) and 100% (incomplete miscarriage)
- Bleeding stops in average of 11 days (miscarriage) and 9 days (incomplete miscarriage)

Expectant
- Success rate 29% (missed miscarriage) and 85.7% (incomplete miscarriage)
- Bleeding stops in average of 12 days (missed miscarriage) and 10 days (incomplete miscarriage)

Adapted from Bagratee JS, Khullar V, Regan L, et al. A randomized controlled trial comparing medical and expectant management of first trimester miscarriage. *Human Reprod* 2004;19(2):266–71; and Trinder J, Brocklehurst P, Porter R, et al. Management of miscarriage: Expectant, medical, or surgical? Results of randomised controlled trial (miscarriage treatment (MIST) trial. *BMJ* 2006;**332**(7552):1235–40.

manner: 3 pills/day for 3 days, then 2 pills/day for 2 days, then 1 pill/day until the package is finished. This regimen is known as an "OCP taper." Women should skip the placebo pills in the first pack and start the next pack of 1 pill/day to avoid withdrawal bleeding so soon after the previous bleeding episode. Ideally, estrogen therapy should not be initiated on patients until the diagnosis of endometrial carcinoma has been excluded. A general guideline is that for women over the age of 40 years, endometrial sampling should be obtained prior to or within a week of initiating hormone therapy. Additionally, OCPs and estrogens are not recommended for women with hypertension or who smoke; these medications should be used with caution, and only in consultation with a gynecologist.

Disposition

First-trimester vaginal bleeding

Patients in whom the diagnosis of ectopic pregnancy is made conclusively by EVUS require immediate GYN consultation, because either surgical or medical management is required. Management depends on the patient's hemodynamic stability, and whether she meets criteria for methotrexate therapy.

Patients with first-trimester bleeding, a β-hCG <1,500 mIU/mL, and an indeterminate EVUS may be sent home from the ED with a diagnosis of "possible ectopic pregnancy" if they are hemodynamically stable. These patients require close follow-up and a repeat β-hCG measurement in 48 hours by their OB/GYN physician. It is appropriate for these follow-up arrangements to be made prior to ED disposition in order to ensure compliance with the recommendation. When discharging patients from the ED, it is essential that they comprehend the potentially life-threatening nature of their condition, understand specific instructions for when to return to the ED in advance of their scheduled follow-up, and have access to transportation (driver) or a telephone (to call emergency medical services). Reasons to return to the ED include dizziness, syncope, increased abdominal pain, heavy vaginal bleeding greater than one pad per hour, or fever. Patients should also be advised to remain at pelvic rest (nothing inside the vagina), maintain hydration status, and gradually resume a bland diet. There is no evidence that bed rest is necessary.

Patients diagnosed with inevitable or septic miscarriage require immediate GYN consultation for possible surgical intervention (and broad-spectrum antibiotics in the case of septic miscarriage).

Patients with threatened and completed miscarriage do not necessarily require GYN consultation in the ED. In patients in whom ectopic pregnancy has been excluded, follow-up with their OB/ GYN in 1–2 weeks is appropriate. Careful discharge instructions with return precautions described above should be given and understood.

Patients with incomplete and missed miscarriages may be managed differently in different practice environments. Some gynecologists and patients prefer expectant management with arranged follow-up in 1–2 weeks for possible elective D&C, whereas others prefer to perform semi-urgent D&C at the time of initial diagnosis. However, increasingly more incomplete miscarriages are managed medically with misoprostol. Whenever possible, patient preference should be considered regarding urgent D&C versus expectant management of incomplete miscarriage. Current literature suggests that all three management options are well accepted by patients.

It is important to discuss the common nature of spontaneous miscarriage with the patient, emphasizing to her (and her partner, if appropriate) that she did nothing to precipitate it. Many women will have a significant emotional response to a miscarriage, blaming themselves, what they ate, or what they may have done (such as exercise, intercourse, falling, or lifting something). A few minutes of reassurance and correct information from a caring provider can frame the situation in a way that facilitates emotional healing. Additionally, remember that pregnant women are at increased risk of intimate partner violence, either causing the miscarriage or resulting from it.

Non-pregnant abnormal uterine bleeding

Patients with AUB typically do not require urgent GYN consultation unless they require admission to the hospital due to severe anemia or acute hemorrhage. Women over 40 years of age for whom hormone therapy is indicated should have endometrial sampling either prior to or within 1 week of hormone initiation. Because this procedure generally falls outside of the scope of emergency medicine practice, specific follow-up should be arranged from the ED. Patients under 40 years of age may begin outpatient management with hormone therapy, with gynecology follow-up arranged in 1–2 weeks.

Specific ED return precautions include symptoms of severe anemia (excessive fatigue, orthostasis, syncope), increased bleeding, or intractable pain. Patients should contact and follow up with their gynecologist or primary care provider if the bleeding does not improve within 3–5 days of therapy.

Pearls, pitfalls and myths

- Obtain a pregnancy test on *every* woman of childbearing age with vaginal bleeding, abdominal or pelvic complaints. A negative history for pregnancy and/or sexual activity is unreliable, and the differential diagnosis, evaluation and treatment are based on whether or not the patient is pregnant.
- Every patient with first-trimester bleeding without a documented IUP must have ectopic pregnancy ruled out. This includes a quantitative β-hCG and EVUS in the ED. Appropriate follow-up *must* be arranged for those with indeterminate results. There is no β-hCG level below which clinicians should not consider ectopic pregnancy or not obtain an ultrasound. Over 80% of patients with ectopic pregnancy have a β-hCG < 1,000 mIU/mL, and 79% of ED patients with first-trimester bleeding will be diagnosed in the ED by EVUS.
- Patients diagnosed with ectopic pregnancy by EVUS may be candidates for outpatient methotrexate treatment if they meet strict criteria and have no contraindications. However, the decision to treat patients with methotrexate should be made by the consulting gynecologist.
- Patients treated with methotrexate must be able to return to the ED immediately should they experience increased abdominal pain, because methotrexate does not alter the risk of rupture for ectopic pregnancy (4%) in the short term (approximately 1 week). When these patients return to the ED with increased abdominal or pelvic pain, an EVUS and gynecology consultation should be obtained immediately to rule out ruptured ectopic pregnancy.
- Patients on ovulation-induction agents are at significantly increased risk for heterotopic pregnancy, as high as 1 in 34 pregnancies. It is therefore essential to specifically ask every patient if she has taken fertility medications.
- Patients diagnosed with intrauterine fetal demise (also referred to as missed miscarriage) do not require an emergent D&C. It is appropriate to manage these patients expectantly, if desired by the patient and the consulting gynecologist.
- Patients diagnosed with incomplete miscarriage by examination and/or EVUS are excellent candidates for medical management with misoprostol.
- Patients with possible septic miscarriage should receive prompt IV broad-spectrum antibiotics, even before the confirmatory EVUS is obtained or gynecology consultation arrives, as these infections may progress very rapidly.

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44 Vomiting

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Scope of the problem

Vomiting is a common presenting complaint to the emergency department (ED), accounting for over 2 million annual visits. It is one of the top five reasons for ED visits among children. Vomiting has no age or gender predilection, and has many etiologies. The most common causes of vomiting are acute gastroenteritis, febrile systemic illness and drug-related effects. The emergency physician's main responsibility is differentiating emergent and life-threatening causes of vomiting from those caused by benign entities. The remainder of ED management is focused on symptom relief and rehydration.

Pathophysiology

Vomiting is induced by physical stimulation of the back of the throat (gag reflex), mucosal irritation of the upper digestive tract, stimulation of the vomiting center in the medulla oblongata, stimulation by biochemical emetic stimuli on the chemoreceptor trigger zone in the area postrema, or severe emotion. Nausea frequently precedes vomiting, marked by reduced gastric tone and peristalsis. Regurgitation, by contrast, is the passive retrograde movement of esophageal contents into the mouth, as occurs in gastroesophageal reflux.

True vomiting refers to the rapid ejection of gastric contents. The abdominal muscles rapidly contract, while the cardia of the stomach, esophagus and throat remain open with the glottis closed. Copious salivation usually precedes vomiting, serving to lubricate the digestive tract and dilute the gastric acid. Repeated contractions of the abdominal muscles against a relaxed stomach produce *retching*. The repetitive abdominal contractions build up a pressure gradient in the stomach prior to vomiting. This allows gastric contents to move to the upper portion of the stomach. Retching may also occur without expulsion of gastric contents, referred to as "dry heaves." Repeated vomiting can lead to hypovolemia, metabolic alkalosis and hypokalemia. A forceful episode of vomiting or retching can lead to gastrointestinal (GI) bleeding (i.e., Mallory–Weiss tear) or esophageal perforation (Boerhaave's syndrome).

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 44.1).

History

What did you vomit (food, blood, mucus)?

The composition of the vomitus (emesis) is important for determining both the etiology of vomiting and treatment. If the vomitus contains coffee-ground material, this most often represents blood that has been exposed to the stomach's acidic environment. If the blood is bright red, an esophageal or vascular source of bleeding is likely. It is important to determine the volume of blood and whether it occurred initially with vomiting or after several episodes of vomiting. Mallory–Weiss tears generally occur following several episodes of forceful vomiting. Vomitus containing undigested food may be seen with gastric outlet obstruction, gastric retention and psychogenic vomiting. Vomitus with mucus must be differentiated from coughing or spitting up phlegm. Other potential contents of vomitus include bile, often associated with small bowel obstruction, and feculent material, associated with large bowel obstruction.

Was there anything that preceded or is associated with the vomiting (coughing, abdominal pain)?

Vomiting does not mean the same thing to all people. Frequently, patients will cough, which evokes a gag reflex, causing them to "vomit." Vomiting as a consequence of severe coughing is known as post-tussive emesis. Abdominal pain may follow vomiting, and may be related to forceful abdominal muscle contraction. Vomiting that follows the development of abdominal pain is nonspecific; however, pain relieved by vomiting may suggest gastric outlet obstruction. A report of vomiting prior to chest pain may be an ominous sign, likely due to a non-GI cause.

How many times did you vomit?

Although the exact answer is not necessary, this provides an indication of whether the vomiting occurred only once or several times. If the patient has vomited several times, the likelihood of dehydration or underlying pathology (e.g., Mallory–Weiss tear) is increased.

When did the vomiting occur?

Vomiting in the morning is more common during pregnancy and uremia. Vomiting associated with increased intracranial pressure (ICP) may be more frequent when ICP is higher, such as in the morning or with bending movements. Vomiting less than 1 hour after eating may be related to gastroparesis or gastric outlet obstruction.

Table 44.1 Vomiting red flags

History	Concerning diagnosis
Hematemesis	Severe anemia, shock, Mallory-Weiss tear, gastric perforation
Suicidal ideation, severe depression, overdose	Acetaminophen and/or aspirin ingestion
Headache or head trauma	Intracranial hemorrhage, brain tumor, increased ICP, meningoencephalitis
Previous abdominal surgeries, unable to pass gas	Bowel obstruction, adhesions, hernia
Vertigo	Brainstem or cerebellar lesion
Pregnancy	Hyperemesis gravidarum
Bilious vomiting (child)	Intestinal malrotation (volvulus), intussusception
Projectile vomiting (infant)	Pyloric stenosis
Ill contacts, flu season	Gastritis, gastroenteritis (associated diarrhea), viral syndrome
Alcohol abuse	Alcoholic ketoacidosis, pancreatitis, hepatitis
Insulin-dependent diabetes	Diabetic ketoacidosis
Hematuria	Abdominal aortic aneurysm, ureteral colic
Elderly	CNS process, electrolyte disorder, GI obstruction (mass, volvulus), myocardial infarction
Examination finding	**Concerning diagnosis**
Tachycardia or hypotension, dry mucous membranes, poor skin turgor	Acute dehydration, hypovolemic shock
Nystagmus, anisocoria, papilledema, focal neurologic findings, confusion or lethargy	CNS insult or disease process (including hemorrhage, mass, increased ICP, uncal or transtentorial herniation, meningoencephalitis, meningitis)
Abdominal surgical scars, distended abdomen, tympany, borborygmi	Bowel obstruction (small or large bowel), volvulus
Tenderness or mass on pelvic examination	Ovarian torsion, PID/TOA
Fecal impaction	Tumor, Hirschsprung's disease, bowel obstruction
Abnormal testicular examination or lie	Testicular torsion
Abdominal tenderness	Cholelithiasis, cholecystitis, appendicitis, bowel obstruction, perforated viscus
Gravid uterus, Chadwick's sign	Pregnancy, hyperemesis gravidarum
Altered behavior or level of consciousness	Alcohol or drug intoxication, CNS insult or disease process

CNS: central nervous system; GI: gastrointestinal; ICP: intracranial pressure; PID: pelvic inflammatory disease; TOA: tubo-ovarian abscess.

Have long have you been vomiting?

The duration of vomiting may provide a clue to the diagnosis. *Acute* vomiting (<1 week) is typically associated with obstructive, ischemic, toxic, metabolic, infectious, neurologic and postoperative causes. Chronic vomiting (>1 week) occurs with motility disorders, partial obstructions or neurologic conditions, or may be related to pregnancy or a functional disorder.

Have you been able to tolerate fluids between the vomiting episodes?

This question helps to assess the patient's hydration status.

Associated symptoms

Gastrointestinal

Ask about abdominal pain, diarrhea, constipation, or inability to pass gas. If constipation or the inability to pass gas accompanies vomiting in a patient with prior abdominal surgery, bowel obstruction must be considered. Vomiting preceded by abdominal pain has a broad differential. Abdominal pain temporarily relieved by vomiting suggests delayed gastric emptying, as with gastroparesis from diabetes or gastric outlet obstruction. When vomiting is associated with diarrhea, an infectious etiology is commonly responsible.

Neurologic

Ask about headache, dizziness, vertigo, weakness, numbness or tingling of the extremities. Serious causes of vomiting include subarachnoid hemorrhage and increased ICP from obstruction, tumor, or infection. Any cause of increased ICP can cause vomiting with or without preceding nausea by direct interaction with vomiting centers in the area postrema. Causes of vertigo (e.g., labyrinthitis, Ménière's disease) produce vomiting by stimulation of the vestibular nuclei and subsequent stimulation of brainstem nuclei. Vomiting is a common symptom of migraine headache. However, a sudden-onset, severe or atypical headache accompanied by vomiting may represent a subarachnoid hemorrhage. Meningitis may also present as headache and fever associated with vomiting.

Genitourinary (in females)

Ask about dysuria, back pain, vaginal discharge, vaginal bleeding, lower abdominal cramping, sexual history, history of sexually transmitted infections (STI) and pregnancy. Vomiting is associated with pyelonephritis, pelvic inflammatory disease, tubo-ovarian abscess, ovarian torsion, ectopic pregnancy and normal first-trimester pregnancy.

Genitourinary (in males)

Ask about testicular or groin pain. Testicular torsion or an incarcerated inguinal hernia can present with vomiting and groin or testicular pain. Kidney stones (nephrolithiasis) at the ureterovesical junction (UVJ) and ureteral colic cause pain in the groin or testicle and vomiting.

Cardiopulmonary

Ask about chest pain, cough and fever. Vomiting can be associated with myocardial infarction. Remember not all vomiting is true vomiting; the patient may have phlegm production from pneumonia.

Other

Ask about other symptoms. These include recent chemotherapy if the patient has cancer, ingestions if the patient is depressed or suicidal, other family members who may be vomiting if the illness is acute, recent trauma to the abdomen or head, what the patient ate (e.g., home canned goods, raw fish, food at a picnic), and atypical hobbies (e.g., foraging for wild mushrooms).

Past medical

The patient's medical history is an important adjunct to determining the source of vomiting. Common conditions associated with vomiting include hypertension, liver disease, cholelithiasis, excessive alcohol use, previous surgeries, recent head or abdominal trauma, thyroid disorders, renal insufficiency or failure, chemotherapy, diabetes, pregnancy, cardiac disease, or peripheral vascular disease. A thorough medication list of all current and recent medications, including over-the-counter, herbal and "natural" medicines, must be obtained.

Physical examination

A systematic physical examination is very important, as several etiologies of vomiting involve more than one anatomic system. The history of present illness, medical history and associated symptoms will help guide which portions of the examination need increased attention.

General appearance

The patient's general appearance can range from normal to severe distress as a result of discomfort, continued vomiting, dehydration, or the underlying cause of vomiting. Pallor, coolness of extremities, and even a slight "greenish" or "grayish" overall appearance are common during vomiting.

Vital signs

Attention to the patient's pulse and blood pressure is essential in determining the patient's hydration status. However, the absence of tachycardia or hypotension does not exclude significant dehydration or disease. Also, orthostatic changes in blood pressure or heart rate do not reliably determine the patient's volume status. A patient with symptomatic orthostasis (dizzy or lightheaded with standing) may have significant fluid loss. The presence of fever may indicate an infectious or inflammatory response, or a toxic ingestion requiring prompt evaluation. If the patient is pregnant, fetal heart tones should be documented if possible.

Head, eyes, ears, nose and throat

Head

Examine the head for hematomas and bruising.

Eyes

Visual acuity should be measured in patients with vomiting and visual complaints to screen for conditions such as acute angle closure glaucoma. Visual fields should also be tested to determine the presence of a deficit. Examination of the pupils should be performed to look for symmetry and normal reactivity. Extraocular movements should be evaluated; nystagmus may be associated with vestibular disease or a posterior fossa abnormality. A funduscopic examination is necessary to look for papilledema, which suggests increased ICP (e.g., brain tumor).

Nose

The nose should be inspected for evidence of bleeding if the patient complains of vomiting blood.

Mouth

Examine the mucous membranes for evidence of bleeding or dehydration. Loss of dental enamel may be associated with bulimia.

Neck

Examine the neck for abnormal enlargement of the thyroid, possibly associated with thyrotoxicosis.

Pulmonary

Examination of the lungs may identify evidence of pneumonia or aspiration associated with vomiting.

Abdomen

Evaluation of the abdomen will often be the most important part of the physical examination and should be performed on every vomiting patient. On inspection, the presence of previous surgical scars should be noted and their etiology determined. The abdomen should be observed for peristaltic waves when obstruction is suspected. Abdominal distention should be noted and may be due to excess fluid or air within the peritoneal cavity. Bowel sounds should be assessed. The presence of high-pitched, rushing bowel sounds (borborygmi) may indicate intestinal obstruction. Decreased bowel sounds may be associated with ileus. Localized tenderness or peritoneal signs will guide therapy and diagnostic studies. The abdominal examination may be difficult to interpret if the patient has had several episodes of emesis. Serial examinations or observation may be necessary to determine the etiology of vomiting.

Genital

Tenderness of the lower abdomen in female patients should be evaluated by a pelvic examination to identify cervical motion tenderness, adnexal masses, pus from the cervical os and vaginal bleeding. Male patients with persistent vomiting require a testicular examination to palpate for masses or tenderness, and to assure normal lie and size.

Rectal

A digital rectal examination should be performed on most patients to look for the presence of stool, bleeding, or a mass. This is particularly important if the patient has hematemesis (vomiting blood).

Neurologic

A complete neurologic examination should be performed on all patients with headache or suspicion of intracranial pathology associated with vomiting.

Extremity

Distal pulse presence, amplitude and symmetry should be noted. Any lower extremity edema should be noted and recorded.

Skin

Assess the patient's skin turgor when evaluating volume status. Jaundice, pallor, temperature, or flushing of the skin should be noted as well.

Differential diagnosis

The differential diagnosis for vomiting is summarized in Table 44.2.

Table 44.2 Differential diagnosis for vomiting

Diagnosis	Symptoms	Signs	Work-up
Acetaminophen toxicity	Anorexia, nausea, vomiting and malaise. Psychiatric history not always apparent.	May have normal physical exam or RUQ tenderness.	Serum acetaminophen level at 4 hours after ingestion. Poison control consultation.
Acute appendicitis	Epigastric or periumbilical pain migrates to RLQ over 8–12 hours (50%). Later presentations associated with higher perforation rates. Pain, low-grade fever (15%), and anorexia (80%) common; vomiting less common (50–70%).	Mean temperature 38°C (100.5°F). Higher temperature associated with perforation. RLQ tenderness (90–95%) with rebound (40–70%); rectal tenderness in up to 30%.	WBC usually elevated or may show left shift. Urine may show sterile pyuria. CT is sensitive and specific. US is preferred first imaging study in women and children with RLQ pain.
Acute gastroenteritis	Nausea and vomiting begin before pain. Pain usually poorly localized. Diarrhea is a key element in diagnosis; usually large volume, watery.	Abdominal examination nonspecific without peritoneal signs. Watery or no stool noted on rectal examination. Fever is often present.	Symptomatic care with antiemetics and volume repletion. Key is not assuming this diagnosis and missing serious disease.
Acute pancreatitis	Mid-epigastric or LUQ pain that is constant, boring and often radiates to the back, flank(s), chest, or lower abdomen. Usually severe but may be mild. Supine position exacerbates pain. Nausea and vomiting are common.	Patient usually in moderate distress. Low-grade fevers and tachycardia are frequently present. Patients may present in hypovolemic shock. Abdominal examination notable for epigastric tenderness. *Cullen's sign*, a bluish discoloration around the umbilicus, and *Grey-Turner's sign*, a bluish discoloration of the flanks, are late signs of hemorrhagic pancreatitis.	Serum lipase is the best test to diagnose pancreatitis. Serum amylase is neither sensitive nor specific, but more readily available.
Aspirin intoxication	Altered mental status, headache, tinnitus, abdominal pain, nausea and vomiting.	Tachypnea, abdominal tenderness.	Chemistry panel, LFTs, CBC, coagulation profile. Aspirin and acetaminophen levels.

(*continued*)

Table 44.2 Differential diagnosis for vomiting (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Biliary tract disease	Crampy RUQ pain radiates to right subscapular area. Prior history of pain is common.	Temperature normal in biliary colic. RUQ tenderness.	US demonstrates anatomy, stones, or duct dilatation. Hepatobiliary scintigraphy demonstrates gallbladder function. CT may demonstrate pathology.
Boerhaave's syndrome	Acute, severe, unrelenting and diffuse pain in the chest, neck and abdomen with radiation to the back and shoulders. Back pain may be the predominant symptom. Pain is often exacerbated by swallowing after 6–12 hours.	Patients appear ill. Tachycardia and tachypnea, abdominal rigidity with hypotension and fever often occur early. Cervical subcutaneous emphysema or *Hamman's crunch* (air in the mediastinum being moved by the beating heart) may be auscultated.	Initiated based on clinical suspicion of the diagnosis. CXR and water-soluble contrast esophagography most often make the diagnosis. Endoscopy and CT of the chest are useful adjuncts. Thoracentesis may identify GI contents, but not recommended.
Bowel obstruction	Presentation depends on the site of obstruction. Most patients have abdominal pain, described as crampy, intermittent and usually referred to the periumbilical area. Pain may be episodic, lasting a few minutes at a time. Bilious vomiting is often present if the obstruction is proximal. Inability to have a bowel movement or pass flatus is common.	Abdominal tenderness may be minimal and diffuse, or localized and severe. Abdomen may be tympanitic to percussion with active, high-pitched bowel sounds with occasional "rushes" on auscultation.	AAS usually suggests the diagnosis. If AAS is unclear, a CT may be ordered. Laboratory work includes CBC and electrolytes, but these are neither sensitive nor specific for the diagnosis.
Cholecystitis	RUQ pain that initially is persistent, not colicky, beyond 6 hours. Associated nausea, vomiting and anorexia. History of fever and/or chills is not uncommon. Patients may have either a history of similar attacks or documented gallstones.	Moderate to severe distress with signs of systemic toxicity including tachycardia and fever. RUQ abdominal tenderness. Generalized peritonitis is rare. *Murphy's sign* (worsened pain or inspiratory arrest resulting from deep, subcostal palpation on inspiration) is generally present.	WBC count, serum bilirubin, alkaline phosphatase and aminotransferase levels are often normal. Serum lipase or, if unavailable, amylase to rule out pancreatitis. Urine pregnancy test in females. UA to look for other cause of abdominal pain. US is the diagnostic modality of choice.
Diabetic ketoacidosis	Polyuria and polydipsia. Unexplained nausea, vomiting and abdominal pain are frequently seen, especially in children.	Abnormal vital signs may be the only physical findings at of presentation. Tachycardia and orthostasis or hypotension usually present. Kussmaul respirations with severe acidemia. Characteristic fruity odor on the breath found in some patients. Diffuse abdominal tenderness may also be present.	Serum glucose determination, chemistry panel, ABG or VBG to determine degree of acidemia. Work-up to determine concomitant infection or myocardial infarction. Fluid hydration and repletion of potassium.
GI bleed	Hematemesis, coffee-ground emesis, melena, or hematochezia. Vomiting and retching followed by hematemesis is suggestive of a Mallory–Weiss tear. A history of an aortic graft should suggest the possibility of an aortoenteric fistula. Alcohol, NSAIDs, and GI bleed history should be determined.	Altered mental status, hypotension, tachycardia, decreased pulse pressure or tachypnea. Paradoxical bradycardia can occur in the face of profound hypovolemia. Petechiae and purpura suggest an underlying coagulopathy. Tenderness, masses, ascites, or organomegaly on abdominal examination. Digital rectal examination needed to detect the presence of bright red, maroon, or melanotic stool.	The most important laboratory test is blood type and crossmatch. CBC, electrolytes, coagulation studies are also indicated.
Intussusception	6–18-month-old previously healthy infant. Intermittent episodes of severe abdominal pain. Vomiting is rare in the first few hours but usually develops after 6–12 hours. May present with lethargy.	Fever (as high as 41 C [106 F]). Respirations may be shallow and grunting. Apathy or lethargy may be the only presenting sign in up to 10% of cases. Classic "currant jelly" stool is a late manifestation and present in only 50% of cases. Examination between attacks may reveal sausage-shaped mass in the right side of the abdomen (66%).	Diagnosis considered on the basis of the history. AAS may reveal a mass or filling defect in the RUQ of the abdomen. Thirty percent of X-rays are normal. US may identify mass. Barium or air-contrast enema may be necessary, and possibly therapeutic.

(*continued*)

Table 44.2 Differential diagnosis for vomiting (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Mesenteric ischemia	Severe pain, colicky, starting in periumbilical region and becomes diffuse. Often associated with vomiting and diarrhea.	Early examination can be remarkably benign in the presence of severe ischemia. Bowel sounds often still present. Rectal examination is important because mild ischemia may present with only hemoccult-positive stools.	Pronounced leukocytosis usually present. Elevations of amylase and creatine phosphokinase levels can be seen. Metabolic acidosis due to lactic acidosis often seen with infarction. Plain films of limited benefit until late. CT, MRI and angiography are accurate to varying degrees.
Myocardial infarction	Chest pain, shortness of breath, or abdominal discomfort predominate. Associated symptoms include nausea, vomiting, diaphoresis, dyspnea, lightheadedness, syncope and palpitations. A detailed history is essential.	May appear well or exhibit signs of shock. An S3 is present in 15–20% of patients and may imply a failing myocardium. A new systolic murmur may signify papillary muscle dysfunction, a flail leaflet of the mitral valve with resultant MR, or a VSD. Rales are associated with LV dysfunction and left-sided CHF. JVD, HJR and peripheral edema suggest right-sided CHF.	ECG is the single best test to identify patients with AMI. Cardiac markers (CK-MB and troponin) should also be obtained.
Ovarian torsion	Acute, severe unilateral pain felt in the lower abdomen and pelvis; may be related to a change in position. May be intermittent. Nausea and vomiting are common.	Possible palpation of a mass on bimanual pelvic examination.	Pelvic US, CBC, urine pregnancy test.
Pelvic inflammatory disease	Abdominal pain, pain with intercourse. Nausea and vomiting are common.	Fever typically present. Lower abdominal tenderness common. Discharge from cervical os likely present. Cervical motion tenderness pronounced.	Cervical cultures for GC and Chlamydia, CBC. IV hydration, pain medication and antibiotics. Pelvic US to exclude a tubo-ovarian abscess.
Pregnancy	Nausea and vomiting, breast pain, weight gain.	May be normal. On pelvic examination, uterus is larger than normal.	Urine pregnancy test.
Pyloric stenosis	Infant (age >1 week but <3 months) with nonbilious projectile vomiting. Vomiting usually becomes projectile within a week of symptoms. Vomiting occurs just after or near the end of feeding; infant re-feeds as if never fed.	Hungry infant who has failed to gain weight over several weeks, or has lost weight and is dehydrated or lethargic. Peristaltic waves can sometimes be seen passing from left to right across the upper abdomen, just prior to an episode of vomiting. May palpate an “olive” (pyloric tumor near the lateral margin of the right rectus muscle just below the liver edge).	Abdominal US is recommended, chemistry panel and CBC. IV hydration and glucose administration, especially if ill-appearing.
Subarachnoid hemorrhage	Sudden onset of a severe constant headache that is often occipital or nuchal. “Sentinel hemorrhage” in 15–31% of cases. Vomiting often with onset of headache. *Cerebellar hemorrhage*: sudden onset of dizziness, vomiting, marked truncal ataxia and inability to walk.	Awake to lethargic. Markedly elevated BP. Contralateral hemiplegia, hemianesthesia, and aphasia or neglect (depending on the hemisphere involved). *Cerebellar hemorrhage*: gaze palsies and increasing stupor; patients may rapidly progress to coma.	Fingerstick glucose, noncontrast head CT, ECG. LP if CT negative. Laboratory tests may be indicated.
Testicular torsion	History of an athletic event, strenuous physical activity, or trauma just prior to the onset of scrotal pain. May awaken from sleep with unilateral testicular pain. Severe pain felt in lower abdominal quadrant, inguinal canal, or the testis.	Involved testis is aligned along a horizontal rather than a vertical axis. The axis of alignment can be determined only with the patient in an upright position. Diminished or absent cremasteric reflex on involved side.	If testicular torsion cannot be excluded by history and physical examination, emergency scrotal exploration is the definitive diagnostic procedure. In patients with indeterminate clinical presentations, color-flow duplex is test of choice. Doppler US and radionuclide scintigraphy may be used.

(*continued*)

Table 44.2 Differential diagnosis for vomiting (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Ureteral colic	Acute onset of flank pain radiating to groin. Nausea and vomiting are common. Patient usually writhing in pain. May have bloody urine.	Vital signs usually normal. CVAT with percussion; generally benign abdominal examination.	UA usually shows hematuria. Helical or spiral CT (CT urogram) performed without contrast has replaced IVP as the test of choice.
Vertigo	Sudden or gradual onset of sensation of room spinning (depending on etiology), associated with nausea and vomiting. May be related to head position, tinnitus, ear ringing, buzzing, or headache in peripheral vertigo. Other neurologic symptoms may be present in central vertigo.	Vertigo sensation may be reproduced depending on the patient's position. Nystagmus may be present with symptoms. For peripheral vertigo, other neurologic abnormalities should be absent.	Peripheral: Symptomatic therapy. Central: CT head (or MRI) and neurologic consultation warranted.
Volvulus (duodenal stenosis or atresia)	Bilious vomiting, with or without abdominal distention; streaks of blood in the stool. In older children, pain is usually constant, not colicky.	Newborns, may present with the sudden onset of an acute abdomen and shock. May be pale with grunting respirations. Approximately 33% of infants appear jaundiced.	AAS, CBC, chemistry panel and fluid rehydration.

AAS: acute abdominal series; ABG: arterial blood gas; AMI: acute myocardial infarction; BP: blood pressure; CBC: complete blood count; CHF: congestive heart failure; CK-MB: creatine kinase–MB fraction; CRP: C-reactive protein; CT: computed tomography; CVAT: costovertebral angle tenderness; CXR: chest X-ray; DKA: diabetic ketoacidosis; ECG: electrocardiogram; GC: gonorrhea; GI: gastrointestinal; HJR: hepatojugular reflux; IV: intravenous; IVP: intravenous pyelogram; JVD: jugular venous distention; LFTs: liver function tests; LP: lumbar puncture; LUQ: left upper quadrant; LV: left ventricular; MR: mitral regurgitation; MRI: magnetic resonance imaging; NSAID: nonsteroidal antiinflammatory drug; RLQ: right lower quadrant; RUQ: right upper quadrant; UA: urinalysis; US: ultrasound; VBG: venous blood gas; VSD: ventricular septal defect; WBC: white blood cell.

Diagnostic testing

The history and physical examination should determine which laboratory studies are necessary to investigate the etiology of vomiting.

Laboratory studies

Bedside glucose

A bedside glucose should be performed on patients who look ill and have been vomiting.

Complete blood count

A complete blood count (CBC) is not usually helpful unless blood loss is suspected. An elevated hemoglobin may suggest dehydration although this finding is neither sensitive nor specific.

Chemistry panel

In an otherwise healthy young adult with mild, limited vomiting, a chemistry panel adds little to the diagnostic approach. Patients with clinical evidence of dehydration or severe, protracted vomiting should undergo electrolyte testing. In patients with underlying disease, especially diabetes, or tenuous health status with comorbidities, more comprehensive laboratory testing guided by history and suspected diagnosis is important.

Liver function tests and lipase

Liver function tests (LFTs) and lipase should be considered in patients with epigastric pain, right upper quadrant pain, liver failure, or jaundice.

Troponin or cardiac markers

In the setting of vomiting in a patient with cardiac risk factors (including age), cardiac markers such as troponin are important to consider. Vomiting may be due to acute coronary syndrome, which may be silent. Furthermore, the stress of vomiting may result in physiologic stress on the heart and ischemia or infarction.

Urinalysis

A urinalysis should also be ordered to evaluate urine concentration and identify urinary tract infection (UTI). The presence of ketones is common with a prolonged starvation state but also raises concern for diabetic ketoacidosis or hyperemesis gravidarum. Hematuria may be associated with a ureteral calculus.

Pregnancy test

A urine or serum pregnancy test should be ordered on all females of childbearing age with vomiting.

Other tests

If the patient has liver disease and hematemesis, a prothrombin time/international normalized ratio (PT/INR) and type and crossmatch should be ordered. Thyroid function testing may identify suspected thyrotoxicosis. Specific serum drug levels (e.g., acetaminophen, salicylates, digoxin, theophylline) may identify medication-related toxicity which may be unintentional (e.g., elderly patient) or intentional (e.g., ingestion).

Electrocardiogram

All patients with vomiting of suspected cardiac origin should have an ECG. Older patients, especially those who appear ill or are tachycardic, should also have an ECG.

Radiologic studies

Plain films

A kidney–ureter–bladder (KUB) X-ray series is not indicated in the evaluation of vomiting. An acute abdominal series (AAS) may be indicated for patients with previous surgeries, a hernia on physical examination, suspected ingestion of radiopaque drugs, tympany, or high suspicion for bowel obstruction. An upright chest X-ray (CXR) may be indicated if the "vomiting" is actually a gag reflex evoked by coughing, due to pneumonia, or to look for signs of esophageal rupture (e.g., pleural effusion).

Computed tomography

Computed tomography (CT) of the brain should be performed on any patient with suspected increased ICP, intracranial bleeding, head trauma, or intracranial pathology (brain tumor or cysts). An abdominal CT should be considered for patients over the age of 55 years with an unclear vomiting etiology, or those with persistent abdominal pain, history of abdominal trauma, or suspected appendicitis or mass not obvious on physical examination.

Ultrasonography

If one suspects biliary pathology, a right upper quadrant (RUQ) ultrasound should be performed. An abdominal ultrasound is the initial diagnostic imaging study for children with suspected pyloric stenosis or intussusception. A pelvic ultrasound should be performed on female patients in whom there is suspicion for ovarian torsion, tubo-ovarian abscess, or an undocumented pregnancy. A testicular ultrasound should be obtained whenever testicular torsion or mass is suspected.

Nasogastric tube

If the patient is vomiting blood, a nasogastric (NG) tube should be placed to determine the quantity and intensity of bleeding.

General treatment principles

A majority of patients will not have the specific etiology of vomiting identified during their ED visit. These patients still require and deserve symptomatic therapy.

Volume repletion

Regardless of the etiology of vomiting, the ABCs (airway, breathing, circulation) must be addressed first. If the patient is in circulatory collapse, venous access with two large-bore peripheral IVs must be established and a fluid bolus of at least 20 mL/kg normal saline administered.

For a stable patient with vomiting, the administration of fluids can be accomplished in several ways. Oral rehydration may be attempted, especially in children. Depending on the underlying etiology of the vomiting, IV rehydration may be more effective and efficient. An IV also allows parenteral administration of antiemetic medication.

Antiemetics

Most vomiting patients are treated symptomatically by rehydration and relief of nausea and vomiting. However, this may not be possible if the underlying cause is metabolic or neurologic; these cases require treatment of the underlying disease to treat the vomiting.

There are several types of medications (antiemetic agents) used to alleviate vomiting (Table 44.3). Ondansetron is a first-line agent for treatment of nausea and vomiting because of its safety, efficacy and relative lack of sedation. Unlike other antiemetics, ondansetron is safe for use in children over 1 month of age. It is commonly administered as an oral dissolving tablet (ODT) with good success. Prochlorperazine or promethazine are commonly used in adults to treat vomiting of unknown etiology.

The primary side effect of all antiemetics, except the 5-HT antagonists, is sedation. Therefore, the patient must have a ride home and be cautioned not to drive for the following day or when taking the medication (if discharged with a prescription). Possible side effects of the phenothiazine antiemetics and metoclopramide are akathisia and dystonia. Akathisia is a condition of restlessness that includes nervousness, anxiety, and a feeling that one's skin is "crawling." This can be treated with diphenhydramine or a benzodiazepine. A dystonic reaction can be far more serious, and may include rhythmic contractions of the neck and back as well as repetitive protrusion of the tongue. A dystonic reaction requires treatment with diphenhydramine or benztropine for 48 hours, with possible hospital admission if severe. No further doses of antiemetics that cause dystonia should be given.

Special patients

Pediatric

In children, vomiting can represent a benign, self-limited illness or a severe underlying illness. In newborns and infants, consider sepsis, inborn errors of metabolism and obstructive lesions of the GI tract. Projectile vomiting is concerning for pyloric stenosis; bilious vomiting is an ominous finding suggesting volvulus or intussusception. Causes of

Table 44.3 Medications for vomiting

Agent	Dose	Comments
Dopamine antagonists		
Prochlorperazine	Adult: 2.5–10 mg IV Q 3–4 hrs; 5–10 mg IM Q 3–4 hrs; 5–10 mg PO TID-QID; 25 mg PR; max 40 mg/24 hr Pediatric (≥2 yr): 0.4 mg/kg/24 hr PO/PR TID-QID; 0.1–0.15 mg/kg/dose IM TID-QID; max 40 mg/24 hr	More common side effects include drowsiness, extrapyramidal reactions such as akathisia and parkinsonism, and anticholinergic effects such as blurred vision, dry mouth, and constipation. Akathisia that can develop any time over 48 hrs post administration; decreasing the infusion rate reduces the incidence of akathisia without affecting symptom relief.
Promethazine	Adult: 12.5–25 mg IV/IM/PO/PR Q 4–6 hrs; max 100 mg/24 hr Pediatric (≥2 yr): 0.25–1 mg/kg/dose IV/IM/PO/PR Q 4–6 hrs; max 25 mg/dose	**Avoid** use in children <2 yr because of risk for fatal respiratory depression. More sedating than comparative agents. Potential for phlebitis and severe tissue injury (necrosis, gangrene) following IV administration; if IV administration is necessary, dilute with NS and administer over 10–15 min.
5-HT$_3$ antagonists		
Ondansetron	Adult: 4–8 mg IV single dose Pediatric: up to 40 kg: 0.1 mg/kg; >40 kg: 4 mg IV single dose	Considered the first-line antiemetic agent. Most common side effects are headache, constipation, and asthenia; not associated with akathisia or sedation.
Prokinetic agents		
Metoclopramide	Adult: 10 mg IM/IV, may repeat Q 4–6 hrs Pediatric: 1–2 mg/kg/dose IV/IM/PO Q 2–6 hrs	Most common side effects are restlessness, drowsiness and fatigue. May cause extrapyramidal symptoms, most commonly acute dystonic reactions. Akathisia that can develop any time over 48 hrs post administration; decreasing the infusion rate reduces the incidence of akathisia without affecting symptom relief.

IV: intravenous; IM: intramuscular; PO: per os; PR: per rectum; Q: every; QID: four times a day; TID: three times a day.

vomiting in children include infectious (acute gastroenteritis, otitis media, pneumonia), metabolic (diabetic ketoacidosis), mechanical (obstruction) and neurogenic (increased ICP). The most common cause of vomiting in an older child or adolescent is infectious gastroenteritis. Evaluation of a child's hydration status is particularly important. Vital signs may be normal despite significant dehydration; dry mucous membranes, decreased urine output and mental status changes are better indicators of dehydration. Mild to moderate dehydration due to vomiting of benign etiology may be treated with a weight-appropriate dose of ondansetron ODT, followed by attempt at oral rehydration 15–30 minutes after administration. In many EDs, this may be initiated in the triage area. Moderate to severe dehydration should be treated with an initial IV normal saline bolus of 20 mL/kg. If vomiting has been protracted, a bedside glucose should also be obtained as part of the initial evaluation. This is especially important in children, who have lower glycogen stores compared with adults. Vomiting in children may also be a presenting symptom of diabetes or diabetic ketoacidosis. Further evaluation and treatment should be directed at determining whether a serious underlying cause of vomiting is present.

Elderly

The general evaluation and treatment principles for vomiting also apply to the geriatric population. The response of geriatric patients to dehydration may be blunted by their chronic illnesses or medications; vital signs may not demonstrate hypotension or tachycardia despite severe dehydration. In the elderly, a serious cause of vomiting is found more frequently than in younger adult populations. Etiologies such as cardiac disease, intracranial or intracerebral processes, metabolic conditions, renal or liver dysfunction, and postoperative bowel obstruction due to adhesions should be considered. Therefore, the diagnostic work-up (laboratory and imaging) is typically more extensive, as the cause of vomiting in this population is rarely benign. Attention to the ABCs remains essential. Aggressive rehydration may be complicated by underlying illness and cardiac disease in this population.

Pregnancy

Pregnant patients commonly present to the ED with significant nausea and vomiting (often referred to as hyperemesis gravidarum or "morning sickness"). These patients should be aggressively hydrated, as volume depletion raises their risk of miscarriage. Antiemetics are commonly prescribed for pregnant women who need relief from vomiting. Selection of a specific agent should be based on the patient's history, and both the safety profile and pregnancy classification of the drug. Despite the classification (FDA risk category C) of many of these agents, physicians commonly use them in the treatment of pregnant patients. Close follow-up with the patient's obstetrician is essential.

Palliative care

Intractable vomiting may be seen in patients who are at the end of life. Rapid symptom control in these patients is extremely important in order to provide comfort. Atypical agents helpful in treating vomiting, such as steroids, may be necessary in addition to standard antiemetic agents.

Disposition

Admission

All patients with life-threatening causes of vomiting, serious illness related to vomiting, an unclear diagnosis and poor response to fluids and antiemetic therapy, or vomiting refractory to therapy should be admitted to the hospital. The elderly and patients who may have difficulty with timely follow-up (e.g., indigent, alcohol/drug abuse, lack of transportation) may also require admission. Many patients who require gentle or prolonged rehydration may require observation in a clinical decision unit.

Consultation

Consultation with a specialist will depend on the underlying etiology of the vomiting.

Discharge

In order to assure continued hydration, the patient's ability to keep down a small amount of fluid (water) without vomiting should be assessed prior to discharge. This is known as a "PO challenge." If the patient receives adequate IV hydration in the ED, an oral fluid challenge is not essential before final disposition. Occasionally, patients without serious causes of vomiting, who have received IV fluids and feel better, may go home despite being unable to tolerate PO fluids. Most patients with simple vomiting due to a benign etiology and without signs of significant dehydration or metabolic derangement may be discharged home if they tolerate liquids in the ED and follow-up in 24–48 hours can be assured.

Pearls, pitfalls and myths

- Patients may confuse coughing or spitting up phlegm with true vomiting. Patients often confuse hematemesis (vomiting blood) with hemoptysis (coughing blood).
- The history and physical examination usually help determine the cause of the vomiting.
- Although the etiology of vomiting is not always identified in the ED, therapy should not be withheld.
- Regardless of the etiology, evaluation of patients who are vomiting should focus on the ABCs.
- Resuscitation of the markedly dehydrated individual, regardless of the etiology of vomiting, needs to be addressed in an urgent fashion.
- Treatment of life-threatening etiologies must often be initiated before a firm diagnosis of vomiting has been established.

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45 Weakness

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Scope of the problem

Emergency physicians are likely to encounter at least one patient with the chief complaint of weakness daily. In contrast to other presentations, the true meaning of the patient's complaint may be difficult to ascertain. It is important to identify what symptoms the patient and/or family members are trying to convey.

On one hand, the patient may complain of a global lack of energy, extreme fatigue, lightheadedness, or simply feeling "ill." In this regard, the differential diagnosis extends from benign etiologies (mild dehydration, viral syndrome, hypothyroidism, mild depression) to life-threatening emergencies requiring immediate intervention (acute myocardial infarction [AMI], sepsis, pericardial tamponade). Other clues may help establish the correct diagnosis, but the isolated complaint of "weakness" may be associated with any of these disease entities or a myriad of others.

On the other hand, the patient may present complaining of a specific distribution of weakness associated with true impairment of motor function. These patients with motor weakness may present in a number of ways. They may have hemiparesis (weakness of one side of the body without complete paralysis) or hemiplegia (complete paralysis of one side of the body), indicating ongoing ischemic stroke or transient ischemic attack (TIA). Patients may also present with a pattern of symmetric ascending paresis or paralysis (e.g., Guillain–Barré syndrome [GBS]), focal peripheral motor weakness (e.g., carpal tunnel syndrome), symmetric proximal muscle weakness (e.g., polymyositis), weakness in a bulbar muscle distribution causing diplopia or ptosis (e.g., myasthenia gravis [MG], botulism), or a combination of the above findings (e.g., multiple sclerosis [MS]).

This chapter focuses primarily on the "weak" patient presenting with true objective motor function impairment. Acute ischemic stroke (AIS) and TIA are discussed in detail, as these disease processes are the most important causes of motor weakness that emergency physicians encounter, other than weakness affecting the airway or respiratory musculature.

Acute stroke accounts for approximately 1 million hospitalizations per year in the United States, and is the third leading cause of death and the number one cause of adult disability. Emergency physicians must also be prepared to identify and treat other etiologies of motor weakness.

Anatomic essentials

Accurate diagnosis and effective treatment of a patient with motor weakness hinge on an emergency physician's ability to identify the underlying lesion(s) responsible for the patient's disability. First, emergency physicians must determine whether the patient's weakness arises from the central nervous system (CNS) or peripheral nervous system (PNS), or the muscles themselves.

Central nervous system

CNS disturbances result in a constellation of signs and symptoms due to the specific underlying lesion(s). Most often, acute weakness is caused by an abrupt cervicocerebral vascular occlusion which deprives a given dependent region of brain parenchyma its supply of oxygenated blood. When examining cerebral blood flow, the vascular distributions may be divided into the anterior circulation (carotid artery distribution) and the posterior circulation (vertebrobasilar distribution).

In the anterior circulation, the carotid terminus divides into the anterior cerebral artery (ACA) and the middle cerebral artery (MCA). ACA occlusions cause sudden onset of contralateral upper and lower extremity weakness and numbness, with the lower extremity being affected greater than the upper extremity. There may also be associated gait apraxia (clumsiness), incontinence and slowed mentation. MCA occlusions result in the acute onset of contralateral upper and lower extremity weakness and numbness, with the upper extremity being affected greater than the lower extremity. A contralateral facial droop is usually present, with hemiparesis of the extremities. Contralateral homonymous hemianopsia (visual field disturbance) is often present, and conjugate gaze may be affected with the eyes pointing towards the side of the lesion.

Depending on which cerebral hemisphere is affected, further deficits also occur. If the dominant hemisphere (usually the left brain) is deprived of blood flow, aphasia is commonly present. The aphasia may be *expressive* (the patient knows what he or she wants to say but cannot get the words out), *receptive* (the patient cannot understand what is being spoken or written), or *global* (both expressive and receptive aphasia present). If the non-dominant hemisphere is affected (usually the right brain), hemineglect (the patient unconsciously ignores the affected side of the body) may be present (Table 45.1).

Vertebrobasilar arterial occlusions result in a constellation of symptoms that may include ipsilateral cerebellar dysfunction (severe vertigo, nausea and vomiting, tinnitus and deafness, ataxia and nystagmus), cranial nerve (CN) dysfunctions, and hemiparesis and/or hemisensory deficits. The variety of findings observed by the emergency physician depends on the specific arterial distribution affected (Table 45.1).

Some CNS disturbances are caused by inflammatory or demyelinating disorders. Demyelinating disorders

Table 45.1 Clinical stroke syndromes and site of arterial occlusion

Stroke syndrome	Arterial occlusion site	Clinical manifestations
Anterior cerebral artery occlusion	Anterior cerebral artery	Contralateral upper and lower extremity motor weakness and sensory loss, with lower extremity more affected than upper; apraxia; incontinence; slowed mentation
Central midbrain syndrome (Tegmental syndrome)	Paramedian branches of basilar artery	(a) Ipsilateral oculomotor nerve palsy (b) Hemichorea of contralateral limbs (c) Contralateral loss of cutaneous sensation and proprioception
Dorsal midbrain syndrome (Parinaud syndrome)	Usually caused by compression of extra-axial lesion (pinealoma)	Paralysis of upward gaze
Lateral inferior pontine syndrome	Anterior inferior cerebellar artery	(a) Ipsilateral limb ataxia (b) Ipsilateral loss of facial cutaneous sensation (c) Hiccup (d) Ipsilateral Horner's syndrome (e) Nausea/vomiting/nystagmus (f) Contralateral loss of pain and temperature sensation (g) Ipsilateral facial paralysis (h) Deafness and tinnitus (i) Ipsilateral gaze paralysis
Lateral medullary syndrome (Wallenberg syndrome)	Posterior inferior cerebellar artery (often lesion in vertebral artery)	(a) Ipsilateral limb ataxia (b) Ipsilateral loss of facial cutaneous sensation (c) Hiccup (d) Ipsilateral Horner's syndrome (e) Nausea/vomiting/nystagmus (f) Contralateral loss of pain and temperature sensation (g) Dysphagia (h) Hoarseness with ipsilateral vocal cord paralysis (i) Loss of ipsilateral pharyngeal reflex
Lateral mid-pontine syndrome	Short circumferential artery	(a) Ipsilateral limb ataxia (b) Ipsilateral loss of facial cutaneous sensation (c) Hiccup (d) Ipsilateral Horner's syndrome (e) Nausea/vomiting/nystagmus (f) Contralateral loss of pain and temperature sensation (g) *Trigeminal nerve impairment*: Chewing difficulty (bilateral lesions) or ipsilateral jaw deviation with mouth opened (unilateral lesions)
Lateral superior pontine syndrome	Superior cerebellar artery	(a) Ipsilateral limb ataxia (b) Ipsilateral loss of facial cutaneous sensation (c) Hiccup (d) Ipsilateral Horner's syndrome (e) Nausea/vomiting/nystagmus (f) Contralateral loss of pain and temperature sensation (g) Absence of specific cranial nerve signs
Locked-in syndrome	Basilar artery occlusion causing bilateral ventral pontine lesions	Complete quadriplegia, inability to speak, and loss of all facial movements despite normal level of consciousness; patients may communicate with eye or eyelid movements
Medial inferior pontine syndrome	Paramedian branches of basilar artery	(a) Contralateral hemiparesis (b) Contralateral loss of proprioception and vibratory sensation (c) Ipsilateral limb ataxia (d) Ipsilateral gaze paralysis (e) Ipsilateral lateral rectus paralysis (f) Gaze-evoked nystagmus
Medial medullary syndrome	Paramedian branches of basilar artery	(a) Contralateral hemiparesis (b) Contralateral loss of proprioception and vibratory sensation (c) Ipsilateral limb ataxia (d) Ipsilateral tongue weakness
Medial superior pontine syndrome	Paramedian branches of basilar artery	(a) Contralateral hemiparesis (b) Contralateral loss of proprioception and vibratory sensation (c) Ipsilateral limb ataxia (d) Internuclear ophthalmoplegia (e) Palatal myoclonus

(*continued*)

Table 45.1 Clinical stroke syndromes and site of arterial occlusion (*cont.*)

Stroke syndrome	Arterial occlusion site	Clinical manifestations
Middle cerebral artery occlusion, dominant hemisphere	Middle cerebral artery (usually left)	Contralateral upper and lower extremity motor weakness and sensory loss, with upper extremity more affected than lower; contralateral facial droop; homonymous hemianopsia; gaze deviation to side of lesion; language disturbances (expressive, receptive, and/or global aphasia)
Middle cerebral artery occlusion, non-dominant hemisphere	Middle cerebral artery (usually right)	Contralateral upper and lower extremity motor weakness and sensory loss, with upper extremity more affected than lower; contralateral facial droop; homonymous hemianopsia; gaze deviation to side of lesion; hemineglect
Ventral midbrain syndrome (Weber syndrome)	Paramedian branches of basilar artery	(a) Contralateral hemiparesis (b) Contralateral supranuclear facial paresis (c) Ipsilateral oculomotor nerve palsy

may cause a confusing variety of signs and symptoms, such as the combination of unilateral visual disturbance and variable motor weakness seen in patients with MS.

Peripheral nervous system

PNS dysfunction may cause motor weakness as a result of pathophysiologic processes involving the neuromuscular junction or the peripheral nerves themselves. Disruption of neuromuscular junction causes motor weakness by inhibiting normal motor end plate stimulation to facilitate muscle contraction. This may be the result of a toxin-mediated process (e.g., botulism) or abnormal antibodies attacking the motor end plate (e.g., MG). Dysfunction of the neuromuscular junction also occurs iatrogenically with drugs that act at the neuromuscular junction (e.g., succinylcholine, vecuronium). Peripheral nerve malfunction that impairs motor strength may result from direct damage to peripheral nerves (e.g., GBS), toxins (e.g., tick paralysis, arsenic poisoning), compressive neuropathies (e.g., carpal tunnel syndrome), or peripheral vascular occlusions.

Primary muscle dysfunction

Primary muscle dysfunction resulting in motor weakness may be caused by an inflammatory myopathy (e.g., polymyositis, dermatomyositis) or abnormalities in ion channels found in skeletal muscles (e.g., hypokalemic periodic paralysis).

Red flags

Emergency clinicians must be adept at recognizing "red flags" (warning signs and symptoms) from the history and physical examination that raise concern for life-threatening or dangerous diagnoses (Table 45.2).

History

Obtaining an accurate and complete history can be challenging in the patient presenting with acute weakness. Many patients have difficulty pinpointing the exact timing of their weakness. In addition, some stroke and TIA patients suffer from aphasia and are unable to relay their history. With non-dominant hemispheric strokes and TIAs, the patient may be completely unaware that a large neurologic deficit exists. In many cases, family members, emergency medical service (EMS) personnel and bystanders are critical resources for obtaining a thorough history.

What is the distribution of motor weakness?

The distribution of motor weakness corresponds to the underlying anatomic lesion(s). Hemiparesis is indicative of stroke, TIA, or possible mimics. Isolated extremity weakness is likely the result of a compressive radicular or peripheral neuropathy, or peripheral vascular occlusion. When bilateral weakness is encountered, further historical points must be explored. GBS is associated with a symmetric ascending paralysis initially involving the lower extremities, then progressing in a cephalad direction. Bilateral motor weakness encompassing both cranial and peripheral nerve distributions is likely the result of inflammatory (e.g., MS), toxic/metabolic (e.g., botulism), or autoimmune processes (e.g., MG). When bilateral weakness is associated with a discrete sensory level (below which the patient has loss of sensation) and/or bladder dysfunction, a lesion in the spinal cord is suspected. Bilateral weakness that affects the proximal musculature to a greater degree than distal motor strength suggests a myopathy (e.g., polymyositis, dermatomyositis). These patients commonly have difficulty climbing stairs or rising from a chair, as well as problems with personal grooming.

Was the onset of weakness sudden or gradual?

The sudden onset of motor weakness implies a vaso-occlusive etiology. Ischemic strokes and TIAs are the result of thrombotic or embolic occlusion of a cervicocerebral artery, and in general occur with sudden onset. The symptom onset may be difficult for patients to identify, as they may be unaware of their symptoms. In addition, the patient may have been asleep when the stroke or TIA began, making the determination of onset impossible. The sudden onset of extremity weakness caused by the abrupt occlusion of a major artery supplying that extremity will

Table 45.2 Weakness red flags

History	Concerning diagnosis
Transient neurologic deficit	TIA with potential for CVA
Sudden onset of severe headache	Intracerebral/subarachnoid hemorrhage
Unilateral neck pain (may be severe, ripping or tearing)	Acute arterial dissection (anterior neck pain → carotid artery dissection; posterior neck pain → vertebral artery dissection)
Neck pain with stiffness	CNS infection, subarachnoid hemorrhage
Ascending paralysis	Guillain–Barré syndrome
Descending paralysis	Miller-Fischer variant of Guillain–Barré syndrome, botulism
Fever	CNS infection, abscess, endocarditis with septic emboli
Diplopia	Posterior circulation ischemia, myasthenia gravis, botulism, neoplasm
Saddle anesthesia, bowel/bladder dysfunction	Cauda equina syndrome
Monocular visual complaints	Multiple sclerosis (optic neuritis), amaurosis fugax
Nausea and vomiting	Increased intracranial pressure, posterior circulation ischemia
Chest pain	AMI, thoracic aortic dissection
Muscular pain	Myositis, rhabdomyolysis
Coordination problems, including gait disturbance	Cerebellar lesions, posterior circulation ischemia
Ingestion of improperly canned foods, infant honey ingestion	Botulism
IV drug abuse	Wound botulism, endocarditis with septic emboli
Severe back pain	Spinal epidural abscess, aortic dissection, herniated nucleus pulposis, transverse myelitis
Subacute/chronic abdominal pain	Heavy metal toxicity
Tick envenomation, camping, hiking	Tick paralysis
Episodic neurologic disturbances with periods of recovery/recurrence, often in a peculiar or nonspecific pattern	Multiple sclerosis
Fatigability of symptoms, worsened with heat	Myasthenia gravis
Vertigo	Posterior circulation ischemia
Examination finding	**Concerning diagnosis**
Oculomotor dysfunction	CVA, myasthenia gravis, botulism, mass lesion, Wernicke-Korsakoff syndrome
Focal unilateral neurologic deficit	Any cause of focal deficits, especially acute CVA
Nuchal rigidity	CNS infections, subarachnoid hemorrhage
Ataxia	Cerebellar disease
Heart murmur, fever	Endocarditis with septic emboli
Bilateral motor/sensory deficits	Transverse myelitis, spinal cord lesion/ischemia
Mixed upper/motor neuron findings	Amyotrophic lateral sclerosis
Areflexia, lower extremity weakness	Guillain–Barré syndrome
Proximal muscle weakness	Polymyositis, myasthenia gravis (with repeated activities)

AMI: acute myocardial infarction; CNS: central nervous system; CVA: cerebrovascular accident; TIA: transient ischemic attack.

likely be accompanied by paresthesias, pain, pallor and pulselessness. Patients presenting with the gradual onset of progressive motor weakness probably suffer from a non-vascular pathophysiologic process. Subacute motor weakness is more likely associated with inflammatory disorders of the nervous system (e.g., MS, transverse myelitis) or musculoskeletal system (e.g., polymyositis, dermatomyositis), compression neuropathies (e.g., carpal tunnel syndrome), autoimmune disorders (e.g., MG, GBS), or toxic/metabolic processes (e.g., botulism, hypokalemic periodic paralysis).

Were there any significant events surrounding the onset of weakness?

Many stroke mimics are associated with easily identifiable conditions that accompany the onset of weakness. Seizures preceding the onset of weakness may imply postictal (Todd's) paralysis. Ongoing migraine headache in a young female associated with motor weakness might indicate a complicated migraine. Severe sudden headache with motor weakness should alarm the clinician to a possible subarachnoid hemorrhage. Unilateral motor weakness in the setting of trauma could indicate a subdural or epidural hematoma or unusual cord injury (e.g., Brown-Séquard syndrome), whereas bilateral motor weakness accompanied by a sensory level deficit and/or priapism likely indicates a complete cord injury. Central cord syndrome, caused by a cervical cord injury, manifests as upper extremity weakness greater than lower extremity weakness, motor findings greater than sensory findings, and distal strength more affected than proximal strength. Severe migratory chest or neck pain accompanying motor weakness should alert the physician to the possibility of arterial dissection syndromes.

Is there a temporal pattern to the weakness?

Patients who complain of weakness worsening with repetitive motions may be exhibiting symptoms of neuromuscular junction pathology (e.g., MG). These patients may report difficulty with blinking, chewing, typing, or other motor tasks requiring frequent repeated movements. Increased clinical suspicion for dyskalemic periodic paralysis occurs when the patient complains of sudden episodic and spontaneously resolving motor weakness.

When did the weakness begin?

Emergent stroke therapy is extremely time-dependent. The option of intravenous (IV) thrombolytic therapy for AIS is available only within a strict short time window of 3 hours from symptom onset, although current research suggests that this window can be expanded to 4.5 hours. It is therefore critical that the emergency physician establishes the exact time of symptom onset from the patient or witnesses. This includes the last time the patient was seen "normal." Therefore, a patient waking up with new symptoms consistent with a stroke has "time zero" set as the time he went to sleep, not the time his symptoms were first noticed in the morning.

Associated symptoms

Headache

Ischemic stroke and TIA are not usually associated with severe headache initially. Acute motor weakness accompanied by a significant headache is worrisome for subarachnoid hemorrhage, arterial vascular malformation, or epidural/subdural hematoma. When headache occurs with ischemic stroke, the evaluating physician must consider the possibility of increased intracranial pressure. Stroke symptoms accompanying a migraine-type headache may indicate complicated migraine, classically a disease of young adult females.

Visual changes

Double vision (diplopia) of acute onset may be associated with posterior circulation stroke. In isolation, this complaint usually implies a process affecting the neuromuscular junction. Monocular visual complaints may occur with optic neuritis of new-onset MS.

Nausea and vomiting

The presence of nausea and vomiting may be a warning sign of increased intracranial pressure or a posterior circulation ischemic event. Careful evaluation for intracranial lesions and cerebral edema is warranted. Severe vomiting and/or diarrhea may predispose the patient to electrolyte imbalances leading to motor weakness.

Chest or neck pain

The presence of ongoing migratory chest or neck pain may indicate the presence of an acute arterial dissection (e.g, thoracic aorta or carotid/vertebral artery dissection). Appropriate radiologic studies should be obtained emergently when arterial dissection is suspected. Also, AMI may also be associated with AIS.

Abdominal and/or back pain

Abdominal and/or back pain accompanied by lower extremity weakness could signify expansion or thrombosis of an abdominal aortic aneurysm (AAA) with concomitant spinal cord infarction. Back pain with unilateral lower extremity weakness may indicate herniated lumbar disk with nerve root impingement. Back pain with bilateral lower extremity weakness, sensory level and priapism in the setting of significant trauma is worrisome for complete spinal cord injury. Similar symptoms without a history of trauma should alert the physician to the possibility of acute aortic dissection with cord ischemia, cauda equina syndrome, primary spinal cord lesions, or compressive spinal cord lesions, such as epidural hematoma or abscess.

Musculoskeletal pain and tenderness

Musculoskeletal pain and diffuse muscular tenderness associated with motor weakness (especially proximal weakness) is suggestive of myopathy (e.g., polymyositis, dermatomyositis). Severe muscular pain, dark urine or oliguria, and motor weakness may indicate acute rhabdomyolysis in the appropriate setting.

Rash

The complaint of rash, particularly in the periorbital region, is associated with dermatomyositis.

Past medical

When eliciting a patient's history in the setting of acute motor weakness, the emergency physician must focus on identifying risk factors for the suspected etiology of weakness. When AIS or TIA is suspected, the physician should inquire about risk factors (Table 45.3). A recent history of viral illness accompanied by the acute onset of ascending bilateral motor weakness is classic for GBS. Occupational exposures to heavy metal toxins (e.g., arsenic poisoning) or to repetitive hand motions, such as hammering or typing (e.g, carpal tunnel syndrome), may provide clues to the diagnosis. Social history should include questions about possible cocaine use in the setting of stroke, suspected subarachnoid hemorrhage, or TIA. Heavy alcohol use accompanies alcohol-induced myopathies. Family history may be positive for familial causes of weakness (e.g., hypokalemic periodic paralysis).

A complete list of the patient's current medications should be carefully reviewed. The use of corticosteroids or certain lipid-lowering agents may induce drug-related myopathy. Although not usually a cause of true motor weakness, medication use in the elderly is commonly attributed to subjective complaints of weakness and dizziness.

Table 45.3 Risk factors for acute ischemic stroke or transient ischemic attack

History of TIA	Older age
History of stroke	Hypertension
Cigarette smoking	Diabetes mellitus
Atrial fibrillation	Coronary artery disease
Hyperlipidemia	Male gender
Carotid stenosis	Cocaine or amphetamine use

Physical examination

The primary aim of the physical examination is to both localize and quantify the extent of neurologic deficit(s) present. The distribution and extent of weakness often lend important clues to the underlying lesion(s), and assist with the diagnosis and management of the patient.

General appearance

As with all patients, global assessment of the ABCs (airway, breathing, circulation) takes first priority. Fortunately, most patients presenting with motor weakness do not have major issues in these areas. Exceptions are patients suffering from large intracranial hemorrhage who may need immediate airway management. Patients with advanced GBS or MG may have extremely poor respiratory effort secondary to weakness of their breathing musculature, and may need emergent mechanical ventilation. Trauma patients with spinal cord injuries may suffer neurogenic shock and require interventions to correct circulatory collapse.

Vital signs

As a general rule, most patients presenting with acute motor weakness will not exhibit major vital sign abnormalities. Patients with AIS often present with elevated blood pressure (BP), as underlying uncontrolled hypertension is very common among stroke victims. Wide fluctuations in BP and heart rate may reflect autonomic instability associated with a large intracerebral process or GBS. The presence of fever may suggest an infectious etiology associated with acute motor weakness (e.g., epidural abscess, endocarditis with septic emboli), or may contribute to the precipitation of motor weakness with underlying disease (e.g, MG).

Head, eyes, ears, nose and throat

The head and neck are inspected for signs of trauma associated with epidural or subdural hematoma, or carotid dissection. Carotid auscultation is performed to identify the presence of carotid bruits, which may signify underlying carotid stenosis. Abnormalities of the thyroid gland prompt the examiner to search for thyroid dysfunction as a potential contributor to weakness.

Cardiopulmonary

The heart is auscultated to evaluate rhythm and to detect murmurs. An irregularly irregular rhythm present with atrial fibrillation is an important risk factor for embolic stroke. The presence of a murmur should alert the examiner to the possibility of valvular heart disease and the potential for emboli.

Extremities

Unequal pulses may be present in acute arterial dissection syndromes, which may cause acute motor weakness secondary to distal ischemia of a limb or the CNS.

Skin

The presence of a periorbital violaceous rash may be present with dermatomyositis. Careful examination for the presence of a tick should take place in at-risk patients with motor weakness.

Neurologic

A careful and thorough neurologic examination is critical to the evaluation of the patient with acute motor weakness. The essential neurologic examination consists of six major areas: mental status, CNs, motor function, sensory function, deep tendon reflexes (DTRs), and cerebellar function.

Mental status

An overall assessment of the patient's level of awareness includes assessing the level of consciousness and degree of orientation to person, place, time and situation. The emergency physician should identify any speech deficiencies. Dysarthria (slurred speech) or other profound language deficits, such as receptive aphasia (the patient cannot understand what is being said) or expressive aphasia (the patient cannot get his or her words out) should be noted.

Cranial nerves

A quick, systematic assessment of CN function follows the mental status examination. Tests for visual field deficits by confrontation (examiner faces the patient and slowly brings moving fingers in from the sides until the patient detects them), pupillary light reflexes, and extraocular movements (CNs II, III, IV, VI) are performed. The examiner has the patient smile to look for facial palsy (droop), and also tests facial sensation (CNs V, VII). Testing swallowing and symmetrical palate rise (CNs IX, X), shoulder shrugging (CN XI), and tongue deviation while the tongue protrudes from the mouth (CN XII) completes the CN examination.

Motor function

The motor examination assesses strength in all four extremities. Marked strength deficits may be detected on examination and are graded according to a 0–5 point scale (Table 45.4). Subtle strength deficits may be difficult to elicit. Evaluation for the presence of *pronator drift* may be helpful to detect very mild strength deficits of the proximal upper extremities. The examiner has the patient hold both arms out 90 degrees with palms up and eyes closed and watches for slight pronation of either forearm.

An overall assessment of function is also helpful to the examiner. Subtle deficiencies of motor strength may be identified with somewhat more difficult motor tasks, such as tiptoe or heel walking. Difficulty rising from a chair or stool, or with brushing hair may identify proximal muscle weakness associated with myopathies. Fatigability of a particular motor function (with initially normal strength) points to a disturbance at the neuromuscular junction (e.g., MG).

Table 45.4 Strength scale 0–5

Grade	Description
0	No discernible movement
1	Trace movement detected
2	Movement with gravity taken away
3	Movement against gravity
4	Movement against added resistance but less than normal strength
5	Normal strength

Sensory function

The sensory examination is the least important component of the emergency neurologic examination. A brief assessment of fine touch suffices in the emergency setting. If deficits are detected, more time may be taken to delineate exact distributions of sensory disturbances. If time allows, assessment of vibratory sensation, 2-point discrimination, and sharp/dull discrimination may be performed.

Reflexes

The emergency physician's assessment of DTRs should include reflex testing in both upper and lower extremities. Reflexes are graded on a 0–4 point scale (Table 45.5). Absence of lower extremity reflexes in the setting of acute bilateral lower extremity weakness is a hallmark of GBS. Peripheral reflexes of a particular myotome may be diminished or absent in a peripheral neuropathy or radiculopathy compression syndrome. Asymmetric hyperreflexia and/or clonus (extreme hyperreflexia with the presence of repetitive contraction of a muscle group) may be seen with CNS lesions. *Babinski's sign* (dorsiflexion of the great toe and fanning of the other toes when the plantar aspect of the foot is stroked) may be present in CNS lesions as well.

Table 45.5 Deep tendon reflex scale (0–4)

Grade	Description
0	Reflexes absent
1	Reflexes diminished but present
2	Normal reflexes
3	Reflexes increased
4	Clonus present

Cerebellar function

Finally, the emergency physician should examine the patient for cerebellar dysfunction. Coordination tests such as finger-to-nose and heel-to-shin coordination are performed. In the able patient, standard gait is assessed along with heel-to-toe walking. Difficulty in coordination may be quite apparent on examination of rapid alternating movements (dysdiadochokinesia).

The National Institutes of Health Stroke Scale

In the AIS patient, an important adjunct to the standard neurologic examination is the National Institutes of Health Stroke Scale (NIHSS). This 42-point scale focuses on and grades level of consciousness, visual function, motor function, sensation and neglect, language and cerebellar integrity (Table 45.6). It closely mirrors the standard neurologic examination. As can be seen from the scale, the higher the score, the worse the neurologic deficit associated with ongoing stroke. The NIHSS can provide insight into the location and severity of underlying ischemic stroke, and has been shown to be a strong initial predictor of overall clinical outcome. Perhaps most important, its use provides emergency physicians a powerful and accurate means of communication with the stroke team or neurology consultants.

Table 45.6 The National Institutes of Health Stroke Scale

	Category	Description	Score
1a	LOC	Alert	0
		Not alert, arousable	1
		Not alert, obtunded	2
		Coma	3
1b	LOC questions (month, age)	Answers both correctly	0
		Answers one correctly	1
		Incorrect on both	2
1c	LOC commands (open/close eyes, grip hand)	Performs both correctly	0
		Performs one correctly	1
		Incorrect on both	2
2	Best gaze (follow finger)	Normal	0
		Partial gaze palsy	1
		Forced deviation	2
3	Best visual (visual fields)	No visual loss	0
		Partial hemianopia	1
		Complete hemianopia	2
		Bilateral hemianopsia (blind)	3
4	Facial palsy (show teeth, raise eyebrows, squeeze eyes shut)	Normal	0
		Minor	1
		Partial	2
		Complete	3
5a	Motor arm left[a] (raise 90°, hold 10 seconds)	No drift	0
		Drift	1
		Some effort against gravity	2
		No effort against gravity	3
		No movement	4
5b	Motor arm right[a] (raise 90°, hold 10 seconds)	No drift	0
		Drift	1
		Some effort against gravity	2
		No effort against gravity	3
		No movement	4
6a	Motor leg left[a] (raise 30°, hold 5 seconds)	No drift	0
		Drift	1
		Some effort against gravity	2
		No effort against gravity	3
		No movement	4
6b	Motor leg right[a] (raise 30°, hold 5 seconds)	No drift	0
		Drift	1
		Some effort against gravity	2
		No effort against gravity	3
		No movement	4
7	Limb ataxia	Absent	0
		Present in one limb	1
		Present in two limbs	2
8	Sensory (fine touch to face, arm, leg)	Normal	0
		Mild-to-moderate loss	1
		Severe loss	2
9	Best language[b] (name items, describe pictures)	No aphasia	0
		Mild-to-moderate aphasia	1
		Severe aphasia	2
		Mute, global aphasia	3
10	Dysarthria (speech clarity to "mama, baseball, huckleberry, tip-top, fifty-fifty")	Normal articulation	0
		Mild-to-moderate dysarthria	1
		Unintelligible or worse	2
11	Extinction and inattention (formerly Neglect) (double simultaneous testing)	No abnormality	0
		Sensory or personal inattention/extinction	1
		Profound or complete hemi-inattention	2
	Total (0–42)		

LOC: level of consciousness.

[a] For limbs with amputation, joint fusion, etc., score a "9" and explain.

[b] For intubation or other physical barrier to speech, score a "9" and explain.

Differential diagnosis

Tables 45.7 and 45.8 provide diagnostic possibilities and suggested evaluations for patients with weakness.

Table 45.7 Differential diagnosis of acute ischemic stroke or transient ischemic attack

Diagnosis	Symptoms	Signs	Work-up
Arterial dissection syndromes	Patients usually present with focal motor weakness accompanied by severe tearing pain in the anterior neck and jaw (carotid dissection), posterior neck and back of head (vertebral dissection), chest and/or back (thoracic aortic dissection), or abdomen and/or back (abdominal aortic dissection).	Hypertension is frequently present. Focal motor weakness follows distribution of artery (arteries) affected and may involve an entire cerebrovascular distribution. Asymmetric BP and/or unequal pulses in extremities may be present with aortic dissection.	BP measurement in all four extremities for aortic dissection. CT of chest and/or abdomen with IV contrast (vascular surgeon should be emergently consulted prior to imaging if clinical suspicion is high). Angiography preferred for suspected carotid or vertebral dissection, as MRA may miss some dissections.
Bell's palsy	Patients complain of unilateral facial droop, eye pain (from dry eyes), hyperacusis (extreme sensitivity to noise), taste abnormalities, and sometimes retroauricular pain. History of recent URI may be obtained.	Peripheral 7th (facial) nerve palsy is found on examination (ipsilateral upper and lower facial motor weakness present). Conjunctivitis/keratitis may be present on affected side.	Diagnosis of Bell's palsy is clinical. Appropriate treatment and urgent referral are important to initiate from the ED.
Complicated migraine	Patients are often young peripartum females with history of migraine headaches. Patients present with symptoms of migraine headache (with or without pain, photophobia, phonophobia, nausea or vomiting) plus neurologic deficit.	Neurologic deficit may consist of focal motor weakness, sensory abnormality, aphasia, ataxia, or all of the above and may perfectly mimic a hemispheric stroke.	Diagnosis of exclusion. Demographic information provides clue. Subarachnoid hemorrhage, stroke, or space-occupying lesion must be ruled out in patients without history of complicated migraines.
Epidural/subdural hematoma	Classic history for epidural hematoma is head trauma with loss of consciousness followed by lucid period, then declining mental status with eventual comatose state. Subdural hematoma is usually a subacute process following head injury or repeated trauma. Headache, nausea, vomiting, and other symptoms of increased ICP may be present.	Focal neurologic deficits may be present depending on location of hematoma. When hematoma is large enough to cause mass effect, Cushing's response may be observed. Abnormal pupil examination may be helpful in comatose patients.	Head CT is very sensitive for acute epidural and subdural hematoma. Epidural hematoma appears as a convex blood collection between dura and skull that does not cross suture lines. Subdural hematoma has a more concave or flattened appearance and may cross sutures lines.
Encephalitis/meningitis	Patients may complain of fever, severe headache, neck pain/stiffness, photophobia and general malaise. History of recent infectious illness may be present. With acute encephalitis, patients may present with altered mental status.	Fever, nuchal rigidity and photophobia may be found on examination. Petechial rash may be seen with meningococcus. In severe cases, seizures may be observed. Stupor and coma may be present. Focal motor weakness/aphasia may be present but are uncommon.	In all patients with suspected meningitis or encephalitis, diagnostic lumbar puncture is essential. Antibiotics/antiviral agents should be administered as soon as possible. Dexamethasone should precede antibiotics if bacterial meningitis suspected.
Hyperglycemia	Antecedent polyuria, polyphagia and polydipsia may be elicited on history. General malaise and extreme thirst with dehydration may be present. Often patients are elderly diabetics with difficulty caring for themselves.	Variable and similar to hypoglycemia. Focal neurologic deficits are uncommon, but hyperglycemia should be considered in the patient with focal weakness. Fever may be present from concurrent infectious illness, contributing to poor glycemic control.	Immediate bedside serum glucose measurement is warranted in all patients with altered mental status or focal neurologic deficits.
Hypertensive encephalopathy	Patients present with symptoms of diffuse cerebral dysfunction, and may be confused or comatose. When lucid, patients often complain of headache, visual problems, and/or focal neurologic deficits. Shortness of breath may be present secondary to pulmonary edema.	Severe hypertension is present, with diastolic pressure typically > 130 mmHg. Focal neurologic deficits may be found on physical examination. Signs of CNS dysfunction such as stupor, coma, or seizures may be present.	Hypertensive encephalopathy is a clinical diagnosis in the setting of severe hypertension. Ongoing intracerebral hemorrhage should be sought with head CT. Cocaine-induced sympathomimetic toxidrome should be considered.

(*continued*)

Table 45.7 Differential diagnosis of acute ischemic stroke or transient ischemic attack (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Hypoglycemia	*Variable:* patient may present along spectrum from agitated to comatose, with global weakness to focal neurologic deficit. May perfectly mimic virtually any neurologic illness.	Also variable; focal or generalized weakness may be found on examination. Seizure activity may occur. Hypothermia may be present. Diaphoresis is common.	Immediate bedside serum glucose measurement is warranted in all patients with altered mental status or focal neurologic deficits.
Hyponatremia/ uremia	Symptoms vary from paresthesias to diffuse weakness/fatigue to focal motor weakness. History of renal failure may be offered.	Variable presentation. Focal motor findings are less common than subjective weakness and paresthesias but may be present.	Electrolyte panel with renal function in the appropriate clinical setting confirms the diagnosis.
Intracerebral/ subarachnoid hemorrhage	Severe headache usually of sudden onset. History of poorly controlled hypertension common. Patient may present obtunded with sonorous respirations.	Hypertension is frequently present. Focal motor weakness may be found and coincides with affected hemorrhagic distributions. With large hemorrhage causing mass effect, Cushing's response may be observed.	Head CT is very sensitive for acute intracerebral hemorrhage. Lumbar puncture should follow a negative head CT in patients with suspected SAH.
Neoplasm	Patients may present with acute or subacute weakness, and a history of longstanding headaches. Acute severe headache with weakness may occur with sudden neoplastic hemorrhage. Symptoms of increased ICP may be present.	Signs are largely variable. When motor weakness is present, distribution may follow along hemispheric pattern depending on location of tumor. Cranial nerve impairment often present with brainstem tumor. Signs of increased ICP may be present.	Head CT fairly helpful in detecting major neoplasm, but further radiographic evaluation such as MRI usually needed to delineate mass. CT is very sensitive for hemorrhagic neoplasms.
Psychiatric	*Variable:* history of psychiatric illness or prior similar presentation helpful. Secondary gain may be an issue or patient may not be consciously producing symptoms (conversion disorder).	*Variable:* physical examination "tricks" useful to delineate true motor weakness from psychogenic weakness. Conversion disorder very difficult to identify.	High index of suspicion with psychiatric history helpful. Always a diagnosis of exclusion when stroke symptoms are mimicked.
Septic embolus with bacterial endocarditis	Patients may present with fever, general malaise and focal weakness. A history of valvular heart disease and/or IV drug abuse may be elicited.	Classic findings in acute bacterial endocarditis include fever, heart murmur, Roth's spots, splinter hemorrhages, Janeway lesions and Osler's nodes. CNS emboli give rise to hemispheric strokes and resulting focal motor weakness and/or hemiparesis.	Work-up includes head CT, which may demonstrate multiple infarctions caused by showering of emboli. Obtain three sets of positive blood cultures and transesophageal echocardiography in the appropriate clinical setting. Urinalysis often abnormal.
Todd's (postictal) paralysis	The key is the history of having had a seizure. Following seizure, patient presents with focal neurologic deficit(s).	Any array of neurologic deficits may be present, including focal motor weakness, sensory loss, ataxia, aphasia, or all of those listed, mimicking hemispheric stroke. Postictal decreased level of consciousness may be present.	Diagnosis of exclusion and based on history. Traumatic brain injury with or without epidural or subdural hematoma must be considered in a patient with unknown history. Drug levels should be sent to ensure therapeutic levels, and other causes of seizure should be entertained.
Toxicologic	*Variable*: depends on particular toxin.	*Variable*: depends on particular toxin. Focal motor weakness may be observed with a number of toxins.	When toxin suspected, toxicology screens and/or specific toxin levels are ordered.
Trauma	*Variable*: depends on injuries present.	*Variable*: depends on injuries present. Focal neurologic deficits may be present with peripheral arterial/nerve injuries, whereas hemispheric deficits may be seen with intracranial injuries. Spinal cord injuries may present with profound sensory/motor deficit at level of lesion, or may demonstrate central cord syndromes.	A thorough work-up of any trauma patient with neurologic deficits is essential. Emergent head CT, peripheral angiography, or spinal MRI may be needed depending on specific injuries/deficits noted.

BP: blood pressure; CNS: central nervous system; CT: computed tomography; ED: emergency department; ICP: intracranial pressure; IV: intravenous; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging; SAH: subarachnoid hemorrhage; URI: upper respiratory infection.

Table 45.8 Differential diagnosis: other selected causes of motor weakness

Diagnosis	Symptoms	Signs	Work-up
Acute transverse myelitis	Patients may present with rapidly developing paraparesis and sensory level deficit with or without severe acute back pain. Accompanying bladder and bowel dysfunction is common. Acute onset implies a vascular etiology, whereas subacute onset may indicate cord compression syndrome from underlying neoplasm.	*Variable examination findings may be seen*: signs similar to spinal cord injury predominate, including paraparesis/paraplegia, sensory level deficit, diminished rectal tone, and combinations. Examination consistent with complete cord transection is uncommon.	Diagnosis suggested by clinical presentation. Emergent MRI essential to identify underlying compressing lesions such as epidural abscess, epidural hematoma, tumor, or herniated disk. Cord infarction may also be identified on MRI. Work-up for MS is indicated if work-up does not identify cause.
Amyotrophic lateral sclerosis	Hallmark of ALS is exhibition of UMN and LMN symptoms in progressive severity over time. Patients may complain of progressive stiffness, slowed speech, and explosive laughter (UMN symptoms) and/or muscle weakness, muscle wasting, cramping and fasciculations (LMN symptoms). Ocular and sensory complaints uncommon.	Muscle weakness with hyperreflexia may be observed. Muscle atrophy and asymmetric weakness and muscle fasciculations (especially in the tongue) may be seen on examination. In advanced cases, respiratory distress from involvement of breathing musculature may be observed. Babinski's sign may be present. Sensation usually preserved.	Diagnosis of ALS is based on clinical presentation with gradual severe progression over time and UMN and LMN signs present.
Botulism	Presentation depends on type: *Infantile botulism*: patient is < 1 year old and presents with poor feeding, constipation, weakness and failure to thrive. Associated with ingestion of food (usually honey) contaminated with infectious spores of *C. botulinum.* *Food-borne botulism*: associated with ingestion of inadequately sterilized home-canned vegetables containing botulinum toxin. Patients present with visual disturbances, dysarthria, dysphonia, dysphagia, and a severe symmetric descending limb paralysis. *Wound botulism:* presents similarly; patients have a history of a wound contaminated with botulinum spores, often with a history of IV drug abuse.	Patients generally exhibit normal mentation with multiple CN abnormalities (diplopia, ptosis, absent pupillary light reflex), along with profound descending bilateral motor weakness. DTRs are usually intact. Weakness of the neck muscles is common. In severe cases, respiratory distress may be present.	Diagnosis suspected with historical factors of honey ingestion in an infant and home-canned vegetables in others. History of IV drug use raises suspicion for wound botulism. Large numbers of patients presenting with symptoms and signs of botulism should prompt alert of terrorist activity. Diagnosis may be confirmed with identification of botulinum toxin in serum, stool, food, or with stool cultures positive for *C. botulinum*. With wound botulism, serum studies are useful, as are wound cultures positive for the bacteria. The wound may need surgical debridement.
Dermatomyositis	Presenting complaints are similar to those with polymyositis (see below) with the addition of rash, which commonly precedes the muscle weakness.	Similar findings to polymyositis with presence of rash. Classic rash is reddish-purple discoloration of the upper eyelids (heliotrope rash) associated with periorbital edema. Scaly erythematous plaques and papules may be seen, especially over the knuckles (Gottron's papules).	Diagnosis confirmed with same testing as for polymyositis, and by skin biopsy. Other autoimmune/connective tissue diseases or concomitant malignancy should be considered (ovaries, GI tract, lung, breast and non-Hodgkin's lymphoma).
Dyskalemic periodic paralysis	Patients may present with localized or generalized motor weakness. Attacks may occur after carbohydrate-laden meal, during rest after strenuous exercise, or during sleep with weakness apparent on awakening. Cold weather may also provoke motor weakness. Attacks may last minutes or several days. With generalized attacks, the weakness usually spreads from proximal to distal. Typically patients present with their first attack in their first or second decade of life.	Generalized or focal motor weakness may be observed. Diminished or absent DTRs are found on examination. Respiratory difficulties and CN abnormalities are not commonly seen.	With primary (inherited) disorders, a family history of similar episodic weakness is highly suggestive of dyskalemic periodic paralysis. Serum potassium levels may be low, normal, or high, but hypokalemic periodic paralysis is most common. Potassium-wasting processes and thyrotoxicosis must be ruled out. The administration of glucose and insulin may provoke an attack within 2–3 hrs.

(*continued*)

Table 45.8 Differential diagnosis: other selected causes of motor weakness (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Guillain–Barré syndrome	Ascending paralysis is the hallmark symptom. Patients usually in third or fourth decade of life. Antecedent GI or upper respiratory viral illness (2–3 weeks prior) may be reported. Muscle or joint pain and paresthesias/dysesthesias in lower extremities may precede ascending weakness. GI infection with *Campylobacter jejuni* may precede illness by 1–3 weeks. Shortness of breath in severe cases. *Miller–Fischer variant* presents as descending paralysis with ataxia/ophthalmoplegia.	Bilateral motor weakness of the lower extremities in ascending pattern over time (may be unilateral). Absence of lower extremity DTRs is a key finding. Sensory deficits may occur but motor findings predominate. CN abnormalities may be present in severe disease. Respiratory distress may be seen in severe cases.	Clinical picture largely makes the diagnosis. CSF studies may demonstrate markedly elevated protein levels without pleocytosis (albuminocytological dissociation); may not be abnormal early in disease course. FVC and NIF used at bedside to predict impending respiratory failure.
Heavy metal toxicity	Presentation usually vague; high clinical suspicion needed. Patients may complain of generalized motor weakness with abdominal pain, muscle aches, memory loss, peripheral edema, and skin rash on hands and feet.	Patients may exhibit sensory loss in a stocking-glove distribution, hyperpigmentation of palms and soles, and delirium on physical examination.	High clinical suspicion needed. Abdominal radiograph may demonstrate radiopaque metallic flecks. Laboratories may show anemia, leukopenia, eosinophilia, and basophilic stippling. Hair and nail clippings may be evaluated for arsenic levels (the most common acute metal poisoning). In acute poisoning, urine arsenic levels may be measured.
Lambert–Eaton syndrome	Variant of myasthenic syndrome most commonly seen in patients with underlying malignancy (usually small-cell lung cancer). Weakness in the limbs and girdle musculature predominates with relative sparing of the bulbar musculature. Dysphagia may be seen. Autonomic dysfunction commonly causes dry mouth, taste abnormalities and impotence.	In contrast to MG, bulbar musculature largely spared. Fatigability less prominent. On examination, strength may actually increase with prolonged contraction. Weakness of pharyngeal musculature may be observed. Reflexes may be decreased or absent.	EMG demonstrates increased response of muscle to each stimulation (in contrast to MG). Tensilon test has no effect, and serology testing for Ach receptor antibodies is negative.
Multiple sclerosis	Monocular visual disturbances common initial complaint (optic neuritis is initial sign in up to 30% of MS patients); diplopia may also be present. Patients may present with neurologic symptoms including motor weakness, spasticity, paresthesias and dysautonomic symptoms, such as sexual dysfunction and GI/GU symptoms. MS is strongly suggested by two or more prolonged episodes of neurologic dysfunction with intermittent recovery, followed by worsening over a period of months. Symptoms commonly worsen with increased ambient temperature, exercise and fever (Uhthoff phenomenon).	Many neurologic abnormalities may be present on examination depending on anatomical pathology of disease. Afferent pupillary defect may be observed with ophthalmoplegia and/or nystagmus. Abnormalities of the optic disk may be observed on funduscopy. Decreased strength, increased tone, hyperreflexia and sensory abnormalities may be present in affected distribution. The patient may complain of electrical shock-like pain down the back and into extremities upon neck flexion (Lhermitte's sign).	Diagnosis is suggested by historical data. Symptoms tend to present with exacerbations followed by recovery with progression of disease. Optic findings without other explainable etiologies highly suggestive of MS. CSF studies may demonstrate discrete oligoclonal bands in gamma globulins. Increased latency in visual-evoked potentials may be observed. MRI of the brain and spinal cord often reveals plaques.
Myasthenia gravis	Most common initial symptoms are double vision and/or ptosis causing blurred vision, especially after hours of reading. Complaint of weakness or extreme muscle fatigue with repetitive use is common, especially jaw weakness after prolonged chewing. Dysarthria and dysphagia may be present. Limb weakness predominates in the upper extremities. Symptoms may be temporally related to heat, pregnancy, emotional stress, infection, and drugs (e.g., aminoglycosides, macrolides, fluoroquinolones, IV magnesium).	Ptosis and ophthalmoplegia may be observed. On physical examination, extended gaze testing often exacerbates muscle fatigue. Pupillary light reflexes and DTRs are preserved. Hallmark is fluctuating weakness that resolves with rest. *Ice testing* on patient's ptosis (ice packs applied to eyelids for 2–5 minutes resulting in improvement) may aid in diagnosis.	*Tensilon test*: 1–2 mg of edrophonium IV is given. Onset of action is about 30 seconds and its effects last about 5 minutes. If no change or problems are observed, an additional 8 mg is infused (may be given in 4-mg increments). For patients in myasthenic crisis, this maneuver results in increased amounts of Ach at the neuromuscular junction and results in improvement in motor function. EMG and serologic testing for Ach receptor antibodies also aid in diagnosis.

(*continued*)

Table 45.8 Differential diagnosis: other selected causes of motor weakness (*cont.*)

Diagnosis	Symptoms	Signs	Work-up
Polymyositis	Patients primarily complain of progressive weakness, over weeks to months, largely in proximal muscle groups. This may be expressed with difficulties rising from a chair, climbing stairs, brushing hair or teeth, and lifting objects over one's head. Proximal muscle pain may be present. Patients may also complain of dysphagia.	Symmetric motor weakness is observed by testing the functions described (e.g., standing from sitting position). DTRs and sensation are intact. Muscle tenderness may be present.	Serum creatinine kinase and erythrocyte sedimentation rate are increased. Diagnosis is confirmed with EMG studies and muscle biopsy. Concomitant malignancy should be considered (less frequent than with dermatomyositis).
Tick paralysis	Patients generally present in late spring and early summer in tick-prone areas. Patients may complain of symmetric ascending muscle weakness or difficulties with coordination. Paralysis usually develops 4–7 days after tick attaches. History of camping or hiking during this time frame may be elicited.	Patients may demonstrate profound ascending flaccid paralysis with dysphagia and dysarthria. DTRs may be diminished or absent. Mental status is preserved. The tick usually remains attached and efforts must be made to find it in all patients presenting with this clinical picture, especially in high-risk environments.	Diagnosis may be obvious in high-risk patient with attached tick. Clinically, syndrome is difficult to distinguish from GBS without presence of tick, but CSF should be normal in tick paralysis. Removal of tick is curative in 24–48 hrs and confirms the diagnosis.

Ach: acetylcholine; ALS: amyotrophic lateral sclerosis; *C. botulinum: Clostridium botulinum*; CN: cranial nerve; CNS: central nervous system; CSF: cerebrospinal fluid; DTR: deep tendon reflex; EMG: electromyography; FVC: forced vital capacity; GBS: Guillain–Barré syndrome; GI: gastrointestinal; GU: genitourinary; IV: intravenous; LMN: lower motor neuron; MG: myasthenia gravis; MRI: magnetic resonance imaging; MS: multiple sclerosis; NIF: negative inspiratory force; UMN: upper motor neuron.

Diagnostic testing

Laboratory studies

Serum glucose

The most common and convincing stroke or TIA mimic is hypoglycemia. Hypoglycemia must be considered immediately in all patients presenting with acute motor weakness. It should be rapidly identified at the bedside and treated with IV dextrose. Treatment usually results in the immediate resolution of neurologic deficits if hypoglycemia is the cause.

Complete blood count

A complete blood count (CBC) is useful for screening for polycythemia-induced hyperviscosity. In addition, the CBC is used to identify patients with thrombocytopenia (an important consideration for thrombolytic treatment in AIS). Certain leukemic or myelodysplastic syndromes increase a patient's risk for TIA/AIS.

Electrolyte panel

Electrolyte panel and renal function are important for identifying uncommon stroke mimics, such as uremia and hyponatremia. Calcium, magnesium and phosphorus levels should also be routinely checked in the patient with acute motor weakness. Dyskalemic syndromes (most commonly hypokalemic periodic paralysis) may be identified on an electrolyte panel, although serum potassium may be normal.

Coagulation studies

Coagulation studies including international normalized ratio (INR) are important in patients taking warfarin, and must be known prior to initiation of thrombolytic or additional anticoagulation therapy in the anticoagulated patient.

Creatine kinase

Elevations in serum creatine kinase (CK) may be seen in patients with myopathies, such as polymyositis and dermatomyositis. CK levels should be measured in all patients with diffuse motor weakness, especially when proximal muscle weakness or muscle tenderness is present.

Cardiac enzymes

Not uncommonly, AMI occurs concomitantly with AIS and TIA. Embolic strokes may occur as a result of clots arising from the heart, as focal myocardial hypokinesis gives rise to local stasis and clot formation. The emergency physician should consider screening for ongoing myocardial ischemia in all patients with suspected acute cerebral ischemia.

Erythrocyte sedimentation rate

Erythrocyte sedimentation rate (ESR) is a nonspecific marker for inflammatory processes. It may also be elevated in the presence of neoplasm. An elevated ESR may be seen in disorders causing acute motor weakness, including polymyositis, dermatomyositis, or Lambert–Eaton syndrome (LES).

Cerebrospinal fluid studies

Abnormalities in cerebrospinal fluid (CSF) studies may assist in the diagnosis of some etiologies of acute motor weakness. Markedly elevated CSF protein levels without pleocytosis (albuminocytological dissociation) are often detected with GBS. CSF in patients with MS may demonstrate oligoclonal bands in the gamma-globulin region. Patients with transverse myelitis have CSF abnormalities, including a markedly elevated white blood cell (WBC) count (>50–100/hpf) with elevated protein.

Heavy metals

Heavy metal levels are generally not helpful in the acute setting, as most test results are not rapidly available to emergency physicians. Therefore, diagnosis of toxicity must be based largely on clinical suspicion. Diagnosis is confirmed with elevated serum levels of the causative agents (i.e., arsenic, lead).

Electrocardiogram

An electrocardiogram (ECG) and cardiac enzyme studies are used to search for concurrent myocardial ischemia. An ECG is also important for the identification of underlying dysrhythmias, especially atrial fibrillation or atrial flutter, which predispose a patient to stroke or TIA. Imbalances of calcium, magnesium and potassium may be detected on an ECG before laboratory values are available.

Radiologic studies

Head computed tomography

Computed tomography (CT) of the head is the most important radiographic study in the emergent evaluation of a patient with AIS. As thrombolytic therapy for ischemic stroke is time-dependent, immediate head CT is a high priority. All efforts should focus on obtaining and interpreting the CT as quickly as possible (preferably by a radiologist or neurologist). Thrombolytic therapy treatment algorithms hinge on the presence or absence of intracranial or intracerebral abnormalities (e.g., bleeding, mass, edema) on head CT. Emergent head CT may also identify non-vascular lesions of the brain (e.g., neoplasm, subdural hematoma), which may present similarly to stroke or TIA.

In patients presenting with AIS beyond emergent therapeutic time windows, CT scanning remains a useful screening tool for non-vascular lesions, and helps delineate the anatomic distribution and extent of the stroke. CT can identify vasogenic edema or mass effect associated with large ischemic strokes, which may contribute to dangerous increases in intracranial pressure. When patients present outside of the window for emergent stroke therapy, MRI may be the preferred initial imaging study.

CT scanning is rarely of use in patients presenting with generalized weakness. It has relatively low yield in patients with transient neurologic symptoms, especially if these symptoms have resolved (MRI is preferred if neuroimaging is indicated). When patients present with signs and symptoms of peripheral neuropathy or radiculopathy, head CT is not indicated.

CT angiography and CT perfusion studies now have greater roles in emergent stroke management, especially in the acute phase of stroke evaluation. CT angiography can identify large proximal arterial occlusions that may be amenable to interventional therapies. CT perfusion studies may help characterize acute ischemic events in clinically vague situations, and delineate large areas of salvageable penumbral tissue when time of symptom onset is difficult to ascertain. CT angiography of the head and neck is also an effective means to evaluate the posterior circulation in patients with suspected vertebrobasilar disease.

Carotid duplex scanning

Carotid duplex scanning does not presently have a role in the emergent work-up of AIS. However, carotid imaging may identify TIA patients with carotid stenosis amenable to carotid endarterectomy. Because patients with TIA are at significant risk for AIS (approximately 5.3% risk of stroke within 48 hours, 10.5% risk at 90 days), it is imperative to identify patients at greatest risk and those for whom surgical intervention may prevent future stroke as early as possible. Carotid duplex imaging is most commonly performed during hospital admission for a patient with an acute diagnosis of TIA. However, many centers now offer expedited evaluation for TIA in observation units or from the outpatient setting, using carefully established rapid diagnostic protocols.

Echocardiography

Echocardiography can identify cardiac thrombi, often the source of embolic cerebral ischemic events. It may also identify valvular disease, wall motion abnormalities, or low ejection fraction states that increase the risk of cardioembolic etiologies for AIS. All patients with ischemic stroke or TIA and evidence of cardiac dysrhythmia (e.g., atrial fibrillation) or ischemia should undergo echocardiography. Echocardiography with bubble study can detect a patent foramen ovale, which may cause shunting and is a potential cause of embolic stroke, especially in younger patients.

Magnetic resonance imaging and angiography

Magnetic resonance imaging (MRI) presently has limited utility in the emergent evaluation of AIS. Availability of emergent MRI is limited at most institutions, and the test itself is time-consuming. However, MRI and magnetic resonance angiography (MRA) are extremely useful in the evaluation of acute TIA. MRI may pick up subtle changes not apparent on CT, and MRA is more sensitive than carotid duplex studies in identifying carotid lesions. MRA is especially valuable in the evaluation of the vertebrobasilar and posterior circulation TIA. Diffusion-weighted

MRI has become the standard for investigation of AIS at many institutions, and is certain to assume a greater role in the emergent evaluation of AIS and TIA.

MRI has tremendous value in patients presenting with signs and symptoms consistent with acute myelopathy. Acute cord compression syndromes due to epidural abscess, epidural hematoma, mass lesion, or vertebral disk herniation are readily identified on spinal MRI. Spinal cord edema resulting from acute transverse myelitis may also be identified.

Other studies

Tensilon test

The Tensilon test is specifically used in diagnosing MG. To perform this test, the physician administers 1–2 mg of edrophonium (a short-acting acetylcholinesterase inhibitor) IV and watches for signs of improvement in the patient's weakness. The onset of action of edrophonium is about 30 seconds and its effects last about 5 minutes. If no change or problems are observed, an additional 8 mg is infused (may be given in 4-mg increments). For patients in myasthenic crisis, this maneuver results in increased amounts of acetylcholine (Ach) at the neuromuscular junction and results in improvement in motor function. Caution must be used with the Tensilon test, as significantly increased amounts of Ach may result in cholinergic crisis with life-threatening bradycardia, atrioventricular block, bronchorrhea, other respiratory difficulty, or diarrhea, salivation and lacrimation. Atropine may be used to reverse the toxicity. In patients with MG who are already on cholinesterase inhibitor therapy, even greater caution must be used, as these patients may present with weakness resulting from underlying medication-induced cholinergic crisis.

Pulmonary function testing

Though not commonly utilized by emergency physicians, bedside pulmonary function testing (forced vital capacity [FVC], negative inspiratory force [NIF]) may be useful in the patient with motor weakness contributing to marked respiratory difficulty. In the patient with severe GBS or MG, progressive respiratory failure may require emergent mechanical ventilation. The decision to intubate the patient may be difficult, as these patients are usually able to compensate well with tachypnea and accessory muscle use.

The need for mechanical intervention may be identified early with the measurement of FVC and NIF by a respiratory therapist. Generally, an FVC <10–20 mL/kg or NIF <20 cm H_2O (critical range measured at approximately –20 cm H_2O to –60 cm H_2O) indicates the probable need for emergent mechanical ventilation in the setting of respiratory muscle weakness.

The use of routine measures of respiratory function, such as pulse oximetry and arterial blood gases, generally does not render an accurate picture of the clinical degree of respiratory failure in these patients. By the time a drop in pulse oximetry and rise in PCO_2 are detected, the patient may be well beyond the need for mechanical assistance.

Swallowing studies

Aspiration pneumonia is a major contributor to the morbidity and mortality of stroke patients. All patients with AIS/TIA should undergo formal swallowing evaluation before oral intake of any kind is allowed.

General treatment principles

In general, most patients presenting to the ED with AIS or TIA do not have problems with the ABCs. Exceptions arise in patients who present with acute hemorrhagic strokes or 2–5 days after a large ischemic stroke, when ischemia-related cerebral edema begins to exert mass effect.

Acute ischemic stroke

Oxygen

No benefit has been observed with the routine administration of oxygen to patients with AIS. Conversely, some evidence exists that suggests that supranormal oxygenation may worsen outcome. Therefore, supplemental oxygen should be reserved for patients with hypoxia, respiratory difficulty, or risk factors in the setting of AIS.

Hyperthermia

Hyperthermia should be treated in all stroke patients, as elevated core temperature is known to be harmful in the setting of AIS. Conversely, there are studies that have demonstrated a decrease in infarct size with lower core temperatures. The presence of fever in the setting of stroke should prompt emergency physicians to consider and investigate concurrent infectious conditions.

Glucose control

Hypoglycemia in the setting of acute motor weakness requires immediate treatment with IV dextrose, which usually resolves ongoing symptoms rapidly. Some studies have found that hyperglycemia may worsen overall stroke outcome by aggravating ongoing neuronal ischemia. Therefore, serum glucose levels greater than 300 mg/dL should be treated with insulin in the setting of AIS.

Hypertension

The management of hypertension in the setting of AIS is somewhat controversial. There is general agreement that elevated BP should not be aggressively lowered in patients with ongoing AIS or TIA. Many patients with stroke or TIA have underlying chronic hypertension and limited autoregulatory capabilities of cerebral circulation. In ischemic situations, the acute lowering of BP may decrease cerebral perfusion and collateral circulation further, leading to infarct extension and acceleration of neuronal injury. Special situations such as ongoing AMI, hypertensive encephalopathy, aortic dissection, congestive heart failure, acute renal failure and thrombolytic therapy for ischemic stroke call for careful control of mean arterial pressure (MAP) in the setting of stroke. In these situations, easily titratable pharmacologic agents are preferred (Table 45.9), and medications that precipitously drop BP should be avoided. The management of hypertension in candidates for thrombolytic therapy differs from other conditions.

Table 45.9 Pharmacologic agents recommended for BP control in non-thrombolytic candidates

DBP >**140 mmHg**	Sodium nitroprusside (0.5 mcg/kg/min). Carefully reduce blood pressure by 10–20%.
SBP >**220** *or* **DBP** >**121–140** *or* **MAP** >**130 mmHg**	Labetalol (10–20 mg IV push over 1–2 min). May repeat or double every 10 min to maximum dose of 150 mg. *Alternative:* Nicardipine (5 mg/hr drip as initial dose); titrate to desired pressure increasing drip by 2.5 mg/hr every 5 min to max of 15 mg/hr.
SBP <**220** *or* **DBP** ≤**120** *or* **MAP** <**130 mmHg**	Antihypertensive therapy indicated only if ongoing AMI, severe CHF, aortic dissection, acute renal failure, hypertensive encephalopathy

AMI: acute myocardial infarction; CHF: congestive heart failure; DBP: diastolic blood pressure; IV: intravenous; MAP: mean arterial pressure; SBP: systolic blood pressure.

Aspirin

The role of antiplatelet medications in the treatment of AIS remains under debate. In the large International Stroke Trial, aspirin treatment provided no significant benefit with regard to death or disability at 2-week or 6-month outcome measures. However, in the same trial there was significant reduction in the recurrence rate of ischemic stroke. Based on this finding and other data, it is currently recommended that aspirin therapy following AIS begin within 48 hours of stroke onset to help prevent recurrent ischemic stroke. Aspirin use before the administration of thrombolytics is not a contraindication to thrombolytic therapy. Aspirin is held for 24 hours following the administration of thrombolytic therapy before being added back to the patient's daily regimen (50–325 mg). Oral aspirin should be withheld to avoid aspiration until a formal swallowing evaluation is completed.

Anticoagulation

The use of unfractionated and low-molecular-weight heparin (LMWH) for the initial management of AIS is somewhat controversial. The current literature, most notably the International Stroke Trial, does not support the administration of unfractionated heparin, as no published data demonstrate sustained improvement in clinical outcome for ischemic stroke.

LMWH has been studied as well; to date there has been no sustained improvement in clinical outcome for AIS following use of LMWH. Many clinical trials have shown that LMWH leads to a significant decrease in the incidence of deep venous thrombosis (DVT) and pulmonary embolism (PE) following AIS, so its use should be considered to prevent thromboembolic complications of bedridden stroke victims.

Thrombolytics

Background

A discussion of thrombolytic therapy for AIS is incomplete without mention of the landmark study that brought IV tissue plasminogen activator (t-PA) to the forefront of stroke care. In 1995, the National Institute of Neurological Disorders and Stroke (NINDS) Study Group published the results of the NINDS trial of t-PA for AIS. Using recombinant t-PA (rt-PA) under a specific set of guidelines, the NINDS investigators demonstrated a significant improvement in clinical outcome at 3 months for patients with AIS.

The NINDS trial consisted of two major parts. Part I enrolled 291 patients and appraised the efficacy of rt-PA based on a 4-point improvement in NIHSS score or complete resolution of neurologic deficit at 24 hours following treatment. Although Part I did not demonstrate significant improvement in 24 hours, there was a statistically significant benefit found at 3 months for the treatment group in four separate outcome measures (NIHSS, modified Rankin scale, Glasgow outcome scale, and Barthel Index). Part II of the NINDS trial followed, enrolling 333 patients and confirming significant clinical improvement at 3 months for AIS patients found in Part I. In a later publication, the significant improvement in clinical outcome was sustained at 1-year follow-up. Overall, AIS patients treated with rt-PA within the strict parameters of the NINDS trial were found to be 30% more likely to have minimal or no disability compared with the placebo group at 3-month and 1-year outcome measures.

The most feared complication of thrombolytic therapy for stroke is symptomatic intracerebral hemorrhage. In the NINDS trial, the incidence of symptomatic intracerebral hemorrhage (ICH) increased 10-fold in the treatment group compared with placebo (6.4% vs. 0.6%). However, there was no significance difference in mortality at 3 months between the treatment and placebo groups in the trial (17% and 21%, respectively), and no increase in the severely disabled group was observed.

Since 1996 there have been other trials in the United States and Canada that have attempted to reproduce the results of the NINDS trial. Research continues to further determine the optimal pharmacotherapy for AIS. At present, only one pharmacologic therapy is approved by the Food and Drug Administration (FDA) for the emergent treatment of AIS. Based on the available data, the treatment of AIS with thrombolytic therapy carries the Grade A recommendation of the Stroke Council of the American Heart Association, and currently provides the best hope for an improved outcome for acute ischemic stroke patients.

Administration of intravenous tissue plasminogen activator (IV t-PA) for acute ischemic stroke

Having made the diagnosis of AIS, the emergency physician should consider thrombolytic therapy with the help of neurology or stroke team consultants. The therapeutic window for IV t-PA lies within 3 hours from stroke symptom onset. This time window was established by the NINDS and subsequent clinical trials in order to optimize outcome and minimize the incidence of ICH. However, new evidence produced by the European Cooperative Acute Stroke Study (ECASS III) suggests that the time window for the administration of IV t-PA can be safely and efficaciously extended to 4.5 hours. As a result, many stroke centers have subsequently expanded their therapeutic window for treatment of AIS under carefully selected circumstances.

Once the decision to give t-PA to an appropriate patient has been made, a positive outcome is more likely the sooner it is administered within this treatment window.

Prior to the initiation of thrombolytic therapy, the inclusion and exclusion criteria are reviewed (Table 45.10). Extensive discussion with the patient and family must take place to provide all available information necessary to make an informed decision. Primarily, this conversation revolves around the potential benefits as well as the risks of thrombolytic administration.

After assessment of the individual risk/benefit profile of the patient, the decision of whether to treat with thrombolytic therapy is made. The dose of IV t-PA for AIS is 0.9 mg/kg (90 mg maximum dose), with 10% of total dose given IV over 1–2 minutes and the remaining 90% infused over 1 hour.

Table 45.10 Indications and contraindications to thrombolytic therapy in acute ischemic stroke

Indications

- Acute ischemic stroke within 3–4.5 hrs from symptom onset
- Age >18 years (recombinant tissue plasminogen activator has not been studied in pediatric stroke)

Contraindications

- Evidence of intracranial hemorrhage
- Suspicion of subarachnoid hemorrhage
- Recent stroke, intracranial or intraspinal surgery, or serious head trauma in the past 3 months
- Major surgery or serious trauma in the previous 14 days[a]
- Arterial puncture at a noncompressible site or lumbar puncture in the last 7 days[a]
- Major symptoms that are rapidly improving or only minor stroke symptoms[a]
- History of intracranial hemorrhage
- Uncontrolled hypertension at the time of treatment
- Seizure at the stroke onset
- Active internal bleeding
- Intracranial neoplasm, arteriovenous malformation, or aneurysm
- Known bleeding diathesis including but not limited to:
  - Current use of anticoagulants, an international normalized ratio >1.7, or prothrombin time >15 seconds.
  - Administration of heparin within 48 hrs preceding the onset of stroke and an elevated activated partial thromboplastin time at presentation
  - Platelet count <100,000 mm^3

[a] In the National Institute of Neurological Disorders and Stroke trial; not present in the package insert.

t-PA may also be given intra-arterially (IA t-PA) at the site of the vaso-occlusive clot by skilled interventional neuroradiologists, but this therapy is generally only available at specialized referral centers with appropriate resources and personnel. The therapeutic window for IA t-PA extends out to 6 hours following onset of ischemic stroke, according to present research. If available, IA t-PA should be carefully considered; although this therapy is widely utilized in stroke centers, it is currently considered experimental and is not FDA approved for the treatment of AIS.

Management of hypertension in thrombolytic candidates requires special attention, as uncontrolled hypertension is a contraindication to thrombolytic therapy. Before treatment with t-PA can be considered, the pretreatment BP must be gently lowered to a systolic pressure <185 mmHg and a diastolic pressure <110 mmHg. Recommendations for the management of hypertension in thrombolytic candidates are based on Advanced Cardiac Life Support (ACLS) guidelines (Table 45.11). If aggressive means are required to hold the patient's BP within an acceptable range, it may be necessary to withhold thrombolytic therapy, as uncontrolled hypertension is associated with a greatly increased risk of intracranial hemorrhage.

Endovascular mechanical interventional therapies

In addition to thrombolytic therapies for AIS, mechanical intervention for the removal of intracerebrovascular thrombi are being employed in some specialized stroke centers. The Mechanical Embolus Removal in Cerebral Ischemia (MERCI) Retrieval System has been shown to be efficacious in the removal of endovascular thrombi and may be deployed in patients regardless of whether thrombolytics have been given. This device has been approved by the FDA and may play a more prominent role in the future treatment of AIS. The time window for deployment is 8 hours from symptom onset, thus providing

Table 45.11 Emergent antihypertensive therapies in acute ischemic stroke (for thrombolytic candidates)

Pretreatment	
SBP >185 mmHg *or* DBP >110 mmHg	Nicardipine drip (5–15 mg/hr) *or* Labetalol (10–20 mg IVP 1–2 doses) *or* nitroglycerin paste (1–2 inch), *or* Enalapril (1.25 mg IVP)
Post-treatment[a]	
DBP >140 mmHg	Sodium nitroprusside (0.5 mcg/kg/min)
SBP >230 mmHg *or* DBP 121–140 mmHg	Labetalol (10–20 mg IVP) and consider a labetalol drip (1–2 mg/min). *Alternative:* Nicardipine drip (5–15 mg/hr)
SBP 180–230 mmHg *or* DBP 105–120 mmHg	Labetalol (10 mg IVP), may repeat and double up to a maximum dose of 150 mg

DBP: diastolic blood pressure; SBP: systolic blood pressure; IVP: intravenous push.

[a] Monitor vitals every 15 minutes × 2 hours, then every 30 minutes × 6 hours, then every hour × 16 hours.

a therapeutic alternative when thrombolytics cannot be given due to time window constraints. The EKOS MicroLysUS catheter system employs high-frequency ultrasonic waves at its catheter tip in hopes of destabilizing endovascular clots to facilitate thrombolysis. This device has also been approved for sale in the United States by the FDA and has been used to treat AIS in specialized centers with promising results.

Post-treatment considerations

Admission to a specialized neurointensive care unit should occur as soon as possible following thrombolytic therapy or mechanical intervention. Neurologic examination and BP monitoring must be performed at regular intervals. If such monitoring is unavailable, transfer to an appropriate facility should be considered.

Transient ischemic attack

There is no specific emergency treatment for acute TIA. Most importantly, the emergency physician must recognize that TIA represents an immediate significant risk for imminent AIS. Recent literature suggests that the risk of AIS following TIA is approximately 10.5% within 3 months, with about half of these ischemic strokes occurring within the first 48 hours following TIA. A neurology consultation and antiplatelet therapy are recommended. If the patient is already on the maximum antiplatelet regimen (i.e., aspirin plus clopidogrel, aspirin plus dipyridamole), then anticoagulation with warfarin should be considered. For TIA patients with critical carotid stenosis on carotid duplex studies, surgical carotid endarterectomy should be considered.

Guillain–Barré syndrome

Depending on severity, patients with GBS benefit from intensive care unit monitoring for therapy and prevention of complications (infections, venous thromboembolism, cardiac dysrhythmias) and for management of associated pain. In GBS, special attention should be paid to involvement of the respiratory muscles and autonomic dysfunction. Ventilatory assistance is necessary when the patient's FVC falls to <20 mL/kg or NIF <20 cm H_2O (critical range measured at approximately –20 cm H_2O to –60 cm H_2O). Respiratory failure requiring mechanical ventilation occurs in nearly 25% of patients with GBS. Symptoms of autonomic dysfunction include various types of cardiac dysrhythmias (mainly bradydysrhythmias) and blood pressure fluctuations. Approximately 10% of patients with GBS die from the disease or its complications; about 20% remain disabled after 12 months.

Further immunosuppressive therapy is generally initiated on an inpatient basis. Intravenous immunoglobulin (IVIG) and plasma exchange are effective treatments that shorten time to recovery. IVIG 0.4 g/kg body weight for 5 days is the preferred treatment modality; alternatively, plasma exchange with the removal of 200–250 mL/kg over 7–10 days can be used. Treatment should be started as early as possible to slow progression of symptoms and accelerate recovery. CSF filtration is currently being studied, but remains controversial at this time. DVT prophylaxis is warranted, as thromboembolism causes significant morbidity and mortality in patients with GBS. There is no evidence supporting corticosteroid or CSF filtration (liquorpheresis) therapies in the treatment of GBS.

Myasthenia gravis

The emergency physician must recognize and manage the acute deterioration of a MG patient based on clinical findings. As with GBS, observing the patient for respiratory compromise is of the utmost importance. Intubation is usually required if FVC falls below 1 liter. Rapid sequence intubation can be safely done in a patient in myasthenic crisis using succinylcholine at doses of 1.5–2 mg/kg, or lower doses of non-depolarizing agents (e.g., rocuronium or vecuronium). The destruction of acetylcholine receptors in MG creates succinylcholine resistance and increased sensitivity to non-depolarizing agents ideally.

Once the airway is appropriately managed, further therapy should be considered. In a known MG patient in myasthenic crisis, reasons for the exacerbation need to be determined and managed. This includes identifying and treating any source of infection, reducing elevated environmental and body temperatures, and controlling emotional stress. Once a patient is found to be in myasthenic crisis (with or without a positive Tensilon test), drugs designed to increase the amount of Ach at the neuromuscular junction can be initiated. Acetylcholinesterase inhibitors are the primary agents used. Pyridostigmine can be used at an initial dose of 60 mg per os (PO) every 4–6 hours. If the patient is intubated or nil per os (NPO), IV pyridostigmine can be used at one-thirteenth the dose. Neostigmine is an alternative acetylcholinesterase inhibitor; dosing is 0.5 mg IV or 15 mg PO. Both pyridostigmine and neostigmine may cause increased airway resistance in patients with concomitant asthma or chronic obstructive pulmonary disease (COPD); inhaled ipratropium may help prevent this side effect.

If patients are not responding to acetylcholinesterase inhibitors alone, steroids and/or other immunosuppressive agents may be added. However, these may take weeks to have an effect and are not practical in the acute setting. Plasma exchange is a relatively rapid adjunct in the management of the myasthenic crisis. Other therapies available outside of the ED include thymectomy preceded by IVIG, plasma exchange, and IVIG. The reason for improvement in MG patients after thymectomy is unclear; interestingly, 75% of patients have an abnormal thymus gland at the time of diagnosis.

Myasthenic crisis might result from the use of certain drugs in treatment of other diseases. Medications that can precipitate a crisis should be avoided unless they are emergently necessary.

Cholinergic crises (precipitated by cholinesterase inhibitor therapy) are often (but not always) accompanied by muscarinic effects. These crises may be treated with incremental doses of atropine but should be managed conservatively if mild. Acetylcholinesterase therapy should temporarily be discontinued.

Lambert–Eaton syndrome

There is no specific treatment for LES. Fortunately, the syndrome does not usually lead to respiratory or bulbar muscle failure. IVIG and possibly plasma exchange should be considered in the inpatient setting.

Multiple sclerosis

In the acute care setting, effort should be directed at eliminating exacerbating conditions such as fever or infectious processes. Respiratory compromise should be managed aggressively. Due to decreased gastric motility, MS patients have an increased risk of aspiration when endotracheal intubation is required. Due to vesicourethral dysfunction, MS patients are at a higher risk of urinary tract infections, especially if the post-void residual exceeds 100 mL. High-dose methylprednisolone and adrenocorticotropic hormone (ACTH) may shorten MS exacerbations. Consultation with a neurologist and admission are usually required.

Acute transverse myelitis

There is no proven effective treatment for inflammatory transverse myelitis, but corticosteroids are often administered in the acute setting to reduce inflammation. Up to one-third of patients suffering from transverse myelitis recover spontaneously. For acute episodes resulting from compressive lesions, emergent surgical consultation is required for immediate decompression.

Polymyositis or dermatomyositis

Treatment of polymyositis and dermatomyositis involves supportive care along with immunosuppressive therapy using steroids, methotrexate, or other agents (monoclonal antibodies, IVIG, azathioprine, cyclosporine), as well as extensive physical and occupational therapy.

Hypokalemic periodic paralysis

Treatment involves supportive care and potassium replacement. Acetazolamide 125–1,500 mg/day is used for prophylaxis.

Botulism

Treatment of botulism consists mainly of supportive care with special attention to respiratory status. Gastrointestinal decontamination should be considered in food-borne and infantile botulism. A trivalent botulinum antitoxin is available, generally reserved for severe cases and avoided in infantile botulism. The trivalent antitoxin is equine-derived, making acute allergic reaction and serum sickness a concern. Infantile botulism is generally not treated with antibiotics, as this may lead to further lysis of *Clostridium botulinum* in the gut and increase the infant's toxin load. Infants benefit from human botulism immunoglobulin (BIG). The trivalent antitoxin should be avoided in infantile botulism.

Heavy metals

Treatment of heavy metal poisoning is generally withheld until a definitive diagnosis is established. Dimercaptosuccinic acid (DMSA) and dimercaprol (BAL) are heavy metal chelators used in arsenic poisoning. BAL chelation is the preferred therapy for acute poisoning and is given intramuscularly at the dose of 3–5 mg/kg every 4 hours initially (avoid in patients with peanut allergy). DMSA (10 mg/kg) may be given orally every 8 hours. Whole bowel irrigation with polyethylene glycol electrolyte solution can be used as an adjuvant therapy.

Tick paralysis

In addition to supportive care, treatment of tick paralysis involves removing the tick, which results in full recovery in 24–48 hours. Evaluation of any patient with the acute onset of motor weakness should include examination for the presence of a tick, especially in high-risk geographical regions.

Special patients

Special consideration should be given to very young children and elderly patients with generalized acute motor weakness, as mild progression of disease may lead to rapid respiratory decompensation. Patients with sickle cell disease are at high risk for cerebrovascular events even at young ages. Symptoms of underlying major depression are common in patients carrying a diagnosis involving long-term progressive motor weakness, such as ALS or MS; ensure that adequate resources are made available to patients. In addition, make an effort to ensure that the family members and other caregivers of these patients are given outside resources and a chance for respite, if needed.

Disposition

Many experts feel that all patients presenting to the ED with AIS should be admitted to the hospital. Coordinated inpatient efforts should focus on improving the patient's overall outcome, whether or not the patient received thrombolytic therapy. Occupational and physical therapy teams should be involved with the patient's care, as well as speech therapy when appropriate. Rehabilitation efforts should be initiated as early as possible, with special attention to signs of depression, a significant comorbidity of ischemic stroke. Swallowing evaluations help prevent aspiration pneumonia, and DVT prophylaxis should be initiated if patients are bedridden. Some patients may be discharged home after a short hospital stay, but many require extended care in rehabilitation facilities.

For TIA, virtually all patients should be admitted for a complete work-up; the only exceptions are patients in whom a comprehensive evaluation, including laboratory studies, head CT (and in many institutions CTA), carotid Doppler imaging, possibly MRI/MRA, and neurology consultation can be carried out in or from the ED in a timely fashion. Because such an expedited work-up and consultation is not always possible, admission for appropriate work-up and pharmacotherapy determination is recommended in many situations.

In the case of other etiologies of acute motor weakness, the emergency physician's assessment of overall respiratory function is the key to appropriate patient disposition. Patients in whom moderate to severe respiratory compromise exists or is anticipated require admission to an intensive care unit. Others with the potential for respiratory compromise should be admitted and adequately monitored. As a general rule, patients with a diagnosis of acute motor weakness should be admitted for observation and education. If urgent neurology consultation and prompt follow-up are available, less severe cases may be sent home if the patient and family members are comfortable with this arrangement. However, discharge home should only be considered in cases where rapid progression of disease is unlikely and thorough patient education has occurred.

Pearls, pitfalls and myths

- There is no substitute for a careful, thorough neurologic examination. Ample time practicing and reviewing the proper and complete technique helps determine the diagnosis, treatment and disposition of patients presenting with acute weakness.
- A complete neurologic examination includes mental status, CNs, motor strength in all extremities, gross sensation, DTRs, and cerebellar function in *any* patient with the complaint of weakness, dizziness, headache, or visual problems.
- AIS therapy is extremely time-dependent. Protocols, designed to streamline acute stroke patients into the optimal care scenario *before* the patient arrives, are warranted.
- Consider TIA as "unstable angina of the brain," and treat these patients with the proper urgency. Patients with true TIA should not be discharged without adequate work-up *and* initiation or advancement of antiplatelet therapies.
- When the diagnosis is in doubt, a formal neurologic consultation for the patient with true acute motor weakness should be requested.
- If consultation and/or the proper imaging techniques are unavailable, admit or transfer the patient as the situation dictates.
- Intravenous t-PA is not contraindicated in elderly patients who meet all inclusion criteria.

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Section 3

Unique Issues in Emergency Medicine

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46 Child abuse, elder abuse, intimate partner violence

Carolyn J. Sachs, MD, MPH

Scope of the problem

Emergency physicians are specialists dealing with violence-related problems. Emergency physicians treat both victims and perpetrators of violence, often on a daily basis. Additionally, emergency department (ED) staff may become the target of violence at the hands of their patients or their patient's families and associates. This chapter covers treatment of patients suffering from child abuse, intimate partner violence (IPV) and elder abuse. Management of the violent patient is covered in Chapter 11.

In the United States, more than 3,000 children, women and elders die yearly from abuse. Additionally, there are 3 million cases of child abuse, 2 million cases of elder abuse, and 2–4 million cases of IPV reported each year. Emergency physicians are in a unique position to identify abusive situations before they result in permanent physical or psychologic disability or death. Research has demonstrated that the majority of IPV homicide victims were seen in local EDs or other health care settings the year before they were killed in one Midwestern city.

Due to the relative isolation of many victims, a visit to the ED may be the only opportunity for abuse detection. Recognition of victimized individuals often requires a high degree of examiner suspicion. Although physical injuries may be the presenting complaint of many abused patients, these victims (or their caregivers) may not disclose the true mechanism of injury. Victims may fear retaliation by the perpetrator or ambivalence about separation. Caregivers may not disclose the abuse because they themselves are the abusers, or because they are unaware of the abuse by another person. Furthermore, the majority of reported cases of child and elder abuse involve neglect, which may present with medical problems resulting from poor nutrition, poor hygiene, or lack of needed medications and care.

Elder neglect is defined as the refusal or failure of a caregiver to fulfill his or her obligations or duties to an elderly person, including (but not limited to) providing food, clothing, medicine, shelter, supervision, medical care and services that a prudent person would deem essential for the well-being of another.

Intimate partner violence is defined as a pattern of assault or coercive behavior of one intimate partner by the other, including physical, sexual and psychologic abuse. IPV is repetitive (victims typically suffer six episodes per year) and often escalates. Women comprise the vast majority of IPV victims, although physicians must consider the possibility of male victims in the appropriate setting. IPV victims are at higher risk for chronic pain, substance abuse, depression and suicide attempts, which makes IPV a significant public health issue. Over a lifetime, at least one out of three women in the United States will be physically assaulted by a partner. Up to 2% of women presenting to the ED are seeking care for injuries inflicted by their partners. Unlike children and dependent adults, victims of IPV are by and large considered competent adults; therefore, the definition does not include *neglect*.

The following discussion focuses on the four main goals of emergency staff when interacting with victims of violence: identification, treatment, documentation and referral.

Identification

According to current research, patients presenting to EDs are supportive of routine questioning by doctors and nurses about violence. Most cases require investigation on the physician's part, as victims rarely disclose abuse without prompting. Preverbal children or demented elders may be unable to provide any history. Patients often feel embarrassed or guilty about their situation. Victims may feel that they deserve the physical violence, as their parent, caregiver, or intimate partner has repeatedly told them so.

In addition, victims may worry about the consequences of legal intervention. Abusers may be arrested and incarcerated. Victims of child and elder abuse may be removed from the home and placed in foster care or an institution. Victims may also worry about further abuse from the perpetrator for having disclosed an abusive situation.

Although inquiring about abuse may seem difficult at first, recognizing that it is important, legitimate, potentially lifesaving, and often legally mandated should help clinicians overcome their initial resistance. Clinicians can help patients feel more comfortable about disclosing abuse by framing questions in ways that let patients know that they are not alone, that the provider takes this issue seriously, that the provider is comfortable hearing about abuse, and that help is available. With practice, each clinician will develop his or her own style of asking questions about abuse. Patients must be questioned about abuse independently (i.e., without family members or friends present).

1. *Framing questions*. Sometimes it is awkward to introduce the subject of abuse. The following are examples of ways providers can introduce the topic:
   - "Because violence is common in patient's lives, I ask every patient I see in the ED about violence."
   - "I don't know if this is a problem for you, but many patients are dealing with abusive relationships. Some are too afraid or uncomfortable to bring it up themselves, so I've started asking about it routinely."
   - "Some patients think they deserve physical punishment because they have not lived up to someone else's (parent's, caregiver's, or partner's)

expectations. No matter what someone has or hasn't done, no one deserves to be attacked. Have you ever been hit or threatened because of something you did or didn't do?"
  - "Because so many patients I see in the ED are involved with someone who hits them, threatens them, continually puts them down, or tries to control them, I now ask all my patients this."
  - Specifically for caregivers: "It must be difficult to care for such an active child or an elder relative who needs so much attention. How do you cope when you feel frustrated? What sources of respite do you use?"

2. *Direct questions*. However one initially raises the issue of violence, it is also important to include direct and specific questions:
  - "Did someone hit, shove, kick, or otherwise hurt you?"
  - "Has your caregiver, parent, partner, or ex-partner ever hit you or physically hurt you? Has he/she ever threatened to hurt you or someone close to you?"
  - "I'm concerned that your symptoms may have been caused by someone hurting you. Can you tell me about it?"
  - "Does your caregiver, parent, or partner ever try to control you by threatening to hurt you or your family?"
  - "Has your partner ever forced you to have sex when you didn't want to? Has he ever refused to practice safe sex?"
  - "Has your caregiver ever touched you in places that made you feel uncomfortable?"
  - "Has your partner tried to restrict your freedom or keep you from doing things that were important to you, like going to school or work, or visiting your friends or family?"
  - "Does your parent, caregiver, or partner frequently belittle you, insult you, and blame you?"
  - "Do you feel controlled or isolated by your caregiver or partner?"
  - "Do you ever feel afraid of your caregiver, parent, or partner? Do you feel you are in danger? Is it safe for you to go home?"

History

Child abuse

Children presenting with injuries that seem incompatible with the given history or with injuries that have no logical explanation should raise a red flag for abuse. Although the majority of injured children seen in the ED are not victims of abuse, ED staff must maintain a high index of suspicion to identify the approximately 10% of injured children who are abused. To encourage disclosure, examiners must obtain the history in a non-accusatory manner. If the child is verbal, a separate history should be obtained from the child and the caregiver when each is alone (i.e., without other family members or friends present). It may be helpful for an examiner to simply explain that he/she is concerned about the child's safety without blaming any specific person, and will need to call in another person to help with the evaluation (e.g., child protective services or a social worker). A history of mental status changes or seizures should raise concern for intracranial injury from abuse. The most common form of head injury from abuse is due to "shaken baby syndrome," the forcible shaking of an infant resulting in subdural hematomas, retinal hemorrhages and diffuse brain injury.

Elder abuse

In elderly patients, female gender, cognitive impairment and increased dependency are universally considered risk factors for abuse. Certain chief complaints commonly encountered in maltreated elderly include injuries, pressure ulcers, falls, dehydration and functional decline. All of these may be a sign of caregiver abuse or neglect.

Intimate partner violence

Experts consider the following historical factors suggestive of IPV: frequent physician visits for trauma, chronic pain syndromes or gastrointestinal (GI) complaints, delays in seeking medical treatment after an injury, an overprotective partner, injuries during pregnancy, a history of depression or suicide attempts, and a history of prior abuse or abuse in the family. The presence of any of these factors should heighten a clinician's suspicion for violence as an etiology for the visit. Clinicians should be especially diligent in questioning patients who present with depression, anxiety, substance abuse, or symptoms lacking a clear etiology.

Physical examination

The majority of injuries seen in pediatric ED patients are not due to abuse, as active children sustain many injuries unintentionally. These injuries are often termed "accidental" injuries. Most educators prefer the adjective "unintentional" to "accidental" when describing these injuries, as "accidental" implies that nothing could have been done to prevent the injury. In fact, many unintentional injuries can be prevented with safety precautions, such as setting the water heater below 104°F to avoid scald injuries in the bathtub.

Intentionally inflicted injuries (those from abuse) frequently differ significantly from the unintentional injuries. Toddlers learning to walk almost universally display unintentional bruises or cuts to the forehead from frequent collisions with furniture or the floor. Conversely, physical injuries from abuse include bruises without a logical explanation, and many burns, fractures, lacerations, abrasions and head injuries. Perpetrators of abuse may purposely injure victims in areas that are usually covered by clothing. Hence, patients must be completely disrobed to identify such injuries. In children and dependent adults, physical manifestations of neglect may be uncovered during the examination. These include failure to grow and/or reach developmental milestones, dehydration, malnutrition, late-stage bedsores, inappropriate clothing and improper administration of medications. Table 46.1 lists various childhood injuries indicative of abuse.

Table 46.1 Physical indicators of child abuse

- Any unexplained change in mental status should raise concern for occult head injury and "shaken baby syndrome"
- Metaphyseal fractures (Figure 46.9)
- Posterior rib fractures
- Unexplained retinal hemorrhages
- Symmetric extremity injuries
- Multiple injuries at different stages of healing (Figure 46.8b)
- Scapular fractures
- Spinous process fractures
- Sternal fractures
- Vertebral body fractures
- Multiple skull fractures
- Circumferential immersion burns (Figure 46.7a)
- Buttock burns
- Cigarette burns
- Evidence of poor care or failure to thrive
- Blunt instrument marks (belts, bats, rods) (Figure 46.4)
- Patterned burns (Figure 46.6)
- Human hand marks
- Bite marks

Any injury without a logical explanation in adult patients should raise suspicion for abuse. Many of the same injuries described in Table 46.1 should raise red flags for IPV and elder abuse when found in adults. Although unintentional injuries may occur anywhere (given a credible history), certain locations are more difficult to injure accidentally and warrant careful scrutiny. Unintentional injuries tend to occur in a distal and/or lateral anatomic distribution, as these areas have greater exposure and are more likely to be injured when a person runs into objects. Distal and lateral body parts (e.g., outstretched arms, knees and shins) generally provide protection when an individual falls. Central injuries to the face, neck, breasts and abdomen should raise suspicion for intentional trauma. Unintentional injuries also tend to be unilateral. It is rare to sustain symmetrical bruises from an unintentional mechanism. It is always important to consider the history in context of the injury. Examples of intentionally inflicted bilateral injuries include finger tip grab marks on both arms, bruises on the medial aspect of upper arms from having them pinned down by the perpetrator's knees, and inner thigh bruises from forced sexual assault.

Any injury to the genital or rectal area should raise suspicion for sexual assault and abuse. Unintentional injuries to this area, termed *straddle injuries*, are rare. Straddle injuries to the female genitalia usually result in trauma to the anterior portion near the urethra. In females, genital injuries from assault are likely to be more posterior, in the posterior fourchette, fossa navicularis, hymen, and labia minora, but may be found anywhere (Figure 46.1). Females and males may display anal injuries after abuse from sodomy or attempted sodomy (Figure 46.2).

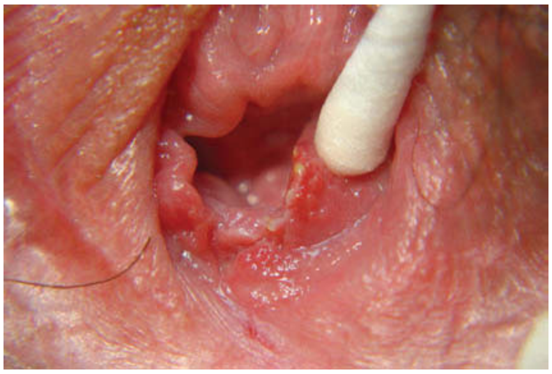

Figure 46.1
Genital injury from assault. 15-year-old non-sexually active female presented 10 hours after sexual assault. The photo demonstrates hymenal trauma with a posterior rim tear and wound necrosis. Courtesy: Malinda Wheeler, RN, MN, CFNP.

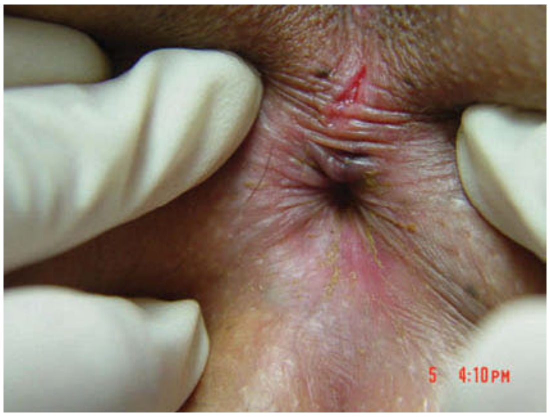

Figure 46.2
Anal injury following sodomy. Courtesy: Malinda Wheeler, RN, MN, CFNP.

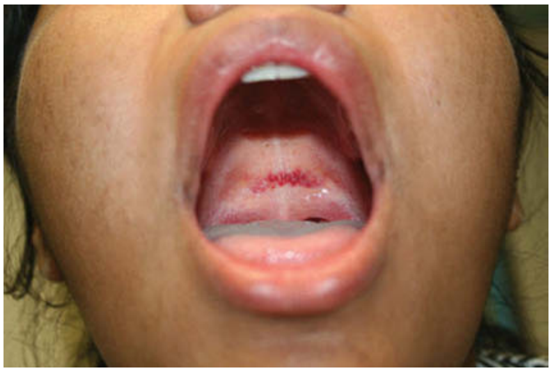

Figure 46.3
Oral petechia after forced oral copulation. Courtesy: Malinda Wheeler, RN, MN, CFNP.

Oral injuries may be found after forced oral copulation of children, intimate partners, or adults (Figure 46.3). Emergency physicians must examine the oral cavity for lacerations, petechiae and contusions in all suspected cases. Intentionally abused victims often display defensive injuries, such as scratch marks from trying to pry

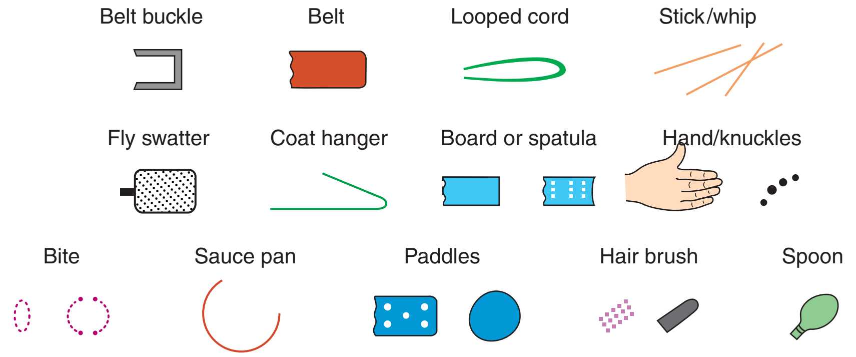
Belt buckle
Belt
Looped cord
Stick/whip
Fly swatter
Coat hanger
Board or spatula
Hand/knuckles
Bite
Sauce pan
Paddles
Hair brush
Spoon

Figure 46.4
Patterns of marks from objects. Reprinted from Johnson CF, Inflicted injury versus accidental injury, *Pediatr Clin North Am* 1990;37(4):791–814, with permission from Elsevier.

off the perpetrator's hands, injuries to dorsum of hands when victim tries to protect his or her face, or forearm contusions and fractures (termed "nightstick" fractures).

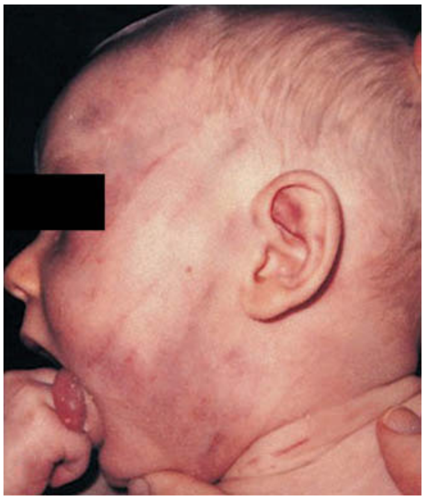

Figure 46.5
The characteristic pattern of parallel lines that results from blows with a belt. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

In patients of any age, "patterned" injuries should raise suspicion for intentionality. Patterned injuries reflect the shape of objects used to inflict intentional injuries (Figure 46.4). They usually have sharper edges and are more geometric than the typical unintentional injury. Common objects that leave patterned injuries include hands, rods, belts and cords (Figure 46.5). After being slapped, a bruise in the shape of the entire digits may be left, with "fingers" visible. Slaps more often result in parallel linear bruises from capillaries breaking at skin areas between fingers. Burn patterned injuries are common in child abuse, but may also be found in IPV and elder abuse (Figure 46.6). These burns occur by three mechanisms: contact burns (e.g., clothes or curling irons), liquid burns (sharply demarcated burns to the wrists or ankles [Figure 46.7] or burns to the genital area inflicted during toilet training), and friction burns (on the torso from being dragged over the ground, or on the wrists and ankles from being tied up).

Diagnostic tests

In cases of suspected child and elder abuse, emergency physicians must evaluate for injuries from prior episodes of abuse. In children, this includes a "skeletal survey,"

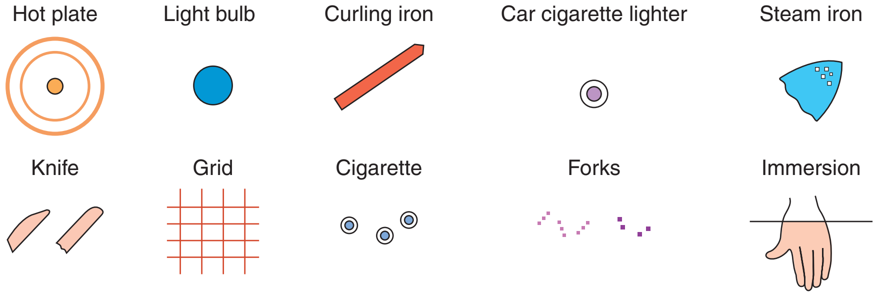
Hot plate
Light bulb
Curling iron
Car cigarette lighter
Steam iron
Knife
Grid
Cigarette
Forks
Immersion

Figure 46.6
Patterns of burns from objects. Reprinted from Johnson CF, Inflicted injury versus accidental injury, *Pediatr Clin North Am* 1990;37(4):791–814, with permission from Elsevier.

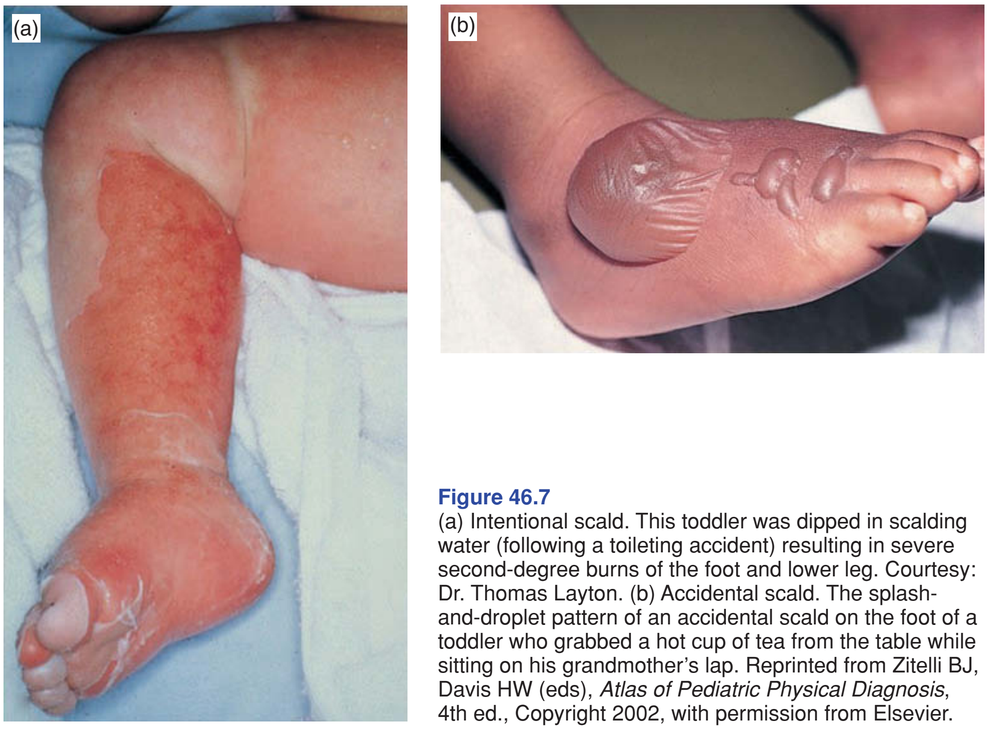

Figure 46.7
(a) Intentional scald. This toddler was dipped in scalding water (following a toileting accident) resulting in severe second-degree burns of the foot and lower leg. Courtesy: Dr. Thomas Layton. (b) Accidental scald. The splash-and-droplet pattern of an accidental scald on the foot of a toddler who grabbed a hot cup of tea from the table while sitting on his grandmother's lap. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

consisting of radiographs of the entire body to detect old or healing fractures (Figures 46.8 and 46.9). Suspected shaken baby syndrome mandates evaluation for intracranial injury with brain computed tomography (CT) or magnetic resonance imaging (MRI), as well as a detailed ophthalmologic evaluation for retinal hemorrhages, if possible. Laboratory studies are rarely helpful in the evaluation of suspected abuse. In clinical situations involving multiple bruises of unknown origin, coagulation studies and platelet testing may help exclude the possibility of a bleeding disorder.

Occasionally, geriatric patients experience such severe abuse and neglect that they are severely dehydrated, or possibly septic from a urinary tract infection (UTI). In this scenario, a comprehensive evaluation including serum chemistries, urinalysis, and radiographs, should be obtained.

Treatment

Child abuse

Most institutions have written protocols for treating victims of child abuse. Emergency physicians should know where to locate these protocols and how to use them. Hospitalization may be indicated for injuries, as well as to allow time for child protective services (CPS) to investigate the source of injury and whether or not the child is safe at home. When there are other children at home, the safety of these children must also be considered.

Appropriate identification of minor abusive trauma and subsequent parental education has the potential to prevent future abuse.

Elder abuse

If the patient no longer has the capacity to make reasonable decisions for him- or herself, law enforcement or adult protective services (APS) should be contacted. APS agencies, established by state statutes, have the ability to assist with immediate evaluation, provide counseling and suggest relocation in suspected cases of elder mistreatment. In some cases of neglect, APS may provide needed assistance to caregivers so that further neglect is avoided and the patient may remain at home. In other cases, APS may establish a court-ordered guardianship or conservatorship to arrange shelter, finances and care. The physician should carefully document the findings of mistreatment or self-neglect and the reasons for declaring the patient incapable of acting in his or her own best interest.

When a patient agrees to intervention, a variety of options can be exercised depending on the type of abuse. If the situation involves physical abuse, severe neglect, or abandonment, and no immediate solution can be arranged, hospital admission is warranted. Admission provides the opportunity for necessary medical treatment and additional time to activate the appropriate social support resources. It also separates the victim from the abuser. Most often, medical complications for a specific problem (e.g., decubiti or dehydration) warrant admission independent of the abuse.

In non–life-threatening situations, a solution can be tailored to fit individual circumstances. Even though the caregiver may be the source of abuse, she/he is also likely to provide the greatest amount of support for the victim. Whenever possible, treatment includes crisis intervention with family members. Options for support should be provided to the family in an attempt to diffuse

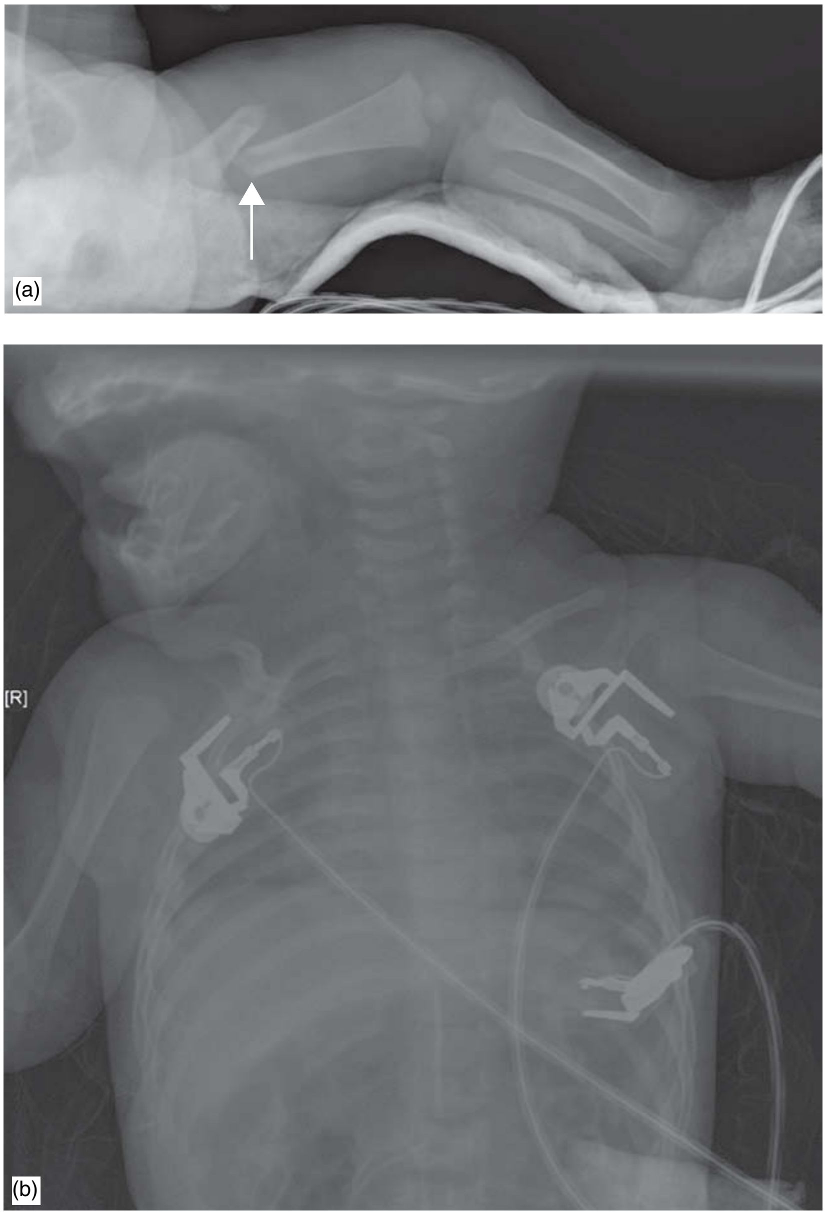

Figure 46.8
(a) Lateral radiograph of the left femur of 6-month-old infant revealing a displaced, rotated mid-shaft femoral fracture resulting from child abuse.
(b) Chest radiograph of the same child demonstrates healing rib fractures bilaterally.
Courtesy: S.V. Mahadevan, MD.

the stress and anxiety that preceded the abuse. Examples include home health aides, respite services, day programs, or transportation assistance if the caregiver is overburdened or ill-equipped to deal with the patient's needs.

Intimate partner violence

Victims of IPV often present with traumatic injuries or medical conditions requiring treatment. Physical symptoms should not be minimized nor ascribed solely to IPV. The main focus of therapy is often treating the victim's psyche. Brief supportive counseling by physicians may result in a dramatic catharsis for a victim who has been suffering in silence. It is very important for the physician to let a victim know that the abuse and violence is *not* her or his fault. Victims universally feel that they did something to deserve the abuse. Abusers repeat this message to their victim with every beating. A few kind, supportive words from an authority figure such as a physician goes a long way in alleviating some of the guilt and shame a victim feels once her abuse has been revealed.

Several comforting phrases to use after any type of abuse is uncovered include:

1. "No one deserves to be physically abused. It doesn't matter what you did or he said you did, you do not deserve to be hit."
2. "You are not the only one who has suffered in this way. Family violence is a common problem."
3. "You don't have to deal with this alone. We can help you and provide connections with resources that can help you."

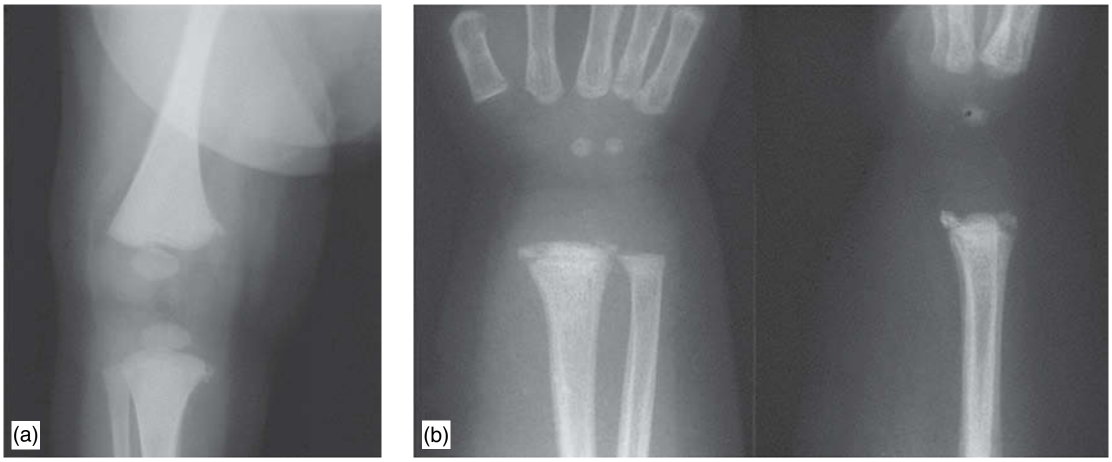

Figure 46.9
Metaphyseal fractures. (a) Metaphyseal “chip” fractures involving the medial aspects of the distal right femur and proximal tibia were found in this infant whose mother confessed to repeated episodes of shaking, after which she would throw the baby down onto a bed or couch. Note the subperiosteal new bone along the lateral aspect of the femur and medial margins of the tibia. (b) Metaphyseal chips are seen on either side of the radial metaphysis in the AP view along with a faint central metaphyseal lucency. In the lateral projection, metaphyseal chips of both radius and ulna are evident. Subtle rims of subperiosteal new bone are present along the diaphyses of both bones. Reprinted from Zitelli BJ, Davis HW (eds), *Atlas of Pediatric Physical Diagnosis*, 4th ed., Copyright 2002, with permission from Elsevier.

Documentation

Most states provide specific reporting forms for child abuse, child sexual abuse, sexual assault and elder abuse. Some states also provide forms to document IPV and other violent injuries. Most of these forms are available in the ED. Practitioners must be familiar with these forms and the organizations (i.e., CPS, APS, law enforcement) to whom these forms must be sent.

Documentation of family violence in the medical chart may be the only written evidence of abuse, and may play a crucial role in assisting the patient. Appropriate documentation by the physician can be crucial in subsequent legal proceedings against the perpetrator or in child custody cases. Many district attorney offices file charges against perpetrators of family violence based solely on carefully documented medical, protective service, and police records, and do not require that the victim press charges or testify in court against the perpetrator. Clear physician documentation can make or break a case in these circumstances.

Guidelines for effective documentation include the following:

1. Record what the patient and/or caregiver tells you using exact words in quotations.
2. Spontaneous utterances such as “He said he was going to beat me until he killed me” can be used in court and for danger assessment.
3. Record prior incidents of abuse, including use of or access to a weapon.
4. Specific threats made by the perpetrator should be recorded in quotations.
5. Record any inconsistencies that lead you to suspect abuse despite caregiver or patient denial.
6. Record the name and relationship of the alleged perpetrator and the time, date and place of assault, using the exact address if possible.
7. Record details of the injury and identify its location on a body map.
8. Record old injuries as well as new ones.
9. Document the services provided during the visit, either in the physician note or the social worker/victim advocate note. These include CPS or APS reports made, physical evidence given to police, photographs taken, referrals given, assessment of the patient’s safety and subsequent medical care offered or recommended.

Photography

Carefully obtained photographs of injuries provide victims and protective agencies with permanent evidence of the assault, even after the injuries have healed. As with appropriate written documentation, photographs can make a difference in subsequent legal proceedings.

Clinicians have several options for photographing injuries; the best method depends on what is available in each setting. Instant photography (i.e., Polaroid) is convenient and often used in EDs. Photographs can be affixed to the chart and are extremely valuable in court, but may lack high-resolution capabilities. Other types of photography (film, digital, or video) can be used as long as a secure chain of evidence can be established. Ideally, every step of the process should be accounted for and signed over on a written form. Most providers that perform sexual assault examinations have a formal protocol for dealing with photographic evidence. It often involves handing over film or a copy of the digitally recorded images to law enforcement at the end of the examination.

These protocols can be easily adapted to family violence photography. Alternatively, law enforcement increasingly carries the needed equipment to obtain photographs after being alerted to the injuries by medical staff.

Although not mandated by all states, consent for photography is required in competent patients by most institutions. In children and dependent adults, when the legal guardian is the suspected perpetrator, most state laws allow photographs and evidence collection without guardian consent. Clinicians should be familiar with their own state and institutional policies.

Photographs should include a standard, such as a ruler, so the size of the injury can be easily determined. With proper photographic documentation, forensic experts can comment on the likelihood that a particular person or a given weapon caused a particular injury. Retrospectively, properly photographed and measured bite marks can often be matched to potential perpetrator. At least one photograph that includes the patient's face with the injury is recommended so the identification of the injured person cannot be challenged. All photographs should be labeled with patient information, date, time, location, and photographer's name.

Physical evidence

Victims of family violence who are also recent victims of sexual assault must be offered an evidentiary examination including the standard physical evidence collection per state protocol and federal Violence Against Women Act (VAWA) funding legislation. In typical cases of family violence, physical evidence, when applicable, should also be collected and turned over to police using the format stipulated by sexual assault protocols. Appropriate specimens include torn or bloody clothing, saliva from bite marks, and bullet, glass, or other weapon fragments. All evidence should be placed in paper (not plastic) bags to avoid bacterial overgrowth and decomposition of the specimen. As with photographs from sexual assaults, transfer of this material must be documented through a written "chain of evidence."

Referral

After identification of abuse, referral is perhaps the physician's most crucial intervention for dealing with victims of violence. Identification, documentation and treatment of injuries mean little unless a victim or caregiver is given the resources needed to change the situation. In some cases, government agencies remove the victim from the dangerous situation. These agencies may take legal action against the perpetrator so others will not suffer similar abuse.

In general, emergency physicians have neither the time nor the expertise to comprehensively counsel a victim or caregiver. Referral services fulfill this important obligation. The level of referral depends on the nature of the abusive situation and, in competent adults, the victim's desire for intervention. Many hospitals and clinics have a dedicated social worker on staff to respond to the ED, and often have a specific team of clinicians that helps evaluate suspected cases of child abuse, sexual assault and elder abuse. Many EDs have an affiliation with a victim's group or local woman's shelter that will assist in caring for victims of IPV. Under some circumstances, these groups will dispatch a representative to the medical setting for immediate counseling, legal advocacy and placement (if necessary).

The revelation of abuse in the medical setting often comes at a time of crisis. This creates a window of opportunity for intervention, at a time when a victim or caregiver is more likely to agree to or desire a change in their situation. Immediate contact with experienced advocates often makes a tremendous difference. Medical personnel may be unaware of these services within their institution or community. The names and numbers of local services must be included in a written ED protocol. In fact, the Joint Commission and several state laws mandate a written protocol for treatment and referral of victims of family violence. Web-based information often provides links to local resources in the user's area.

Furthermore, information on state laws pertaining to abuse can be found through each state's legislative web site.

Child and elder abuse hotlines are available in all locations, but vary by jurisdiction. National hotlines may also provide assistance for elder abuse and maltreatment (ElderCare Locator: 1-800-677-1116) and for child abuse and maltreatment (1-800-4-A-CHILD® [1-800-422-4453]). Online state-specific resources for elder abuse can be found at the web site for the National Center on Elder Abuse (http://www.ncea.aoa.gov/ncearoot/Main_Site/index.aspx). The United States national IPV hotline (800-799-SAFE) can be used by victims and practitioners 24 hours a day, and will automatically direct callers to local shelter services. For hearing-impaired victims of IPV, the TTY number is 800-787-3224.

Safety assessment

All victims of child or elder abuse must receive a safety assessment by either the hospital social worker, abuse team, or protective services. In situations of a competent adult without other resources, a physician or nurse may perform safety assessments. Experts feel that danger increases with increasing numbers of positive answers on the safety screen. The danger assessment screen developed by Campbell, after decades of research in IPV homicide, may be used for this purpose (Figure 46.10). Physician assessment of safety should also include an assessment of suicide and homicide risk by the victim. Suicide risk increases in the presence of IPV; homicide of the batterer is possible if the victim feels that this is her or his only way out. Acutely suicidal or homicidal victims warrant immediate psychiatric consultation and admission.

Reporting

Child abuse

All 50 states mandate reporting of any suspected child abuse and neglect by medical professionals to local CPS.

DANGER ASSESSMENT

Several risk factors have been associated with increased risk of homicides (murders) of women and men in violent relationships. We cannot predict what will happen in your case, but we would like you to be aware of the danger of homicide in situations of abuse and for you to see how many of the risk factors apply to your situation.

Using the calendar, please mark the approximate dates during the past year when you were abused by your partner or ex-partner. Write on that date how bad the incident was according to the following scale:

1. Slapping, pushing; no injuries and/or lasting pain
2. Punching, kicking; bruises, cuts, and/or continuing pain
3. "Beating up"; severe contusions, burns, broken bones
4. Threat to use weapon; head injury, internal injury, permanent injury
5. Use of weapon; wounds from weapon

(If **any** of the descriptions for the higher number apply, use the higher number.)

Mark **Yes** or **No** for each of the following ("He" refers to your husband, partner, ex-husband, ex-partner, or whoever is currently physically hurting you.

____ 1. Has the physical violence increased in severity or frequency over the past year?

____ 2. Dose he own a gun?

____ 3. Have you left him after living together during the past year?
3a. (If have never lived with him, check here____)

____ 4. Is he unemployed?

____ 5. Has he ever used a weapon against you or threatened you with a lethal weapon?
(If yes, was the weapon a gun?____)

____ 6. Does he threaten to kill you?

____ 7. Has he avoided being arrested for domestic violence?

____ 8. Do you have a child that is not his?

____ 9. Has he ever forced you to have sex when you did not wish to do so?

____ 10. Does he ever try to choke you?

____ 11. Does he use illegal drugs? By drugs, I mean "uppers" or amphetamines, speed, angel dust, cocaine, "crack", street drugs or mixtures.

____ 12. Is he an alcoholic or problem drinker?

____ 13. Does he control most or all of your daily activities? For instance, does he tell you who you can be friends with, when you can see your family, how much money you can use, or when you can take the car? (If he tries, but you do not let him, check here: _____)

____ 14. Is he violently and constantly jealous of you? (For instance, does he say "If I can't have you, no one can.")

____ 15. Have you ever been beaten by him while you were pregnant? (If you have never been pregnant by him, check here: ____)

____ 16. Have you ever threatened or tried to commit suicide?

____ 17. Has he ever threatened or tried to commit suicide?

____ 18. Does he threaten to harm your children?

____ 19. Do you believe he is capable of killing you?

____ 20. Does he follow or spy on you, leave threatening notes or messages on the answering machine, destroy your property, or call you when you don't want him to?

____ **Total "Yes" Answers**

Thank you. Please talk to your nurse, advocate or counselor about what the Danger Assessment means in terms of your situation.

Figure 46.10
Danger assessment. From Campbell JC, Webster D, Koziol-McLain J, et al, Risk factors for femicide in abusive relationships: Results from a multi-site case control study. *Am J Public Health* 2003;93(7):1089–97.

Failure to report suspected abuse may result in fines, jail time and successful civil suit against practitioners. Forms to report abuse and agencies that accept these reports vary by jurisdiction.

Elder abuse

Presently, 47 states require reporting of elder abuse to APS or law enforcement. Abuse suffered by nursing home patients should be reported under the national Omnibus Budget Reconciliation Act of 1987 (OBRA 1987). This law established state-run nursing home ombudsman programs that receive and respond to reports of neglect or abuse in nursing homes. Nursing home residents must have access to a designated ombudsman for that facility. Suspected abuse in institutionalized elderly must be reported to the state ombudsman, APS, or law enforcement.

Intimate partner violence

Few states have statutes addressing medical treatment of IPV specifically, but IPV reporting falls under other state statutes. Currently, all but five states require some reporting of injured victims of IPV under laws that require reporting of any injured person. Forty-two states require health providers to report injuries resulting from firearms, knives, or other weapons to law enforcement. Additionally, 23 states require reports of injuries resulting from "crimes" or "violently inflicted injuries." A complete list of state reporting laws pertaining to injured patients is provided in the references section.

Conclusion

The entire ED staff plays a critical role in the identification of abused children, intimate partners and dependent elders. Their role continues to be critical in documenting, treating and referring these victims. ED staff must be comfortable questioning patients about abuse, and must be aware of community resources available to aid these victimized populations. It is best to remain an advocate for these patients, and to arrange an appropriate safety assessment following a complete history and physical examination. Detailed documentation and careful evidence collection must be ensured before considering discharge from the ED.

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47 Environmental emergencies

47A DROWNING

Paul S. Auerbach, MD, MS and Ken Zafren, MD

Scope of the problem

According to the 2002 World Congress on Drowning (WCOD), *drowning* is defined as the process resulting in respiratory impairment from submersion/immersion in liquid. It is no longer defined as death by suffocation after submersion in liquid. Terms that are still active include *water rescue* (alert person with distress while swimming, transient or minimal symptoms, who does not require further medical evaluation), *immersion hypothermia* (prolonged exposure to extremely cold water [<10°C or 50°F] with core body temperature < 32°C [89°F]), and *immersion syndrome* (a form of drowning caused by sudden exposure to very cold [<10°C or 50°F] water that induces a lethal dysrhythmia, perhaps due in part to vagal stimulation). *Cold shock* refers to involuntary gasping and hyperventilation when a person is suddenly plunged into cold water. This can lead to aspiration of water and drowning. Previously used terms that are being phased out of the medical literature are near, wet, dry, active, passive, silent and secondary drowning. Outcomes of drowning events can be reported using the Utstein Style for Drowning format, which includes elements for victim information, scene information, emergency department (ED) evaluation and treatment, hospital course and disposition. The consensus from the WCOD was that drowning outcomes be classified as death, morbidity, or no morbidity. Death from drowning is considered to be any cause of death at any time if there is a clear chain of causality.

Drowning statistics typically lag for a few years. According to the Centers for Disease Control and Prevention, in 2005 there were 3,582 fatal unintentional drownings in the United States. Drowning remains the fourth leading cause of accidental death. An additional 710 people died from drowning and other causes in boating-related accidents. Worldwide, it is estimated that at least 500,000 persons die from drowning each year; this estimate does not include mass-casualty incidents. Because drowning is disproportionately an affliction of the young, the average number of years of life lost per drowning fatality is nearly 30 years. Non-fatal drowning episodes are many times more common than are fatalities, but exact data are unavailable. One study estimated that there are 31,000 ED visits and 8,000 hospitalizations annually for childhood drowning in the United States.

More than one in four fatal drowning victims are children age 14 years and younger. Males are more likely than females to die from drowning. Toddlers have the highest risk of drowning, followed by teenage boys. Adolescent and adult males in surf beach drownings may have had a greater total exposure to the water, as well as more frequent exposure to deeper water. Compared with Caucasians, the fatal drowning rates for African Americans, American Indians and Alaskan Natives are significantly higher. Children from low socioeconomic groups drown at a higher rate than children from high socioeconomic groups. More drownings occur in home swimming pools than in any other place. Drowning risk factors include lack of barriers (e.g., pool fencing), uninhibited access to pools, lack of supervision, teenagers in natural water settings, poor choices when boating (e.g., no life jacket), alcohol use and seizure disorders (bathtub is the highest risk site).

Anatomic essentials

After submersion, alert victims attempt to keep their heads (airway) above water. They may hold their breath and struggle or panic. If they are not successful, and the airway is submersed, voluntary breath holding is maintained until respiratory drive ("air hunger") causes an involuntary inspiration – gasping for breath. Water may be swallowed into the stomach, followed by vomiting and pulmonary aspiration of gastric contents and water. Following a variable period of laryngospasm, the alveoli are flooded, surfactant activity is diminished or eliminated, hypoxia ensues and airway reflexes are lost, resulting in further aspiration. The final pathway is cardiovascular collapse, with bradycardia, then asystole combined with global cerebral hypoxic injury. The duration of laryngospasm during the initial aspiration does not alter the pathophysiology of hypoxemia. Occasionally, alveolar rupture with free air release may occur.

Although aspiration of large volumes (11–22 mL/kg) of fresh or sea water would theoretically cause different changes in blood volume and electrolytes, only a very small percentage of victims aspirate sufficient quantities of water to cause measurable effects prior to the moment of death. The drowning process in humans leads to aspiration of approximately 2–4 mL/kg. Electrolyte abnormalities in drowning victims seldom require treatment. Aspiration of any amount of water causes pulmonary damage, which may result in non-cardiogenic pulmonary edema. Pulmonary injury may be exacerbated by inflammation or contaminants in the water, including bacteria, particulate matter, various chemicals and vomitus.

Most drowning victims rapidly become hypoxemic, hypercarbic and acidemic. Initially, this is due to hypoventilation rather than lactic acidosis secondary to decreased tissue perfusion.

Cardiac arrest occurs within 10 minutes of total alveolar flooding or complete airway obstruction (laryngospasm). Cerebral hypoxia also may exacerbate non-cardiogenic pulmonary edema. Hypoxic–ischemic injury to the central nervous system (CNS) may cause long-term disabling neurologic sequelae, including memory problems, learning disabilities, and permanent loss of basic functioning. Fortunately, many patients recover without neurologic damage. Renal failure and coagulopathy, including disseminated intravascular coagulopathy, may also occur.

Hypothermia associated with submersion in cold water may be protective if the beneficial CNS effects (e.g., brain cooling) supersede the deleterious effects of hypothermia. However, it should be noted that the diving reflex (apnea, peripheral vasoconstriction and bradycardia), considered responsible for survival in children after prolonged submersion, may not function to the extent necessary for survival. Similarly, surface cooling may not occur fast enough to account for cerebral protection. It is possible that aspiration of cold water accounts for core cooling sufficient to be neuroprotective.

Prognosis beyond mere survival relates mostly to neurologic recovery. For warm water submersion, the duration and extent of hypoxia, as determined by submersion time and need for cardiopulmonary resuscitation (CPR), have relevance. At one extreme, it appears that submersion time of 25 minutes or more plus CPR for cardiac arrest are associated with certain death. Scoring determinations include the Glasgow Coma Scale (GCS), Pediatric Risk of Mortality Score system (PRISM), and Conn-Modell neurologic classification system. Individual outcomes cannot be predicted in the ED after a severe drowning event, so aggressive resuscitative efforts are appropriate.

History

History is important to establish the occurrence of drowning, to identify precipitating event(s) such as a dysrhythmia, and to provide clues to associated conditions and potential complications.

Did the patient have a coughing, choking, or vomiting episode near a pool or other body of water?

If no one witnessed the event, and a victim is found unconscious in the vicinity of a body of water, a drowning incident should be suspected.

Is there a possible precipitating event that caused the drowning incident?

Alcohol or other drug intoxication, head or neck injury, cardiac arrest, cerebrovascular accident (CVA), seizures and hypothermia are possible causes. It should be noted that neck injury is likely only in the proper setting for trauma.

Is the patient suicidal or a victim of attempted homicide? Is there a possibility of child neglect or nonaccidental trauma?

This may be a clue to associated conditions and possible complications if the victim survives. Body marks, bruises and bony injuries should invoke suspicion for child abuse.

Was the patient diving into the water or surfing? Is there another mechanism for potential head or neck trauma?

If the history is unclear and the setting appropriate, the possibility of head and neck trauma should be considered. Diving or jumping into water from a height should raise suspicion for spine injury.

Was the patient scuba diving?

This may be a clue to decompression sickness ("bends"), cerebral air embolism, or marine envenomation.

What were the water temperature and duration of submersion?

This information will help guide resuscitation strategy. Submersion in water warmer than 20°C (68°F) and submersion longer than 5–10 minutes are predictors of increased mortality. Survival has not been reported in victims who have been submerged in warm water for more than 30 minutes.

Was the water clean or dirty?

Drowning in heavily contaminated water has a worse prognosis and is more likely to lead to infections.

Past medical

Underlying conditions, such as a seizure disorder, psychiatric illness, medications, alcohol or drug use, may complicate management of the drowning victim.

Physical examination

General appearance

The victim may appear well or ill, and may be conscious or unconscious. He or she may appear dyspneic and be using accessory muscles to breathe. Examine the victim carefully for signs of external trauma, including any skin markings consistent with a marine envenomation. If the victim is dead, check hair-covered areas carefully for entrance wounds.

Vital signs

Core temperature should be measured. Infrared ear thermometry should not be used in water-related accidents for clinical decision making. Tachypnea may provide a clue to pulmonary injury. Heart rate, blood pressure and respiratory rate should be evaluated in relation to core temperature. Shock is uncommon in drowning victims; its presence mandates a search for injuries or other causes of hypotension.

Pulmonary

The chest may be clear or there may be wheezing, rales or rhonchi. Absence of adventitial breath sounds does not rule out aspiration.

Neurologic

Patients arriving in the ED awake and alert have nearly 100% survival with normal neurologic status. Those with an altered level of consciousness who are arousable are also likely to survive without neurologic sequelae. Slightly less than half of comatose patients will survive without neurologic deficits, less than one-fourth will survive with neurologic damage, and the remainder will die from hypoxic–ischemic brain injury or from pulmonary edema. Serial assessment of the Glasgow Coma Scale is mandatory. The remainder of the neurologic examination should be guided by suspected illness or injury.

Head to toe

A thorough examination, such as that performed in any trauma victim or medical patient, should be performed in order to identify associated injuries or conditions.

Differential diagnosis

Most cases of drowning will be clinically apparent by history and physical examination (Table 47.1).

Diagnostic testing

Diagnostic testing is not needed in asymptomatic drowning victims. All other patients should undergo laboratory, electrocardiogram (ECG), and radiographic studies as indicated by history and clinical findings.

Laboratory studies

Complete blood count

Complete blood count (CBC) is likely to be normal or show leukocytosis. It is unlikely to change initial treatment, but may be obtained as a baseline or as a clue to associated conditions. Hemolysis may occur.

Serum electrolytes

Electrolytes are likely to be normal. Abnormalities may indicate aspiration of large volumes of water, therefore guiding treatment. Metabolic acidosis is a clue to tissue hypoxia. Low glucose may have caused an altered level of consciousness leading to drowning. Renal failure infrequently results from drowning.

Coagulation profile

This should be obtained in patients with stigmata of coagulopathies.

Arterial blood gas (ABG)

Many authors advocate ABG measurement on all drowning patients. ABG measurement should be done on intubated patients, but its utility in spontaneously breathing patients is controversial. Most of the important information can be obtained from observation of the patient, pulse oximetry and serum bicarbonate. The decision to intubate can be made on clinical grounds. With the increasing availability of end-tidal CO_2 monitoring and the universal availability of pulse oximetry, the use of ABGs has decreased.

Other studies

Ordering a creatine phosphokinase (CPK) should be considered to rule out rhabdomyolysis. Urinalysis may demonstrate myoglobinuria, although this is not usually important in initial management. A carbon monoxide level should be obtained if there is history of possible exposure. Blood levels of aspirin, acetaminophen and other medications may be indicated based on the history. Further laboratory testing should be dictated by the clinical situation and associated conditions that may be known or suspected.

Table 47.1 Diagnostic possibilities in drowning

Diagnosis	Symptoms	Signs	Work-up
Hypoglycemia, CNS injury or infection, intoxication, myocardial infarction, acute disturbances of cardiac rhythm, or an acute pulmonary event may result in a drowning.	History of coughing, choking, or vomiting near water suggests drowning.	Tachypnea is common in drowning; hypothermia may be present. Drowning victim may have wet skin, hair, or clothing. Toxidromes may be present if toxic exposure led to drowning.	Look for other causes of decreased consciousness if history is unclear. Check blood (fingerstick) glucose, and response to naloxone and thiamine. Further studies may include head CT and/or LP.

CNS: central nervous system; CT: computed tomography; LP: lumbar puncture.

Electrocardiogram

An ECG is obtained to determine the cardiac rhythm and any abnormality. Patients with significant dyspnea, chest pain, a history suggesting ischemic cardiovascular disease, or requiring supplemental oxygen due to hypoxemia should have an ECG.

Radiologic studies

Chest X-ray

A chest X-ray (CXR) should be obtained in all symptomatic drowning patients. The initial CXR may be normal, even in persons who later develop significant complications of aspiration.

Other plain films

Cervical spine films and other plain films should be obtained as indicated by the history or findings on physical examination.

Computed tomography

Head computed tomography (CT) may be necessary if the possibility of significant brain injury or edema exists. In drownings associated with traumatic injury, CT of the cervical spine, chest (CT angiography), and/or abdomen and pelvis should be obtained as indicated.

General treatment principles

Airway, breathing, circulation

As with all emergency patients, treatment begins with the ABCs (airway, breathing, circulation). Ventilation should be initiated as soon as possible in apneic patients. If ventilation is inadequate and the airway is clear, the patient should be intubated. Administer 100% oxygen in the field and then be guided by objective measurement. If the patient is conscious, noninvasive ventilatory support, such as continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BiPAP) are excellent alternatives. CPR should be performed if the patient is pulseless, and should be continued until core temperature is determined. If the core temperature is < 35°C (95°F), the patient should be treated for hypothermia. In patients with core temperature 35°C (95°F) or above, CPR is not likely to be successful if continued for more than 30 minutes. Dysrhythmias and other circulatory problems should be treated according to advanced cardiovascular life support (ACLS) and hospital protocols.

Pulmonary management

If there is significant pulmonary involvement, positive airway pressure is generally necessary. Steroids and prophylactic antibiotics are not helpful immediately after aspiration, although some authors recommend antibiotics if drowning occurred in highly contaminated water. Antibiotics are indicated for patients who develop signs of infection. Intubated patients should have a nasogastric tube placed to decompress the stomach. Intrapulmonary instillation of porcine surfactant has been used for acute respiratory failure after drowning; more research or case observations will be required before a recommendation can be made for its routine use.

Neurologic management

Supportive care, with care to avoid hypotension and hypoxemia, is the key to managing patients with neurologic abnormalities. There are numerous protocols for managing cardio-cerebral resuscitation and promoting CNS salvage. These have at times included controlled hypothermia, fluid restriction, hyperventilation, calcium channel blockers, barbiturates and neuromuscular blockade. None of these has been shown to consistently have a greater effect on neurologic outcome than judicious intracranial pressure monitoring and standard approaches to CNS oxygen deprivation, cerebral edema and blood pressure manipulation. It is worth noting that there is a report documenting survival with no neurologic sequelae of two patients utilizing extended therapeutic hypothermia for several days during extracorporeal membrane oxygenation after drowning and cardiac arrest.

Associated conditions

Associated injuries and conditions should be identified and treated appropriately as in other patients.

Forensic considerations

It is sometimes important for a forensic pathologist to determine the diagnosis of drowning as well as the site of drowning. A number of tests have been evaluated for this purpose, including electrolyte analysis of pleural fluid, microbiological tests to detect common bacterial markers of water fecal pollution, strontium in left ventricular blood, marine bacteria detected in blood, and diatom identification within decomposed organs from a drowned body.

Special patients

Pediatric

Infants and children have demonstrated better ability than adults to survive prolonged submersion in cold water. The possibility of child abuse or neglect should be addressed, with appropriate consultation and reporting according to state law. The psychologic effects on families should be considered when children experience drowning, with appropriate referrals to mental health resources.

Disposition

Historically, all drowning victims were admitted and monitored. The current approach to admission for drowning is more selective. Patients without significant symptoms prior to arrival do not require diagnostic studies and can generally be discharged after a brief observation. Family, social and psychiatric issues should be addressed prior to discharge. If there is any question about the history, if the patient has mild symptoms, or if the patient is a young child, pulse oximetry and a CXR are indicated. If these are normal, the patient should be observed for a minimum of 4 hours. These patients can be discharged if they remain asymptomatic, can be watched closely at home, and have the means to return if necessary. Patients with mild hypoxemia, those who remain symptomatic after observation, or those who develop respiratory symptoms should be admitted to a medical ward. Patients who are intubated and mechanically ventilated require critical care unit admission.

Pearls, pitfalls and myths

Pearls

- Drowning in cold water (<20°C [68°F]) is associated with better survival than drowning in warm water.
- Drowning victims who arrive in the ED without detectable vital signs may survive neurologically intact.
- Initial CXR may be normal, even in patients with pulmonary injury.
- Chest compressions and assisted ventilation improve outcomes in asphyxial cardiac arrest. Therefore, CPR should be started as soon as possible, preferably prior to arrival of emergency medical service (EMS) personnel.
- In drowning patients who are not hypothermic, CPR ongoing for > 30 minutes is unlikely to result in successful resuscitation.

Pitfalls

- Failure to consider the cause of drowning, such as trauma (especially of the head or cervical spine), drug intoxication, hypoglycemia, cardiac arrest, or CVA.
- Failure to address the possibility of nonaccidental causes, such as suicide, homicide, child abuse or neglect.
- Premature discontinuation of resuscitation in a hypothermic drowning victim.
- Reliance on pulse oximetry, tympanic thermometry or other measurements that are not accurate in the setting of hypothermia.

Myths

- Most drowning victims absorb sufficient amounts of water to lead to electrolyte abnormalities.
- The Heimlich maneuver is helpful in drowning victims without foreign body airway obstruction.
- All drowning victims require admission.

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47B HEAT ILLNESS

Ken Zafren, MD

Scope of the problem

In the United States from 1979 to 2003, heat stroke caused over 8,000 deaths. This is greater than the total number of deaths from hurricanes, lightning, tornadoes, floods and earthquakes. However, 10 times this number of elderly patients with underlying cardiopulmonary disease are thought to have died from heat-related complications during the same period.

The rate of death from heat stroke increases markedly during heat waves. A heat wave is defined as at least 3 consecutive days with air temperature rising above 32°C (90°F). In the United States, over 2,000 people died in each of the heat waves of 1980 and 1995. Heat illness is a worldwide problem. The 2003 heat wave in Europe caused 35,000 deaths from heat illness.

The two major heat illnesses are heat exhaustion and heat stroke.

- *Heat exhaustion* is a syndrome characterized by volume depletion. The core temperature is generally < 40.5°C, and mental status is normal. Symptoms include weakness, fatigue and headache. Patients are usually sweating, often profusely.
- *Heat stroke* is a medical emergency characterized by a core temperature > 40.5°C and altered mental status. Patients with heat stroke have hot skin and may or may not be sweating. *Exertional heat stroke* develops over a period of one to a few hours, affecting primarily young healthy people who exercise during hot weather, especially if they are not acclimatized to the heat. *Classic heat stroke* typically develops during heat waves, especially in humid conditions, over a period of days. It disproportionately affects the elderly; the poor, who do not have air conditioning; alcoholics; psychiatric patients; and patients with chronic diseases who are taking medications that increase the risk of heat illness. Mortality is higher in classic heat stroke primarily because of underlying comorbidities. Both forms of heat stroke have a high mortality rate without immediate treatment.

A number of minor heat illnesses have also been described, including heat cramps, heat edema, heat syncope, heat tetany and prickly heat. Malignant hyperthermia is characterized by very high core temperature and altered mental status, but is not considered an environmental illness.

- *Heat cramps* are painful muscle cramps that generally occur after exercise in unacclimatized individuals who sweat freely and replace sweat losses with large amounts of water or other hypotonic fluids. Hyponatremia may also occur in this setting.
- *Heat edema* is a benign condition, most often found in the elderly, in which swelling occurs in the feet and sometimes the hands during the first few days in a hot environment.
- *Heat syncope* is a self-limited condition usually found in unacclimatized persons. Prolonged standing causes venous pooling in the legs which, combined with peripheral vasodilation and volume loss, causes orthostatic hypotension and fainting.
- *Heat tetany* is caused by hyperventilation after brief exposure to intense heat.
- *Prickly heat*, also known as heat rash, lichen tropicus, or miliaria rubra is a maculopapular or vesicular erythematous pruritic rash found in areas of the body covered by clothing. It is caused by obstruction of sweat ducts.

Anatomic essentials

Body temperature regulation is a balance between heat production and heat loss. Basal heat production is approximately 40–60 kcal/m^2 body surface area/hour. Voluntary exercise can increase heat production up to 20 times. Metabolism may also be increased by hyperthyroidism or by ingestion of sympathomimetic drugs. Environmental heat adds to the heat load and can interfere with heat dissipation.

Heat is lost by radiation, conduction, convection and evaporation. Radiation is the exchange of radiant energy with the surrounding environment. Conduction is the exchange of heat by direct contact with a cooler object. Convection is the transfer of heat to (or from) gas or liquid, such as air and water moving by the body. Evaporation is the conversion of liquid to gas, such as sweat to water vapor; this requires energy and removes heat from the body. In hot conditions, conduction, convection and radiation often transfer heat to the body; the evaporation of sweat is the dominant mechanism of heat loss. Evaporation of 1 L of sweat removes approximately 600 kcal of heat.

Heat loss in a hot environment can impose large metabolic demands on the body. Skin blood flow may increase from <0.5 to 7–8 L/min in a hot environment. Exercising in a hot environment may be associated with sweat losses of 1–2 L/hr. Peripheral vasodilation and fluid losses from sweating reduce stroke volume. The heart typically compensates with an increased rate if possible.

Acclimatization is the adaptation of the body to heat stress. After daily exercise in a hot environment for 1–2 weeks, sweat production increases and occurs at lower core temperatures, and sweat contains less sodium chloride. Peripheral blood flow increases. Increased plasma volume leads to higher stroke volume and a lower heart rate, resulting in increased exercise tolerance. Acclimatization is lost over about 1 week in the absence of continued exposure to heat.

Heat exhaustion is a poorly defined clinical syndrome characterized by volume depletion. Various combinations of water and salt depletion can be found depending on the amount of water and electrolytes used to replace fluid losses. Symptoms are similar whether the lost volume has not been replaced ("water depletion") or replaced using water with inadequate salt ("salt depletion"). The terms "water depletion" and "salt depletion" are misleading, since in both cases volume (not free water only) has been lost.

Heat stroke is a life-threatening condition in which the thermoregulatory mechanisms fail, allowing extremely high core temperatures. The brain is particularly vulnerable to damage, but multiple organ systems are affected. Coagulation abnormalities, as well as damage to the liver or kidneys, are common. Cellular damage depends more on the duration of exposure than on the maximum core temperature. In the past, the absence of sweating was considered important to the diagnosis of heat stroke. However, patients with early heat stroke may still sweat. Traditionally, a distinction has been made between "exertional" heat stroke, caused by exercise in a hot environment with increased heat production, and "non-exertional" (classic) heat stroke, due to increased exogenous heat gain and decreased ability to lose heat. In general, exertional heat stroke has a better prognosis than classic heat stroke, but initial treatment in the ED is the same.

History

Does the patient have a reason for heat illness?

The diagnosis of heat illness is usually straightforward. Predisposing factors fit into three broad categories:

- Increased heat gain from the environment
- Increased internal heat production
- Decreased ability to dissipate excess heat

Does the patient have an associated condition predisposing to heat illness or caused by heat illness?

Heat illness may coexist with other diagnoses, such as fever or trauma. Heat illness may cause trauma due to an altered level of consciousness or syncope.

Is the patient predisposed to heat illness by not being acclimatized?

Risk of heat illness is highest in late spring or early summer, during heat waves, and in persons who have recently arrived in warmer climates.

Has the patient had weakness, fatigue, headache, lightheadedness, vertigo, nausea, or myalgias?

These are symptoms of both heat exhaustion and heat stroke, and do not distinguish between the two.

Has the patient had hallucinations?

Hallucinations suggest heat stroke.

Does the patient have vomiting or diarrhea?

These are common in heat stroke, but uncommon in heat exhaustion, and exacerbate volume depletion.

Does the patient have a reason for increased heat gain from the environment?

Exposure to high temperatures and high humidity may lead to heat illness. During heat waves, lack of access to air conditioning is a risk factor for heat stroke. Even during less extreme periods, there are many microclimates that can create considerable heat stress. These include indoor or outdoor areas exposed to direct sun, upper floors of buildings, car interiors, boiler rooms, hot tubs and saunas.

Does the patient have a reason for increased internal heat production?

Has he or she been exercising in a hot environment?

Has the patient been using medications, alcohol, or illicit drugs that increase heat production?

Salicylates increase heat production by uncoupling oxidative phosphorylation. The use of certain drugs can cause increased activity or combative behavior, drug withdrawal, seizures or neuroleptic malignant syndrome. Drugs with sympathomimetic properties, such as cocaine or amphetamines, cause increased muscle activity. Drugs such as phencyclidine (PCP) and lysergic acid diethylamide (LSD) increase metabolism by CNS effects. The drug 3,4-methylenedioxymethamphetamine (MDMA or "ecstasy"), often used at dance parties ("raves"), has prolonged activity that can lead to heat stroke. MDMA can also cause a clinical picture similar to the syndrome of inappropriate antidiuretic hormone (SIADH) that results in high urine sodium and osmolality, despite hypotonic hyponatremia and normal blood volume. Participants at rave parties are often aware of the danger of heat stroke and are told to drink plenty of water. This response exacerbates hyponatremia, which can cause seizures.

Does the patient have a febrile illness causing increased heat production?

The presence of cough, meningismus, or shaking chills suggests a febrile illness due to an infectious etiology.

Does the patient have another metabolic condition causing increased heat production, such as hyperthyroidism or pheochromocytoma?

These conditions can be identified by history or abnormal laboratory tests.

Is the patient dehydrated or volume depleted?

Inadequate fluid intake to replace increased losses from a hot environment can reduce the body's heat dissipation through sweating and peripheral vasodilation.

Has the patient been using alcohol or other drugs that increase fluid losses or interfere with behavioral responses to heat exposure?

Alcohol predisposes to dehydration by inhibiting antidiuretic hormone. The use of alcohol and other drugs may decrease one's level of consciousness or alter judgment, and interfere with the behavioral response of seeking shelter away from hot microclimates. Alcohol and other drugs, especially phenothiazines, may also limit thirst. Any mind-altering substance can interfere with the ability to obtain and drink the fluids required to replace losses.

Was the patient wearing clothing that decreased heat loss?

Clothing that is too warm, especially vapor barrier clothing worn by those trying to lose weight, can markedly decrease heat loss.

Past medical

Cardiovascular disease

Patients with cardiovascular disease may be unable to compensate for changes induced by heat stress, resulting in heat stroke or cardiac complications.

Skin and systemic diseases

Skin diseases that decrease the ability to sweat include eczema, psoriasis, burns or heat rash. Systemic diseases affecting the ability to sweat include ectodermal dysplasia, scleroderma and cystic fibrosis.

Medications

Anticholinergic agents decrease sweating. Phenothiazines impair central thermoregulation and have anticholinergic effects. Cardiovascular drugs, such as beta-blockers, calcium-channel blockers and alpha-agonists, decrease the heart's ability to compensate for the effects of heat and decrease peripheral blood flow. Sympathomimetics limit vasodilation in skin.

Physical examination

The primary goals of the physical examination are to distinguish heat exhaustion from heat stroke, to identify diseases or underlying conditions that may have caused or contributed to heat illness, and to identify other conditions or injuries that require treatment. The emphasis is on heat stroke, because heat exhaustion is not a life-threatening illness.

General appearance

The patient with heat stroke appears ill and most likely will not be sweating. Other than body temperature, alteration of consciousness is the main feature that distinguishes heat stroke from heat exhaustion.

Vital signs

Core temperature is the temperature of internal organs. Its measurement is key to the evaluation and treatment of heat illness. In heat illness, brain temperature is of particular importance, because prolonged exposure to high temperatures can cause permanent brain damage. In heat exhaustion, core temperature is generally <40°C. If altered mental status is present, or the core temperature is >40.5°C, the patient should be diagnosed with heat stroke. Core temperature may be <40°C in heat stroke if the temperature is measured well after the acute event.

The best method of measuring core temperature, especially in intubated patients, is with an esophageal probe. Brain temperature can be accurately measured using an epitympanic probe, which contacts the tympanic membrane. This differs from infrared tympanic temperature measurement, which is unreliable. Unfortunately, epitympanic probes are difficult to obtain in North America.

Rectal probe thermometers have been traditionally used, but rectal temperature changes significantly lag core temperature changes, making it difficult to monitor the success of cooling measures. Bladder temperature is less indicative of core temperature than rectal temperature. Oral temperature varies with respiration and is a poor reflection of core temperature.

In heat stroke, expect tachypnea, tachycardia and normal to low blood pressure. Pulse pressure may be widened. Deviations from this pattern may provide clues to underlying diagnoses. For example, excessive tachypnea may indicate salicylate toxicity. Relative bradycardia may suggest certain infectious diagnoses, such as typhoid fever, or reflect the use of cardiac drugs, such as beta- or calcium channel blockers. High blood pressure may be a clue to thyroid storm or pheochromocytoma. Heat exhaustion also presents with tachycardia.

Neurologic

Any neurologic sign may be found in heat stroke. Dysarthria and ataxia are common, but agitation, stroke-like symptoms, posturing, seizures and coma can all occur. Miosis is often present.

Skin

At the time of collapse from exertional heat stroke, most patients will be sweating profusely. By the time of arrival in the ED, the skin is often hot and dry. The presence of heat rash is a risk factor for heat stroke. Purpura indicates coagulation abnormalities and implies a poor prognosis.

Differential diagnosis

If body temperature is >40.5°C, rapid cooling measures must be initiated immediately. If cardiovascular parameters improve and mental status returns to normal, most other diagnostic possibilities are eliminated. If the core temperature does not decrease or the mental status does not improve, other etiologies must be investigated (Table 47.2).

Unlike heat stroke, most febrile states that produce altered mental status are associated with normal hepatic transaminases. Reye syndrome causes encephalopathy and elevated transaminases without fever.

Diagnostic testing

Laboratory studies

Complete blood count, comprehensive metabolic panel and coagulation profile

CBC, comprehensive metabolic panel (CMP) and coagulation profile are helpful in detecting organ damage and in excluding associated diagnoses. Hemoconcentration is almost always present. Hypoglycemia is common, as are hypokalemia and hypernatremia. Renal failure may occur. Hepatic transaminases are almost always markedly elevated, but may be normal or near normal initially. Coagulation abnormalities imply a poor prognosis.

Creatine phosphokinase

Creatine phosphokinase (CPK) should be measured to assess for rhabdomyolysis. No discrete cutoff exists, but

Table 47.2 Differential diagnosis of heat illness

Diagnosis	Symptoms	Signs	Work-up
Anticholinergic toxicity	Blurred vision	Mydriasis	Clinical diagnosis
Brain abscess, cerebral hemorrhage	Variable – may be identical	Neurologic abnormalities including altered mental status	Head CT
Cerebral (falciparum) malaria	Headache, shaking chills	Variable – may be identical to heat stroke	Head CT, thin and thick smears of blood looking for parasites
Delirium tremens	Anxiety, changes in mental function	Tremors, tachycardia	Careful history
Diabetic ketoacidosis	Thirst, frequent urination, vomiting	Kussmaul breathing, fruity breath, dehydration	Electrolytes, renal function, serum ketones, ABG or VBG
Meningitis, encephalitis	Headache, vomiting often prominent	Meningismus, altered mental status	LP with CSF analysis; other adjunctive studies
Neuroleptic malignant syndrome, malignant hyperthermia	Nonspecific	May be identical to heat stroke, muscle rigidity	Clinical diagnosis based on medication history, response to dantrolene
PCP, cocaine, amphetamine toxicity	Mood disturbances	Variable	History, urine toxicology screen
Salicylate toxicity	Tinnitus	Tachypnea	Salicylate level, electrolytes, renal function
Sepsis	May be identical to heat stroke – sepsis can cause heat stroke	May be identical to heat stroke	Search for source of infection
Status epilepticus	Seizures	May be identical to heat stroke	Usually clinical diagnosis; head CT usually indicated; EEG may be necessary
Thyroid storm	May be nonspecific	Altered mental status; associated stigmata of hyperthyroidism	Thyroid function studies
Typhoid fever	Fever, headache, anorexia, cough, constipation or diarrhea	Relative bradycardia	Blood and stool cultures

ABG: arterial blood gas; CSF: cerebrospinal fluid; CT: computed tomography; EEG: electroencephalograph; LP: lumbar puncture; PCP: phencyclidine; VBG: venous blood gas.

values of >1,000 U/L (approximately 5 times normal) are considered diagnostic. As the rise in CK begins 2–12 hours after muscle injury, early diagnosis and treatment may prevent renal damage.

Amylase and lipase

Both are likely to be elevated if pancreatitis is present.

Urinalysis

Urinalysis (UA) should reveal maximally concentrated urine.

Urine toxicology

Urine toxicology screens may be helpful in identifying drugs of abuse contributing to hyperthermia.

Salicylate level

Abnormal salicylate levels can make the diagnosis of salicylism.

Thyroid panel

Thyroid profile should be obtained if thyroid storm is suspected.

Cerebrospinal fluid

Cerebrospinal fluid (CSF) studies may be indicated if meningitis is suspected as a cause of altered mental status.

Electrocardiogram

An ECG should be obtained to rule out associated diagnoses, including myocardial infarction and drug toxicity.

Radiologic studies

CT of the head may be necessary as part of the evaluation of altered mental status.

General treatment principles

As with all emergency patients, treatment begins with the ABCs (airway, breathing, circulation). The main goal of treatment is rapid cooling. Only after stabilizing measures have been initiated should a more detailed diagnostic work-up be undertaken.

Airway, breathing, circulation

The airway must be controlled. Unconscious patients should be intubated. Supplemental oxygen should be administered. Intravenous (IV) access should be established. Aggressive volume resuscitation may be unnecessary since cooling can be expected to decrease peripheral vasodilation. This decreases the demands on the heart and raises blood pressure. However, patients may be severely volume depleted and may require large volumes of isotonic fluid. Fingerstick glucose may reveal hypo- or hyperglycemia. Continuous cardiac, pulse oximetry and temperature monitoring should be established. Cardiac monitor electrodes can be attached to the patient's back if they will not stick to the chest. A Foley catheter should be placed and urine output monitored. Urine temperature can be measured with a Foley catheter probe if other methods are not available. Placement of an arterial line for continuous monitoring of blood pressure should be considered in hypotensive patients.

Cooling

The patient should be immediately undressed. Ice packs can be placed in the axillae and groin. Evaporative cooling is the method of choice at most institutions. Spraying lukewarm water over the patient and blowing room temperature or even warmed air over the skin prevents cutaneous vasoconstriction and minimizes shivering. As shivering causes undesired heat production, it can be treated with IV medications, such as chlorpromazine 25 mg or meperidine 100 mg. Hypotension is a concern with these drugs. Paralysis with non-depolarizing neuromuscular blocking agents is the treatment of choice for abolishing shivering in intubated patients.

Immersion cooling in ice water is used in some circumstances, primarily in field situations for treatment of exertional hypothermia. This method has a number of practical drawbacks in the emergency department (ED) setting. It may not be safe in patients who are neither young nor healthy. Cardiopulmonary bypass is another alternative but is rarely necessary. It has been used successfully in the treatment of malignant hyperthermia. Peritoneal lavage is also a possibility to decrease core temperature. Venous catheter heat exchangers have been used to produce controlled hypothermia, and are less invasive than cardiopulmonary bypass. They are as yet untested for treatment of heat stroke. Cooling blankets are not effective.

Many experts believe that cooling measures should be discontinued when body temperature reaches 39°C, in order to avoid hypothermia. Although not yet studied, it is possible that mild hypothermia with a core temperature of 32–34°C could improve neurologic outcome in comatose heat stroke patients, as it does in cardiac arrest patients who are comatose after return of spontaneous circulation.

Antipyretics such as acetaminophen and nonsteroidal antiinflammatory drugs (NSAIDs) are effective only in the event of fever and should not be used in heat stroke. Dantrolene is indicated in malignant hyperthermia or neuroleptic malignant syndrome, and has no effect on hyperthermia due to other causes.

Supportive care

Volume status, glucose and electrolyte abnormalities, coagulopathies, seizures and other complications are managed in the standard fashion. Aggressive volume resuscitation is sometimes necessary. Iced normal saline at 4°C may be available in some institutions for inducing therapeutic hypothermia after cardiac arrest. Cold saline can be used in the initial resuscitation, if it is available.

Special patients

Elderly

Geriatric patients have decreased cardiovascular reserve and decreased ability to sweat. Elderly patients may be further limited by cardiac and vascular disease, complicating the management of heat stroke. They often take medications, such as beta-blockers, that decrease their ability to dissipate heat.

Pediatric

Children have a greater surface area-to-mass ratio than adults, allowing for greater exogenous heat gain. They have a higher metabolic rate, increased heat production, and less ability to sweat than adults, limiting their ability to lose excess heat by evaporative cooling.

Obese

Obese patients have a decreased surface area-to-mass ratio and decreased skin blood flow. The physical effects of adipose tissue predispose obese people to heat illness.

Disposition

Patients with minor heat illnesses and most patients with heat exhaustion can safely be discharged home in the company of a reliable adult, with close outpatient follow-up. Infants, the elderly, individuals with underlying conditions predisposing to heat illness or with significant volume depletion, and patients with end-organ damage should be admitted.

All patients with heat stroke should be admitted. Patients who are unstable should be admitted to an intensive care unit. This may require transfer to a hospital offering a higher level of care.

Pearls, pitfalls and myths

Pearls

- Heat stroke presents with altered level of consciousness.
- Consider heat syncope in the differential for syncope.
- Obtain as much history as possible about what the patient was doing and the environmental conditions during the time when the patient collapsed or had changes in behavior.
- Aggressive fluid resuscitation may not be necessary. Aggressive immediate cooling is imperative.

Pitfalls

- Not considering the diagnosis of heat stroke.
- Not measuring core temperature as soon or as often as possible, or using suboptimal methods, such as oral or tympanic thermometers.
- Not considering associated diagnoses, such as trauma or sepsis.
- Not instituting cooling measures promptly.
- Using antipyretics to cool a patient suffering from heat stroke.
- Giving excess fluids to a patient on MDMA, which can worsen hyponatremia and cause seizures.

Myths

- A patient who is sweating cannot have heat stroke.
- A patient in the ED who has a normal core temperature and normal transaminases cannot have heat stroke. (The patient may have cooled off; transaminases may rise only after 12–48 hours).

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47C ACCIDENTAL HYPOTHERMIA

Ken Zafren, MD

Scope of the problem

Hypothermia is defined as a core temperature <35°C (95°F). It can occur at any place and any time. Hypothermia causes over 700 deaths annually in the United States. About half of these deaths occur in patients over 65 years of age.

Accidental hypothermia refers to unintentional hypothermia. It is classified into primary, secondary and iatrogenic types.

Primary hypothermia affects otherwise healthy patients exposed to cold environmental conditions who are unable to maintain core temperature above 35°C. Throughout history, primary hypothermia has been a disease associated with wars. During peacetime, it most commonly occurs in urban areas in association with homelessness and the use of alcohol and other drugs. Also at risk are participants in outdoor activities, such as hiking, skiing, hunting, climbing, sailing, swimming and diving.

Secondary hypothermia is caused by a variety of conditions, including sepsis and trauma. It is also associated with diseases that decrease metabolic rates, such as hypoendocrine states, and conditions that affect hypothalamic function, such as tumors and stroke. Elderly and ill patients with secondary hypothermia are often found indoors in well-heated houses; this may confuse the diagnosis.

Iatrogenic hypothermia may be induced by resuscitation with room temperature fluids or refrigerated blood products. It is a common problem in surgery and neonatology, as well emergency medicine. Ambient temperatures cooler than 28°C in the emergency department (ED) can contribute to iatrogenic hypothermia, especially in trauma and resuscitation rooms.

Therapeutic hypothermia, a form of iatrogenic hypothermia, is used deliberately to induce cerebroprotective cooling, primarily in comatose patients with return of spontaneous circulation after cardiac arrest. This therapeutic modality has been shown to improve survival and neurologic outcomes.

Anatomic essentials

Body temperature regulation is a balance between heat production and heat loss. Heat production can be increased by increasing the metabolic rate from shivering or voluntary activity. Increased metabolic rate is mediated by the thyroid and adrenal glands. Shivering is the most effective means of increasing heat production, but it places a high metabolic demand on the body.

Heat is lost by radiation, conduction, convection and evaporation. These mechanisms are explained in the section on Heat Illness. Wet clothing increases the rate of heat loss up to fivefold, whereas immersion in water increases the rate as much as 25–30 times. Wind and moving water further increase heat loss by disrupting the warm microclimate that can otherwise protect against heat loss from skin. Evaporation of water from the skin surface and from respiration may also cause significant heat loss.

Humans are adapted to tropical environments and have a limited ability to decrease heat loss by physiologic means. The temperature at which an unclothed, resting human in still air can maintain a core temperature of 37°C without increased heat production is 28°C (82°F). This is called the *thermoneutral temperature*. Doubling the metabolic rate decreases the thermoneutral temperature only to 20°C (68°F).

Heat loss can be limited by vasoconstriction and by behavioral responses. The most effective responses are behavioral. These include putting on warm clothing and seeking refuge from cold environments. If these mechanisms are somehow limited, the risk of hypothermia markedly increases.

The cause of death in hypothermia is usually decreased cardiac output. This may be due to decreased cardiac function or fatal dysrhythmia. In contrast, hypothermia protects the brain.

Cardiovascular system. The initial response to hypothermia is increased catecholamine production with peripheral vasoconstriction. This causes increased heart rate, blood pressure and cardiac output. With further cooling, below a core temperature of 35°C, these parameters decrease in a nearly linear fashion. Below 28°C, irritable myocardium causes a marked decrease in the ventricular fibrillation threshold. Effective circulating blood volume decreases to as little as one-third of normal, even in moderate hypothermia.

Central nervous system. Hypothermia causes decreased cerebral blood flow and decreased cerebral metabolism. This protects the brain from ischemic injury in the setting of hypothermia. The cerebroprotective response is the basis for therapeutic hypothermia in comatose patients with return of spontaneous circulation after cardiac arrest. As core temperature decreases, there is a progressive decrease in the level of consciousness. Initial deficits in fine motor coordination and clumsiness are followed by confusion, dysarthria, and impairments of judgment and memory. As core temperature drops, many patients no longer feel cold.

Respiratory system. Initial tachypnea in mild hypothermia is followed by reductions in minute ventilation and oxygen consumption as core temperature decreases. Bronchospasm and bronchorrhea associated with impaired ciliary function can lead to aspiration and pneumonia.

Renal system. In mild hypothermia, peripheral vasoconstriction causes increased renal blood flow and cold-induced diuresis. This decreases intravascular volume. With further cooling, decreased sensitivity to antidiuretic hormone (ADH) limits water reabsorption. The glomerular filtration rate decreases in moderate hypothermia, with increased vascular tone and decreased cardiac output.

In severe hypothermia, decreased tubular hydrogen secretion contributes to metabolic acidosis.

Hematologic and coagulation systems. Erythrocytes, leukocytes, platelets and clotting factors are all affected by hypothermia. Hematocrit increases due to decreased plasma volume. Cold produces bone marrow suppression. Decreased white blood cell count is common. Cold also decreases leukocyte function. Endothelial factors can cause changes in platelet function, causing platelet activation and thrombosis. Disseminated intravascular coagulation (DIC) may occur. On the other hand, thrombocytopenia is common and may contribute to bleeding in severe hypothermia. Cold inhibits enzymatic reactions of the clotting cascade, but the factors themselves are not depleted. Rewarming rather than factor replacement is the appropriate treatment for changes in prothrombin time (PT), partial thromboplastin time (PTT), and international normalized ratio (INR).

Afterdrop refers to the continuing decline in core temperature after a patient is removed from a cold environment. This occurs even if active rewarming is in progress. Afterdrop is primarily due to unavoidable loss of heat from the warmer core to the cooler peripheral tissues. Afterdrop can be as much as 0.5°C, even when minimized using optimal rewarming methods.

History

History may be straightforward, especially in primary hypothermia, or difficult to obtain, as in an unconscious patient found indoors.

Where was the patient found? What was he or she doing?

Patients found outdoors in cold conditions have a reason for being hypothermic, but there may also be predisposing causes for hypothermia, such as trauma or intoxication with alcohol or other drugs.

Is the patient intoxicated?

The use of alcohol and other sedative-hypnotics predisposes to hypothermia by interfering with adaptive behavioral responses to cold. Alcohol causes cutaneous vasodilation, which increases heat loss from the skin.

Does the patient have an immobilizing condition or injury?

The patient may have become hypothermic due to the inability to reach shelter.

Does the patient have a metabolic cause for hypothermia?

Metabolic causes include hypothyroidism, hypoadrenalism, hypopituitarism and hypoglycemia, which lead to decreased metabolic rates.

Does the patient have another cause of hypothalamic dysfunction?

These include head trauma, tumor, stroke, or Wernicke's syndrome.

Could the patient be septic or in diabetic ketoacidosis?

Hypothermia in sepsis carries a grave prognosis. Diabetic ketoacidosis (DKA) also interferes with thermoregulatory mechanisms.

Has the patient been resuscitated with room temperature fluid or chilled blood?

Iatrogenic hypothermia is of particular importance in trauma patients receiving volume replacement therapy. All trauma patients should receive IV fluids warmed to 40°C.

Has the patient received drugs that decrease shivering?

These include phenothiazines, meperidine, buspirone and paralytics. Chlorpromazine is commonly used in treatment of heat stroke to abolish shivering. In mildly hypothermic patients, shivering is a major mechanism to increase core temperature.

Past medical

Use of phenothiazines is a risk factor for hypothermia. Burns or exfoliating skin conditions predispose patients to increased heat loss.

Physical examination

The primary goals of the physical examination are to establish the degree of hypothermia, identify diseases or underlying conditions that may have caused or contributed to hypothermia, and identify associated injuries which require treatment.

Airway, breathing, circulation

As with all patients, the ABCs (airway, breathing, circulation) are of primary importance. These may be difficult to assess in a severely hypothermic patient, because respiratory rate and heart rate may be extremely slow. Respirations are often very shallow, and pulses are likely to be weak and difficult to palpate through cold skin. It is often hard to make cardiac monitor leads adhere to cold, moist skin, and pulse oximeters do not work on cool, vasoconstricted extremities.

Level of consciousness

Alert patients generally have only mild hypothermia. Patients with alterations of consciousness must have etiologies other than hypothermia considered as the cause of impaired consciousness.

Vital signs

Core temperature is the temperature of key internal organs, primarily the heart. Precisely measuring core temperature is key to the evaluation and treatment of hypothermia.

Standard clinical thermometers may record temperatures only as low as 34°C. In case of a low reading in a patient with altered mental status, an electronic thermometer with an esophageal probe should be used. The actual probe is the same, whether it is used in the esophagus or in the rectum. In mild hypothermia, a rectal temperature may be adequate. If a glass thermometer is used, it must be a "low reading" type. Rectal probe temperatures have been used traditionally, but changes in rectal temperature significantly lag changes in core temperature. Bladder temperature is even less reflective of core temperature than rectal temperature. Oral temperatures are notoriously inaccurate. They are affected by a number of factors, especially changes in ventilation, and are useful only to exclude the diagnosis of hypothermia.

Epitympanic temperature measurement is a reasonable alternative to esophageal temperature, especially in patients who are not intubated. These use a special sensor in proximity to the tympanic membrane. They are not the same as infrared "tympanic" thermometers that are widely used. Epitympanic thermometers are not widely available in North America as of 2011.

As the patient cools, the initial response is tachycardia, after which there is progressive bradycardia. The heart rate is about 50% of normal at a core temperature of 28°C. If the heart rate is faster than expected at a given temperature, other causes of tachycardia should be suspected, including hypovolemia, hypoglycemia, or drug ingestion. Blood pressure and respiratory rate also initially increase before declining, as hypothermia becomes more severe. Inappropriate respiratory rates should suggest metabolic acidosis or a CNS lesion.

Abdomen

Decreased intestinal motility may lead to abdominal distention or rigidity, and may mimic or mask an acute abdomen.

Neurologic

Dysarthria and ataxia may be found in mild hypothermia. These are likely to increase in severity at lower core temperatures. Reflexes are initially increased, and then decrease before eventually disappearing as core temperature decreases. Muscle tone increases in the preshivering phase. Shivering patients may not be hypothermic. Shivering is maximal at about 35°C, and gradually decreases until the core temperature is about 31°C, at which point shivering disappears entirely. Severely hypothermic patients may be completely unresponsive and lack all reflexes, including corneal reflexes.

Differential diagnosis

Hypothermia is diagnosed by measuring core temperature. However, conditions other than environmental exposure can cause hypothermia. Although not truly differential diagnoses, associated diagnoses should be considered in hypothermic patients. Table 47.3 is a partial list of etiologies to consider.

Diagnostic testing

Laboratory studies

Complete blood count

Hematocrit increases about 2% with every 1°C decrease in temperature. A moderately or severely hypothermic patient with a "normal" hematocrit is actually anemic. White blood cell and platelet counts are depressed by sequestration.

Metabolic profile

Potassium levels are independent of temperature. However, hypothermia increases the toxic effects of hyperkalemia. Blood glucose is increased in hypothermia, because endogenous insulin is inactive at temperatures <30–32°C.

Coagulation studies

Hypothermia induces a coagulopathy, although coagulation studies are insensitive in the hypothermic patient. PT, PTT, and INR are performed in the lab at 37°C. They may not reflect the coagulopathy and cannot be used to guide initial therapy.

Creatine phosphokinase

Rhabdomyolysis is a potential complication of immobility, which may be associated with hypothermia.

Arterial blood gas

Although blood gas values that are "corrected" for temperature are available, they should not be used. Uncorrected blood gas values should be used to guide treatment at all temperatures. Using "corrected" blood gas values will cause pH abnormalities.

Electrocardiogram

Numerous ECG changes can be found in the hypothermic patient. Prolongation of PR, QRS and QT intervals is usual; T-wave inversion may be seen. Muscle tremor

Table 47.3 Differential diagnosis of hypothermia

Diagnosis	Symptoms	Signs	Work-up
Acute spinal cord transection	Lack of peripheral sensation	Paralysis, vasodilation	Neurologic examination, X-ray, CT, MRI
Alcohol, drugs and other toxic exposures, including benzodiazepines, barbiturates, phenothiazines and carbon monoxide	Depends on agent(s)	Specific toxidromes may be identified	Drug levels, CMP, serum osmolality, COHb level, serum volatiles, urine toxicology screen; consider naloxone and high-flow (or hyperbaric) oxygen
CNS lesions (trauma, CVA, mass), hypothalamic dysfunction (including Wernicke's syndrome)	Variable	Signs of trauma, abnormal neurologic examination	Head CT, thiamine administration
Endocrine dysfunction (hypoglycemia, thyroid, adrenal or pituitary insufficiency)	Often nonspecific	Typical signs of endocrine abnormalities may be present	Fingerstick glucose, laboratory studies, steroid administration
Iatrogenic: fluid resuscitation; exposure in ED, OR and radiology suite; drug administration; heat stroke cooling; emergency delivery	Nonspecific	Nonspecific	Diagnosed by history
Infection, including meningitis, encephalitis, pneumonia, sepsis	Variable	Tachycardia, hypotension	Search for source of infection
Myocardial infarction or other cause of decreased cardiac output, including dysrhythmia, cardiomyopathy	Chest pain, dyspnea, dizziness, syncope, weakness, confusion, or other nonspecific symptoms	May include low cardiac output	ECG, cardiac markers, echocardiography
Pancreatitis, peritonitis	Abdominal pain	Abdominal rigidity, peritoneal signs	Laboratories, abdominal CT
Skin lesions or diseases	Nonspecific	Burns, exfoliative dermatitis	Consider dermatology consult/biopsy

CMP: comprehensive metabolic panel; CNS: central nervous system; COHb: carboxyhemoglobin; CT: computed tomography; CVA: cerebrovascular accident; ECG: electrocardiogram; ED: emergency department; MRI: magnetic resonance imaging; OR: operating room.

artifacts may make obtaining an adequate ECG difficult. The Osborne (J) wave, a slow deflection at the junction of the QRS complex and the ST segment, is a common finding (Figure 47.1). These waves are usually upright in left-sided precordial leads. Dysrhythmias may include sinus tachycardia (in mild hypothermia), sinus bradycardia, atrial fibrillation or flutter, atrioventricular (AV) block, nodal rhythms, premature ventricular contractions (PVCs), ventricular fibrillation (VF) or asystole. Atrial fibrillation is the most common dysrhythmia other than rate disturbances of sinus origin.

Radiologic studies

If trauma is suspected, cervical spine, chest and pelvis films may be appropriate. Other X-rays and CT scans are indicated based on clinical presentation.

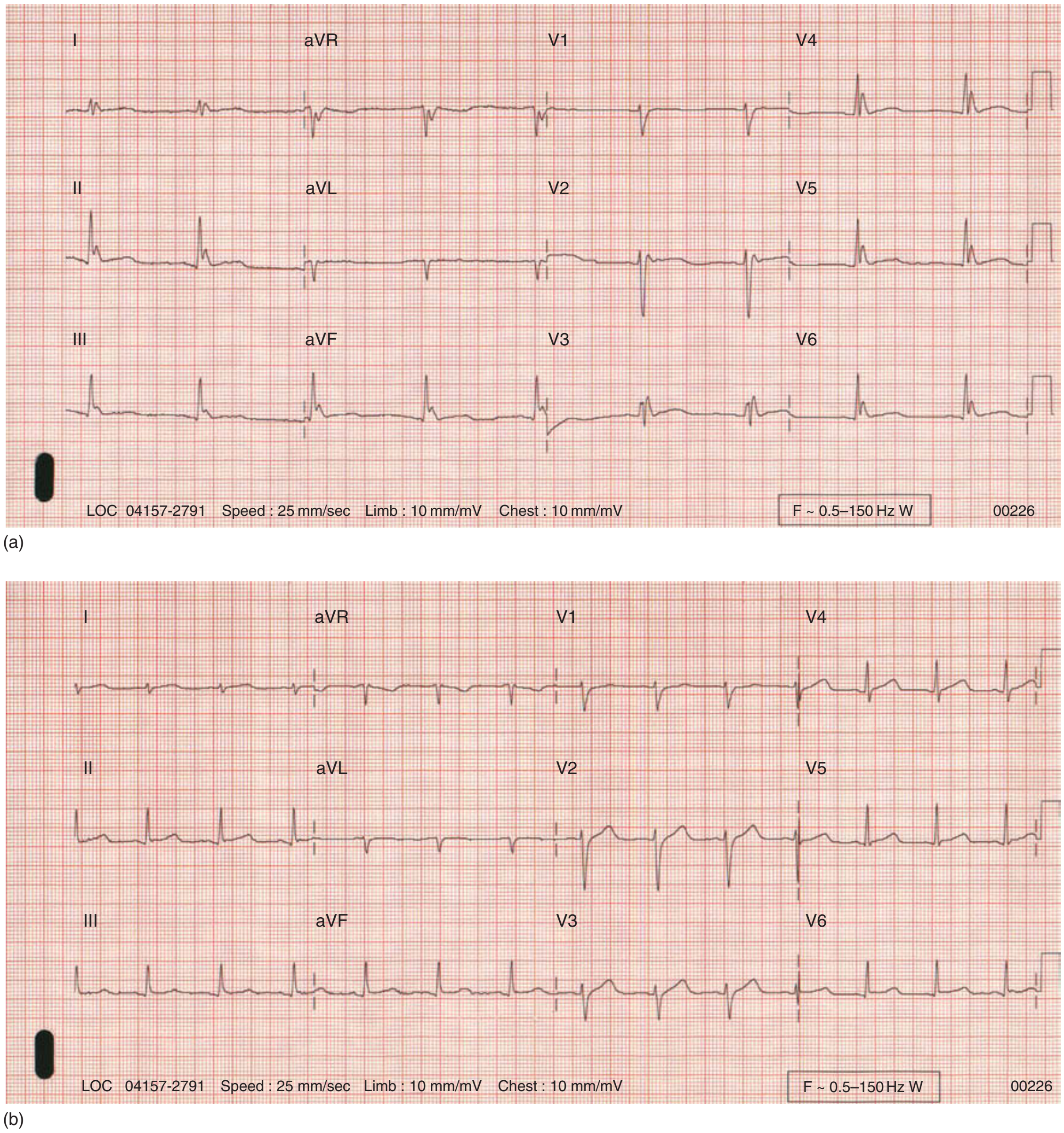
General treatment principles

As with all patients, treatment begins with the ABCs. Subsequently, an appropriate method of rewarming should be initiated, and volume status and electrolyte fluxes must be managed. Handle all moderately or severely hypothermic patients gently to avoid precipitating ventricular fibrillation. Remove wet clothing by cutting it off. The resuscitation room should be heated to 28°C (82°F). Insulate the patient to protect against further heat loss, and begin forced air rewarming as soon as possible.

Initial resuscitation

Endotracheal (ET) intubation is mandatory unless the patient is alert and protecting the airway. The patient should have continuous cardiac and vital sign monitoring. Pulse oximetry is seldom possible (or reliable) in the hypothermic patient due to peripheral vasoconstriction. Both a nasogastric (NG) tube and indwelling urinary catheter should be gently inserted. Monitor the temperature continuously using an esophageal probe.

If the heart is in ventricular fibrillation (VF) or asystole, CPR should be initiated. If VF is present, a single attempt should be made to defibrillate the patient. However, defibrillation is seldom successful if the patient's core temperature is <28–30°C. Start CPR if the patient does not have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LOC 04157-2791 Speed : 25 mm/sec Limb : 10 mm/mV Chest : 10 mm/mV
F ~ 0.5–150 Hz W
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(b)

Figure 47.1
Resolution of Osborne J waves in a patient with (a) hypothermia and (b) rewarming. Courtesy: Joel T. Levis, MD, PhD.

a perfusing rhythm. Atrial dysrhythmias resolve with rewarming and do not require treatment. Further attempts at defibrillation are warranted once the core temperature has increased to 30°C according to ACLS guidelines.

When establishing IV access, central lines inserted into the heart or pulmonary artery are contraindicated. Peripheral lines may impossible to establish, so the use of a jugular, subclavian or femoral catheter, or intraosseous access may be necessary. Hypothermic patients are generally volume depleted. Replace volume with D5NS initially. Lactated Ringer's solution should not be used because the hypothermic liver cannot metabolize lactate. All IV fluids should be heated to 40–42°C to prevent further heat loss. Although fluids are administered to replace estimated deficits rather than for rewarming, they may have a significant rewarming effect due to the decrease in circulating blood volume.

Medications

IV or IO are the only effective routes of administration for medications, but most drugs are not active at temperatures <30°C. Protein binding of drugs increases in hypothermia, further limiting their effects. Medications given in the hypothermic patient are generally ineffective until rewarming takes place, after which they suddenly become effective and possibly toxic. Thiamine and glucose may be given empirically.

Rewarming

Following initial stabilization, the emergency physician must choose a rewarming method. *Passive rewarming* refers to methods that use heat generated by the patient. *Active rewarming* methods add heat to the patient from other sources. Mildly hypothermic patients (core temperature 32–35°C) can be treated with passive external rewarming, utilizing their own body heat and shivering to rewarm. In practice, most of these patients will receive active external rewarming with warm blankets or forced heated air. Hypothermic patients with body temperatures <32°C, with cardiovascular instability, or with underlying conditions predisposing to hypothermia require active core rewarming; this refers to methods that preferentially heat the central organs.

Although there are many methods of active core rewarming, the most common are peritoneal lavage and various blood rewarming techniques. Blood rewarming techniques include arteriovenous or venovenous rewarming, hemodialysis, and cardiopulmonary bypass. Hypothermic patients in cardiac arrest require cardiopulmonary bypass for rewarming. The choice of specific rewarming techniques is complex and depends on institutional resources. Obtaining assistance from the nephrology, cardiothoracic surgery or trauma surgery services may prove life-saving.

Limitations to rewarming

It has been said that "no one is dead unless they are warm and dead." However, there are some people who are cold and dead. Patients who have obvious lethal injuries and those who have do not resuscitate orders should not undergo rewarming attempts. Dependent lividity, apparent rigor mortis, or fixed dilated pupils are not contraindications to CPR.

Rewarming an accidental hypothermia victim with a core temperature <10°C is likely to be futile. The coldest core temperature from which a victim of accidental hypothermia has been rewarmed is 13.7°C following the afterdrop. This patient made a complete neurologic recovery with the exception of a peripheral neuropathy that lasted many years.

Serum potassium levels >12 mEq/L may correlate with an inability to resuscitate a hypothermic patient. This degree of hyperkalemia is a marker of cell lysis. It can be used in the decision to terminate resuscitation efforts only in hypothermic patients with asphyxia, including avalanche victims. It cannot be used to terminate resuscitation in hypothermic patients without asphyxia. A child with a core temperature of 14.2°C and a potassium of 11.8 mEq/L has been successfully resuscitated.

Further management

During rewarming, electrolyte and volume status alterations require active management. Coagulopathies may pose special problems. Also during this time, underlying diseases and traumatic injuries need to be addressed. Close cardiopulmonary monitoring and continued reassessment of neurologic status are crucial.

Special patients

Elderly

Geriatric patients are more prone to hypothermia, tend to have more underlying diseases, and usually have less physiologic reserve than younger adults. They often require more aggressive treatment for hypothermia and its complications. Geriatric patients with hypothermia should generally be treated as if they are septic.

Pediatric

Neonates require aggressive volume resuscitation in addition to the usual treatment of hypothermia. Unless cold exposure is the clear cause of hypothermia, pediatric patients should be evaluated and treated for sepsis.

Immune compromised

Sepsis may be an important cause of hypothermia in immunocompromised patients. If there is any doubt, these patients should be presumptively treated for sepsis and aggressively rewarmed.

Disposition

Otherwise healthy patients with mild hypothermia (32–35°C) due to cold exposure usually have no difficulty being rewarmed. Most of them can be discharged safely unless they have associated injuries, including frostbite, that necessitate hospital admission. All other patients with hypothermia require admission. In some cases, patients may require transfer to a center with the capability to perform cardiopulmonary bypass.

Pearls, pitfalls and myths

Pearls

- Hypothermia can mask the symptoms and signs of other diseases. It is crucial to measure core temperature and consider associated or alternative diagnoses.
- Hypothermic patients can survive without cardiac activity. Contraindications to CPR include any sign of life, Do Not Resuscitate (DNR) status, or obvious lethal injuries. Dependent lividity, apparent rigor mortis, or fixed dilated pupils are not contraindications to CPR.
- A hypothermic patient with a normal hematocrit is likely anemic.

- Atrial dysrhythmias resolve with warming and do not require treatment.
- Insulin is ineffective at temperatures <30°C, as are most other pharmacologic agents.
- IV fluids can be warmed in a microwave oven. The bag should be shaken before administration to prevent hot spots.
- Tympanic, oral and bladder temperatures are unreliable in hypothermic patients.

Pitfalls

- Failure to diagnose hypothermia by failing to measure core temperature.
- Failure to handle the hypothermic patient gently, which may precipitate VF.
- Failure to prevent further heat loss by covering the patient, including the head and neck.
- Failure to diagnose traumatic injuries responsible for hypotension, tachycardia (relative to core temperature), or neurologic dysfunction.
- Being unaware that cardiac and other drugs are not absorbed well orally or intramuscularly, and are likely to remain inactive until rewarming occurs.
- Administration of room temperature or cold IV fluids or blood products.
- Administration of meperidine or phenothiazines, which abolish shivering.

Myths

- The axiom "No one is dead until they are warm and dead" is a myth. The truth is that some people are cold and dead.
- ABGs should be corrected for core temperature. In fact, uncorrected ABGs should be used to guide therapy.
- Hypothermia only occurs in cold climates. Hypothermia can occur under mild conditions both outdoors and indoors.

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47D LIGHTNING INJURIES

Ken Zafren, MD

Scope of the problem

Lightning kills over 1,000 people worldwide every year, although the number of fatalities in developed countries has been decreasing for the last 80 years. The death rate in the United States decreased from over 6 per million to 0.4 per million annually during the twentieth century. At least 70% of lightning strikes are not fatal, but many survivors experience significant sequelae.

Anatomic essentials

Lightning is a direct current that produces extremely high voltage for a very brief duration. Unlike alternating current, the direct current of lightning often flows over the exterior of the body. This is referred to as *flashover*. However, lightning may occasionally enter the body with devastating results.

Lightning produces injury by several different mechanisms:

- Lightning may strike a person directly, which is often fatal.
- Most frequently, current splashes from nearby objects or people standing nearby. This is known as *side flash*.
- Contact injury is produced when a person is in direct contact with an object that is hit or splashed by lightning. Contact with plumbing or with wiring entering a building may cause injury to a person inside a building who is using an appliance (including a telephone) or water.
- Step voltage or ground current causes injury by flowing between two parts of the body in contact with the ground at different distances from the lightning strike, due to the voltage difference between these two points of contact.
- Lightning can enter the body through the mouth, ears, or orbits.
- Blunt injury can occur from the shock wave produced by lightning and the muscle contractions due to the current. Victims can be thrown a significant distance, or can lose balance and fall. Pressure injuries, including tympanic membrane rupture, frequently occur. Blunt injury occurs when falling or thrown objects hit the victim.

Direct lightning injuries are due to high voltage; the secondary effects are due to heat production and explosive force. Death at the time of injury is most commonly due to cardiac arrest. Respiratory arrest is often prolonged due to paralysis of the respiratory center in the medulla. This may lead to hypoxic cardiac arrest if not treated with ventilatory support. High-voltage brain injury or blunt head trauma may also cause death. Other direct injuries include contusions, tympanic membrane rupture, hematologic abnormalities such as disseminated intravascular coagulation (DIC), burns, and a variety of neurologic conditions.

In a lightning strike, the heart may stop instantly during myocardial depolarization, resulting in asystole or ventricular fibrillation. Cardiac activity usually resumes promptly, although it may be in jeopardy because of prolonged respiratory arrest. Various ECG changes can be seen in lightning strikes. Myocardial infarction occasionally occurs. Hypertension is common, although it generally resolves within a few hours without treatment. Hypotension may result from traumatic hemorrhage.

Neurologic injuries are often transient. Immediate injuries are typically transient, although they may be fixed and severe. Temporary neurologic symptoms include seizures, paralysis, deafness, blindness, confusion, amnesia and coma. Temporary paralysis of the extremities is called *keraunoparalysis*. It is due to intense vasospasm and usually clears within hours. Delayed injuries, which are likely progressive, include seizures, neuromuscular disorders, ataxia, extremity weakness, paralysis, or chronic pain.

All organ systems may be affected. Pulmonary, gastrointestinal and renal injuries may be immediate or delayed, and may be due to hypotension or other injuries. About half of lightning victims will have eye injuries, most commonly cataracts. These may be immediate or appear as long as 2 years later. Pupillary findings, including fixed, dilated pupils, are common and often transient. Hearing loss, vertigo and damage to the auditory system are also seen. Over half of victims have tympanic membrane rupture, which usually heals without intervention. Psychiatric sequelae are common.

Burns are common. Burns are caused by the direct effects of lightning or by secondary heat production. Most direct burns are superficial, resulting from the rapid flashover effect. Burns also occur from vaporization of sweat or moisture in clothing, from melted synthetic clothing, and from heated metal objects. The terms *feathering* and *Lichtenberg figures* are synonymous; these refer not to burns but to skin markings that are pathognomonic of lightning injury (Figure 47.2). Lichtenberg figures may be palpable; they are probably caused by superficial bruising. Entry and exit burns rarely occur.

History

The history in lightning injury is variable. On one hand, the history may be straightforward when the strike is witnessed. On the other hand, lightning can strike "out of the blue," at great distance from a lightning storm, with the strike not witnessed. Victims frequently have no memory of the event. History obtained from witnesses may prove helpful.

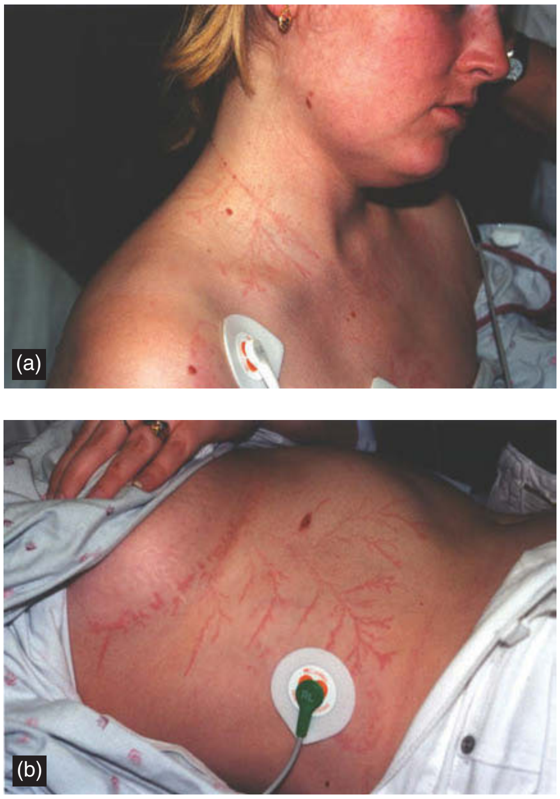

Figure 47.2
Feathering following lightning strike. (a) *Lichtenberg figures* originating from 14K gold necklace being worn by the victim at the time of the lightning strike. The necklace was melted into three sections. (b) *Lichtenberg figures* radiating caudally following area of skin in contact with cloth-covered wire from an underwire bra. Courtesy: Sheryl Olson, RN, BSN.

Was the patient found outdoors or in a building not protected from lightning? Was a known lightning storm in the vicinity? Was it a hot, humid day when lightning was likely?

In these circumstances, an unconscious or confused patient may have been a lightning strike victim.

If the event was witnessed, what was the mechanism of injury?

Was the patient struck directly, splashed, or affected by ground current? Did the patient fall, get struck by a falling object, or get thrown?

Did the patient require resuscitation at the scene?

If the patient required resuscitation from cardiorespiratory arrest, the prognosis is much worse.

Did the patient lose consciousness or have any neurologic deficits that have resolved?

The nature of the injuries and the time course of symptom resolution may provide clues to the severity of delayed injuries.

Past medical

This information is as important as it would be for any patient with multiple injuries.

Physical examination

The main goal of the physical examination is to identify direct and indirect injuries caused by a lightning strike. In cases in which the mechanism of injury is uncertain, the physical examination may help identify lightning as the cause of the patient's presentation.

General appearance

Victims with minor injuries are usually alert and seldom have burns. Confusion, combativeness, or coma indicates a greater degree of injury. Paralysis, especially of the legs, may also be seen. The patient may be seizing. Severely injured victims may be in cardiac arrest. Signs of blunt trauma are common and should be identified.

Vital signs

Mildly injured victims will have normal vital signs or mild hypertension that resolves without treatment. Hypotension in a lightning victim should prompt search for traumatic hemorrhage.

Head and neck

Burns on the head indicate severe injury. Blindness and deafness are common and are often temporary. Various eye and ear injuries, especially tympanic membrane rupture, are frequent and mandate more detailed examination. Hemotympanum and CSF otorrhea are signs of basilar skull fracture. If there is any question of cervical spine injury, the neck should be stabilized during the physical examination.

Skin

The presence of feathering (Lichtenberg figures) is pathognomonic for lightning injury. Linear and punctate burns also provide evidence of lightning injury. Burns are usually superficial. Partial-thickness burns may become more evident with time. Full-thickness burns are uncommon, but may also occur. These may be punctate burns or may be caused by heating of metal objects or burning clothing.

Abdomen

Ileus may be present and may cause abdominal tenderness.

Table 47.4 Differential diagnosis of lightning injuries

Diagnosis	Symptoms	Signs	Work-up
Cardiac dysrhythmias	Variable	Variable	ECG, cardiac monitoring, cardiac markers
Central nervous system or spinal cord trauma; cerebrovascular accident	Visual or hearing loss, amnesia, headache, paresthesias, extremity weakness	Altered level of consciousness, including coma; confusion, localized weakness, paralysis	Appropriate imaging and specialty consultation
Multiple trauma, assault	Depends on injuries	Depends on injuries; scattering of clothing at scene may be due to lightning	Depends on injuries
Myocardial infarction	Chest pain, dyspnea, nausea/vomiting, diaphoresis	May be absent	ECG, cardiac markers, angiography
Seizure disorder	Amnesia for the event	Seizure or postictal state (altered level of consciousness or confusion); tongue injury, incontinence of urine or stool	Serial examination, head CT; may require EEG
Syncope due to cardiac dysrhythmias or valvular disease	Syncope	Syncope without warning	ECG, echocardiography
Toxic ingestion, envenomation	Depends on the toxin or venom	Depends on the toxin or venom	Careful history and physical examination; laboratory testing as indicated

CT: computed tomography; ECG: electrocardiogram; EEG: electroencephalogram.

Neurologic

Mental status should be noted. It may be normal or the patient may be confused, combative, or comatose. Amnesia is common. Pupillary findings are an unreliable indicator of cranial nerve function. Autonomic dysfunction may cause non-reactive pupils, mydriasis, anisocoria, or Horner's syndrome. Lower extremity keraunoparalysis is found in two-thirds of severely injured patients; one-third have upper extremity keraunoparalysis. Paralysis of the extremities is the result of sympathetic stimulation with severe vasospasm. The affected limbs appear mottled and are cold to the touch. They may be numb and pulseless. Spinal paralysis, paresis and cerebellar dysfunction may occur. Hemiplegia and aphasia have been reported, and simulate a cerebrovascular accident (CVA).

Differential diagnosis

Given a history of a lighting storm and witnesses to the strike, the diagnosis of lightning injury is straightforward. Victims with feathering, or punctate or linear burns should be treated for lightning injury. Victims found without appropriate historical or physical findings may represent other diagnoses. (Table 47.4); however, these injuries may be the result of lightning even if a strike is not witnessed.

Diagnostic testing

Laboratory studies

Testing will depend on the severity of injury and on associated clinical factors.

Complete blood count

Obtain a CBC for all patients with moderate to severe injury to assess for hemorrhage.

Basic metabolic panel

Obtain a basic metabolic panel (BMP) to assess for renal injury or electrolyte abnormalities.

Creatine phosphokinase

Obtain a CPK, UA and urine myoglobin to screen for rhabdomyolysis.

Cardiac markers

Cardiac markers are obtained in moderate to severe injury, when the ECG is abnormal, or when the patient complains of chest pain.

Coagulation

Coagulation studies should be ordered in moderate to severe injury to assess for DIC.

Electrocardiogram

An ECG should be obtained to assess for possible cardiac injury or abnormalities. Prolongation of the QT interval is the most frequent rhythm abnormality. Atrial fibrillation, premature ventricular contractions and ventricular tachycardia have been reported. Elevation of the ST segment and T-wave changes may also be seen. Ischemic changes do not always reflect vascular distribution patterns in lightning strike victims; areas of focal necrosis may be found. ECG changes may be delayed 24 hours or more.

Radiologic studies

A CXR should be obtained to identify pulmonary injury or pneumothorax. Other plain films should be obtained based on suspected injuries.

Head CT is indicated in patients with altered consciousness or suspected head injury. CT of the cervical spine should also be obtained in unconscious or multiply injured patients. CT angiography of the chest, CT of the abdomen and pelvis, and CT of the thoracic and lumbar spine have the same indications as in other patients, including those with suspected trauma.

General treatment principles

As with all patients, treatment begins with the ABCs (airway, breathing, circulation). Lightning victims should be treated according to cardiac and trauma care guidelines. Hypothermia, if present, should be addressed.

Cardiovascular care

Asystole and VF are seen more often in the field than in the ED. Atrial fibrillation, premature ventricular contractions and ventricular tachycardia may occur. There is no evidence to suggest that dysrhythmias due to lightning injuries should be treated differently than those due to primary cardiac causes. Dysrhythmias may not occur until 24 hours after the injury. Premature ventricular contractions have been reported up to 1 week later. Most dysrhythmias resolve within days, but some may persist for months. Transient hypertension is the rule and does not usually require treatment. Delayed hypertension has been reported up to 72 hours after the injury. Persistent cases have been treated with beta-blockers or other antihypertensive agents.

Volume replacement

All patients require IV access, but volume resuscitation is seldom necessary. Hypotensive patients and those with extensive burns should receive appropriate volume resuscitation. Most lightning burns are superficial and do not require special treatment. The presence of hypotension should prompt a search for a source of bleeding.

Neurologic treatment

Neurologic injuries are treated as in any trauma patient, although some exceptions exist. Fixed, dilated pupils may be the result of local eye injury and do not necessarily reflect CNS injury. Paralyzed extremities that are cool, mottled and pulseless should be observed, as these are signs of keraunoparalysis. The limb generally returns to normal in a few hours once the vasospasm resolves. Fasciotomies are almost never needed in these cases.

Miscellaneous injuries

Eye injuries may include immediate or delayed cataracts. Ear injuries, such as tympanic membrane rupture, usually heal spontaneously and do not require specific treatment. Any eye or ear injury resulting from lightning strike merits ophthalmology or ear, nose and throat (ENT) consultation. Ileus can occur and may require treatment with NG tube decompression.

Special patients

Pregnant patients

Pregnant patients are not at increased risk for mortality from lightning exposures, but fetal death occurs in about half of cases due to the high conductivity of amniotic fluid. Fetal ultrasound and tocodynamometric fetal monitoring are mandatory after 20 weeks gestation. If the fetus is viable, the remainder of the pregnancy is considered high risk.

Disposition

Most experts believe that asymptomatic patients with a normal ECG, including those with feathering, may be safely discharged after several hours of observation. These patients need close follow-up with neurology, ENT, and ophthalmology, as delayed sequelae are common.

Mildly injured patients who improve initially should be admitted for neurologic and cardiac monitoring, with consultation from specialty services as indicated.

Most patients with significant lightning injuries should be admitted to a referral hospital with a full spectrum of consultative services. The trauma, neurosurgery, cardiology, neurology, ENT and ophthalmology services are often consulted. Pregnant women beyond 20 weeks gestation will require admission for fetal ultrasound and a minimum of 4 hours of tocodynamometric fetal monitoring. The treatment for fetal demise is uterine evacuation.

Pearls, pitfalls and myths

Pearls

- Apneic patients often survive without neurologic sequelae if ventilated adequately until spontaneous respirations resume. Cardiac activity usually resumes quickly if the patient is ventilated early and does not develop hypoxia.
- The amount of visible (external) damage does not always correlate well with the severity of internal injuries.
- Lightning current usually flows over the body, but can also enter through various orifices, including the eyes, ears and mouth.

- Feathering (Lichtenberg figures) is pathognomonic of lightning injury. Punctate or linear burns suggest lightning injury.

Pitfalls

- Not resuscitating a lightning victim who has fixed, dilated pupils.
- Not treating keraunoparalysis expectantly.
- Not considering the diagnosis of lightning injury in a patient found comatose or confused, given the appropriate conditions.
- Not looking for a source of bleeding in a patient with hypotension following lightning injury.
- Not considering delayed and long-term sequelae, or providing appropriate specialty follow-up care.
- Treating a lightning victim like a patient with a high-voltage electrical injury. Large volume resuscitation, fasciotomies and other aggressive treatments common in high-voltage injuries are almost never necessary in lightning injuries.

Myths

- Most lightning strikes are fatal.
- Lightning cannot strike inside a building.
- It is dangerous to touch a lightning victim.
- "If you are not killed by lightning, you will be okay."
- Lightning injury causes few permanent sequelae.
- Lightning victims may recover after prolonged resuscitation because they are in suspended animation.

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47E TERRESTRIAL VENOMOUS BITES AND STINGS

Robert L. Norris, MD

Scope of the problem

In 2007, there were more than 70,000 calls to US poison control centers regarding bites and stings. Although the majority of these injuries can be handled with simple first aid alone, a number of victims will present to the emergency department (ED) for care. Presentations can be for acute systemic toxicity, local wounds, or anaphylactic reactions in a sensitized individual.

The venomous creatures exacting the greatest toll in terms of human injury include arthropods, especially hymenoptera or stinging insects (bees, wasps, hornets, yellow jackets and fire ants), a handful of spider species, scorpions, a few miscellaneous species, and venomous reptiles, particularly snakes.

The major impact of hymenoptera is seen in the 0.3–3.0% of the human population that is dangerously allergic to these insects and at risk of anaphylaxis if stung. The vast majority of deaths related to venomous creatures in the United States are due to hymenoptera-induced anaphylaxis.

The spiders of major consequence can largely be divided into two groups: those that may cause dermonecrosis (e.g., the recluse spiders [*Loxosceles* species, the best known of which is the brown recluse, *L. reclusa*]) (Figure 47.3) and those that cause a neurotoxic picture (e.g., the widow spiders [*Latrodectus* species] (Figure 47.4), which are found throughout much of the world, the South American huntsman spiders *[Phoneutria* species], and the Australian funnel-web spiders [*Atrax* and *Hadronyche* species]).

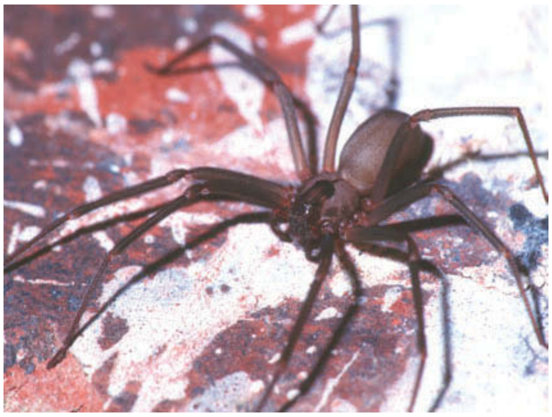

Figure 47.3
Brown recluse spider (*Loxosceles* species). Note the characteristic dorsal, violin-shaped marking. Courtesy: Michael Cardwell.

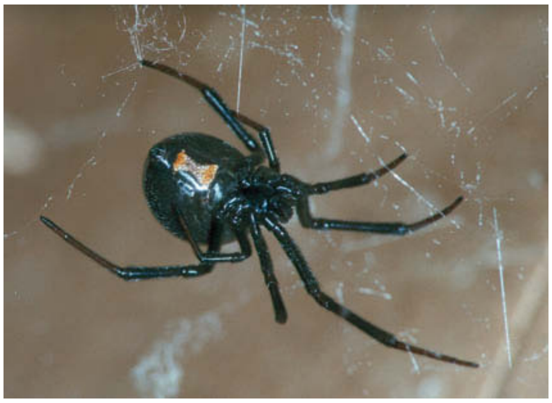

Figure 47.4
Black widow spider (*Latrodectus* species). Note the characteristic central hourglass. Courtesy: Michael Cardwell.

Scorpions are of limited medical importance in the United States, with only one potentially dangerous species (*Centruroides sculpturatus* [formerly *Centruroides exilicauda*]). They are much more important in other regions of the world, particularly Central and South America, Africa, Asia and the Middle East. In Mexico, for example, scorpions take a higher toll in terms of human mortality than do venomous snakes. The most dangerous venomous scorpions fall into the genera *Centruroides, Tityus, Buthus, Buthacus, Androctonus, Leiurus, Mesobuthus* and *Parabuthus*.

Although a number of caterpillars are capable of stinging humans, most are little more than painful nuisances except for the South American *Lonomia* caterpillars, which are capable of inducing life-threatening coagulopathy. Centipedes, found throughout much of the temperate and tropical world, can inflict a painful sting, but require only local wound care and analgesics, and are not discussed further in this chapter.

Venomous snakes fall into the families Viperidae (subfamilies Viperinae [vipers] and Crotalinae [pit vipers]) (Figure 47.5), Elapidae (including cobras, mambas, coral snakes, and all of the venomous snakes of Australia), and Hydrophiidae (sea snakes). There are a few species of snakes in the family Colubridae that have salivary secretions that can be severely toxic to man (e.g., the boomslang [*Dispholidus typus*] and the twig snake [*Thelotornis kirtlandi*], both of Africa). Most Colubridae are largely harmless. There are two clearly venomous species of lizards, the Gila monster (*Heloderma suspectum*) and the Mexican beaded lizard (*Heloderma horridum*), but bites by these species are quite rare unless an attempt is made to pick up the animal. Venomous lizards are not discussed in this chapter.

Anatomic essentials

The venomous creatures discussed in this chapter all produce venoms in specialized glands and deliver these venoms using sophisticated anatomic structures (e.g., fangs or stingers) to their victims or prey. These venoms are complex and lead to various local and/or systemic derangements depending on the species involved. A brief overview of the major venom types and characteristic clinical sequelae is found in Tables 47.5 and 47.6. One should keep in mind that venoms can vary not only between species, but also between individuals within a species, and that venoms affect different individuals in different ways. Therefore, a physician tasked with managing a victim of venomous bite or sting must be vigilant for atypical presentations and sequelae.

Table 47.5 Overview of venoms of major groups of terrestrial creatures

Arthropod Venoms

Hymenoptera

Bees, wasps, yellow jackets, hornets, fire ants

- Peptides such as melittin (which may be allergenic) and apamin
- Vasoactive amines (histamine, serotonin, acetylcholine, epinephrine, norepinephrine and dopamine)
- Enzymatic components (phospholipase A, hyaluronidase, and acid phosphatase) – major allergens
- Fire ant venom contains piperidine alkaloids and low-molecular-weight polypeptides

Spiders

- Venoms can be quite variable. Although almost all groups of spiders are venomous, the vast majority of species have fangs too small to penetrate human skin and are, therefore, of no medical significance.

Recluse spiders

- Sphingomyelinase D – may produce dermonecrosis and systemic hemolysis

Widow spiders

- Alpha latrotoxin – a neurotoxin that stimulates the release of neurotransmitters from nerve terminals (epinephrine, norepinephrine and acetylcholine)

Australian funnel-web spider

- Atratoxin or robustoxin – neurotoxins found in different species

Wandering spiders

- Neurotoxic venoms

Scorpions

- These venoms are quite variable in different species
- Most toxic scorpions contain neurotoxic and/or cardiotoxic components
- Many species contain serotonin (increases pain)

Toxic caterpillars

- Most venoms poorly studied

Lonomia sp.

- Venoms procoagulant and anticoagulant, inducing consumptive coagulopathy

Snake Venoms

- Very complex

Viperids (vipers and pit vipers)

- Components can affect essentially any body system
- Among the most deleterious fractions are low-molecular-weight components that cause cellular membrane and intercellular disruption (responsible for tissue destruction)
- Most viperid venoms contain components that can cause systemic coagulopathy (due to affects on the coagulation cascade at various sites)

Elapids

- Many of these venoms possess significant neurotoxicity, which can cause death due to respiratory depression
- Some species have components that cause severe tissue necrosis (e.g., spitting cobras)

Hydrophids (sea snakes)

- Neurotoxicity
- Myotoxicity

Colubrids

- Most species in this family are harmless
- A few species can envenom humans with toxic secretions from a special salivary gland (Duvernoy's gland); these secretions are hemorrhagic.

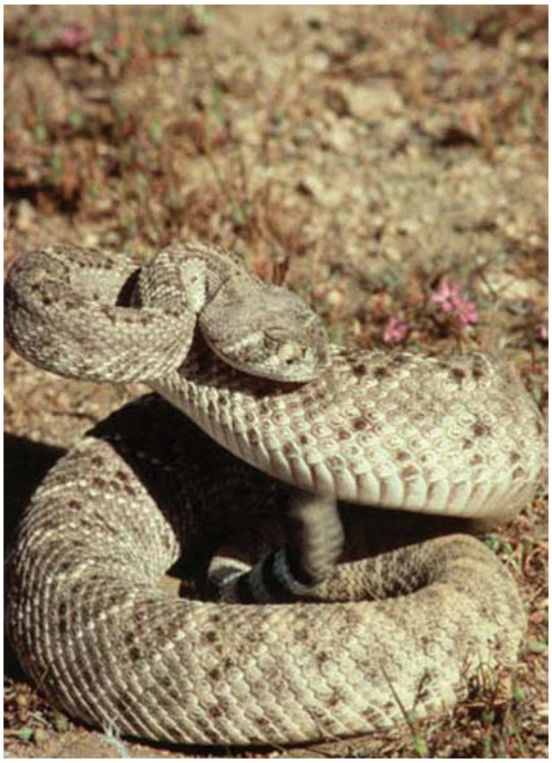

Figure 47.5
Rattlesnake (*C. atrox*). Courtesy: Michael Cardwell.

History

What was the offending animal?

It is easiest to manage a victim of a venomous bite or sting if the animal that inflicted the injury can be identified. Often this is easy, particularly if the creature was seen and can be accurately described (or if it was killed and brought with the patient). More than 50% of venomous snake bites in the United States occur to people who are intentionally handling the animal, making identification more straightforward in this situation. In some cases, identification of the offending animal can be difficult or impossible. A good example of this is the bite of the brown recluse spider, which is often initially painless so

Table 47.6 Descriptions of clinical syndromes that can be anticipated after bites/stings of venomous terrestrial creatures

Hymenoptera stings

- Typical local reaction: initial pain, followed by itching wheal or hive limited to the sting site.
- Exaggerated local response: pain and itching that progress over many hours to days to involve the entire extremity.
- Anaphylaxis: a spectrum of diffuse hives, wheezing, laryngeal edema, hypotension, abdominal pain, vomiting, diarrhea, and possibly death.

Spider bites

- Species capable of inflicting a bite through human skin: pain, a local papule that may develop a small eschar that heals over a period of days.
- *Recluse spiders:* bite is usually painless; delayed onset of pain due to ischemia at the bite site; ischemic tissue develops an ulcer that may be progressive and, in rare cases, become severe, undermining normal skin and creating a necrotic “volcano lesion”; rarely, systemic envenomation can present with flu-like symptoms (fever, nausea, vomiting, etc.) with acute hemolysis and potential development of renal failure.
- *Widow spiders:* bite felt as a pinprick, gradual onset of muscle pain and cramping in the involved extremity (which may spread to the trunk, resulting in respiratory distress or a rigid abdomen) that progress for several hours and may persist for 72 hours; associated nausea, vomiting, sweating, headache (possibly due to elevated blood pressure or intracranial bleeding), rapid heart rate or palpitations.
- *Funnel-web spiders:* severe pain, perioral tingling, muscle fasciculations, diaphoresis, lacrimation, hypertension, tachycardia, pulmonary edema and respiratory distress.
- *Wandering spiders*: immediate, severe pain, mild swelling; most bites are mild, but may be severe, particularly in children and the elderly, with autonomic nervous system overdrive (hypertension, hyperglycemia, diaphoresis, priapism, pulmonary edema).

Scorpion stings

- Non-neurotoxic scorpions (e.g., most US species): initial intense pain that passes quickly; possibly mild soft tissue swelling and local bruising; anaphylaxis is rare.
- Neurotoxic scorpions (e.g., *Centruroides sculpturatus*): pain; minimal local tissue changes; restlessness, roving eye movements, salivation, respiratory distress, opisthotonus/emprosthotonus (may mimic seizures), elevated blood pressure. In very severe cases, pulmonary edema and cardiovascular collapse.

Lonomia caterpillar stings

- Local pain, headache, may be followed in hours to days by diffuse ecchymoses and systemic bleeding (with potential for fatal intracranial hemorrhage). May cause acute renal failure.

Snake bites

- *Viperids:* most cause local soft tissue swelling and pain within minutes of the bite; pain may be severe and swelling may progress over hours to involve the entire extremity and even the trunk; ecchymosis; over time, necrosis of local tissues; systemic findings may include nausea, vomiting, muscle fasciculations, altered taste sensations, weakness, bleeding (from almost any anatomic site), respiratory distress and shock.
- *Elapids:* most can cause neurotoxicity with resulting muscle weakness (generally starting with the muscles of the head and neck innervated by cranial nerves – ptosis, diplopia, difficulty swallowing) that may progress to peripheral paralysis and respiratory failure; cardiovascular instability may also occur; onset of signs or symptoms of envenomation may be extremely rapid or may be delayed many hours. Some species, such as the Indian cobra (*Naja naja*) can also cause local tissue damage, whereas others, such as the African spitting cobras (e.g., *Naja nigricollis*) have tissue necrosis as their primary complication.
- *Hydrophids:* primary findings include neurotoxic symptoms and signs similar to elapids, as well as pain related to myotoxicity.
- *Colubrids*: most of these species are completely harmless (bites result in no more pain that would be expected from multiple tiny pinpricks); bites by the dangerous boomslang or twig snake can cause potentially fatal systemic hemorrhage.

the victim may not present for hours or days – until local ischemic pain begins or a necrotic lesion develops.

How long before presentation did the bite(s) or sting(s) occur?

Some bites, such as venomous snake bites, usually prompt the victim to seek medical care promptly. On the other hand, a victim of some spider bites may not present for hours to days after envenomation.

What prehospital management measures were tried?

A number of first aid techniques for venomous bites or stings may be instituted by well-meaning “rescuers.” Many of these measures are of no value; some may even be worse than the bite or sting itself. For most arthropod envenomations, ice application and elevation of the affected body part are adequate first-aid therapies. It is difficult, however, to make recommendations for prehospital management of snake bites, as there are no proven techniques that positively impact outcomes in such cases. Techniques to avoid include application of electric shocks, incision and suction, use of tourniquets, or application of topical poultices, such as meat tenderizer. It is reasonable to immobilize the bitten extremity, but attention should be focused at delivering the snake bite victim to medical care as quickly as possible.

What is the victim’s tetanus status?

Because any bite or sting results in a break in the skin with concomitant tetanus risk, adequate immunization status should be ensured and updated as indicated.

Past medical

Comorbid conditions, such as heart or lung disease, may make the overall envenomation syndrome worse. Prior history of a bite or sting by a similar creature may be important if the victim suffered an allergic reaction to the injury or if antivenom therapy was required. Victims suffering an anaphylactic reaction to a hymenoptera sting who are currently on beta-blocker therapy are much more difficult to treat, and may be refractory to epinephrine (Chapter 13).

Physical examination

When approaching a victim of severe envenomation, it is best to conduct a rapid, focused initial examination while simultaneously beginning lifesaving treatment (e.g., epinephrine administration to a victim of anaphylaxis). Once treatment is underway, a more thorough examination should be performed.

General appearance

Check for any evidence of cardiac or respiratory distress, including airway involvement.

Vital signs

These can be quite variable depending on the envenomation syndrome.

Hymenoptera sting with anaphylaxis: The victim may have a rapid heart rate, low blood pressure, and rapid respiratory rate. In a preterminal state, the heart rate may begin to fall and blood pressure may be unobtainable.

Spider bites and scorpion stings: With serious bites by neurotoxic species, blood pressure, heart rate, respiratory rate and possibly temperature may be elevated. As the severity increases, the victim may develop hypotension, and, with severe scorpion stings, pulmonary edema may ensue.

Caterpillar stings: Stings resulting in systemic toxicity may cause an elevation in heart rate, respiratory rate and blood pressure, largely due to pain. Hypovolemic shock may follow severe *Lonomia* envenomation with systemic hemorrhage.

Snake bite: Vital signs are variable. Viperid bite victims often have an elevation in respiratory rate, heart rate and blood pressure (often due to accompanying anxiety), but in severe envenomation, may present with vital signs consistent with shock due to venous pooling, third spacing and hemolysis.

Cardiac and pulmonary

Listen for quality and regularity of cardiac sounds and for adventitial lung sounds. Hymenoptera-induced anaphylaxis may result in acute bronchospasm and wheezing. In severe scorpion, widow spider, or snake envenomation, the victim may develop pulmonary edema with accompanying rales on lung examination.

Abdomen

The abdominal examination is rarely revealing. Occasionally, with widow spider bites, the victim will complain of severe abdominal pain and will have what appears to be severe guarding. This has led to negative exploratory laparotomies in cases in which a history of spider bite was not available. Widow spider venom–induced rectus abdominis spasm can, however, be differentiated from peritonitis by the lack of rebound following this spider bite, and by the fact that these victims tend to be restless, unable to find a position of comfort, unlike patients with acute peritonitis who prefer to lie as still as possible. Stool hemoccult testing should be performed to assess for occult blood in the setting of viperid snake bite or *Lonomia* caterpillar stings.

Neurologic

A careful examination focusing on cranial nerve (CN) and motor function (including respiratory effort and peripheral muscle strength) is important when the patient has been bitten or stung by an organism with neurotoxic venom (e.g., elapid snake bite or neurotoxic scorpion sting).

Skin

A close evaluation of the bite/sting site is important. Any retained bee stings (commonly referred to as "stingers") can be scraped away. There may be diffuse urticaria if the victim is experiencing an allergic reaction to the venom. Spider bites may present with minimal local findings (e.g., widow spider bites), or may have significant necrosis (e.g., severe recluse spider bites). Scorpion stings usually have few local findings (possibly some mild soft tissue swelling or bruising at the site following non-neurotoxic scorpion stings). Cutaneous ecchymoses following *Lonomia* stings may herald progression to coagulopathy. For snake bites, the bitten extremity may demonstrate puncture wounds (though the bite pattern can be misleading), ecchymosis and soft tissue swelling (Figure 47.6). The extremity should be marked at two or more sites proximal to the bite, and circumferences measured at these points every 15 minutes to help gauge progression of the envenomation until it is clear that the victim has stabilized.

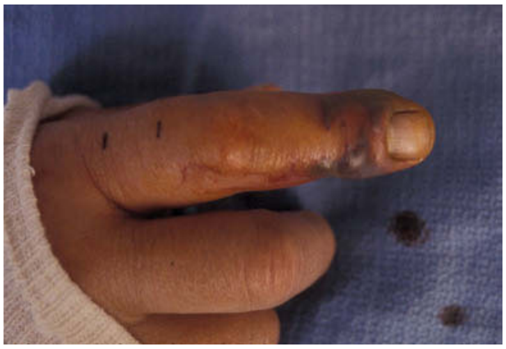

Figure 47.6
Rattlesnake bite to the distal index finger. Note the swelling, hemorrhagic blebs and bloody discharge.

Lymphatic

Many arthropod and snake venoms are absorbed via the lymphatic system and may cause an impressive lymphangitis. Regional nodes draining a bitten extremity should be assessed for lymphadenopathy.

Differential diagnosis

The differential diagnosis is usually straightforward in cases of venomous bites and stings. Often the victim will have witnessed the creature that inflicted the injury and be able to describe (or actually produce) it. If a snake is brought in with the patient, it should be evaluated cautiously, as even a dead snake or a decapitated snake head can have a bite reflex up to 1 hour after being killed, and can still render a serious bite.

In cases of delayed presentation of possible spider bite, arriving at the diagnosis can be more difficult when no spider was seen. In this scenario, the victim usually presents with a painful, swollen, red papule with or without an area of central necrosis. Such a lesion can indeed be caused by a spider, but is more likely the result of a bite from a flea, tick, bedbug, or other arthropod. It also may be caused by a non-arthropod source, such as an infected plant puncture wound, or a local response to a systemic illness (e.g., toxic epidermal necrolysis, erythema nodosum, or diabetic ulcer). Methicillin-resistant *Staphylococcus aureus* (MRSA) infections are commonly mistaken for unwitnessed spider bites.

Diagnostic testing

Laboratory studies

In most mild envenomation cases, laboratory studies are not necessary. There are very few diagnostic studies available to aid medical care providers treating victims of bites or stings. In Australia, an enzyme-linked immunosorbent assay kit is available to aid in identifying the offending species in suspected snake bites. These kits detect venom in wound aspirate, serum, or urine.

Laboratory studies may, however, be helpful in certain cases (Table 47.7).

Radiologic studies

Chest X-ray

A chest radiograph should be obtained in an envenomation syndrome whenever there are signs of respiratory distress, or if the victim has significant cardiorespiratory comorbidity.

Head computed tomography

Any victim of envenomation (especially venom that induces coagulopathy) who develops severe headache or altered mental status should be evaluated with a head CT to assess for possible intracranial hemorrhage.

Table 47.7 Laboratory testing for terrestrial venomous bites and stings

Complete blood count (CBC)
- *Suspected recluse spider bite with systemic (flu-like) symptoms:* to rule out hemolysis and thrombocytopenia.
- *Venomous snake bite and Lonomia caterpillar sting:* to obtain a baseline hematocrit (in case the victim develops coagulopathy with systemic bleeding or hemolysis); the white blood cell (WBC) count may be elevated (without secondary infection).

Electrolytes, renal function and creatine phosphokinase
- *Suspected recluse spider bite with systemic symptoms or any abnormalities on the CBC*: to rule out renal dysfunction.
- *Venomous snake bite*: to rule out significant rhabdomyolysis or renal insufficiency. Sea snakes (and some viperids and elapids) can cause significant rhabdomyolysis with resulting hyperkalemia, myoglobinemia, myoglobinuria and complicating renal failure. In these cases, a serum myoglobin and CPK should also be measured.
- *Lonomia caterpillar sting*: to monitor for acute renal failure which may complicate severe envenomation.

Coagulation profile
- *Venomous snake bite, suspected recluse spider bite with possible systemic toxicity, and Lonomia caterpillar sting*: a serum sample should be sent for measurement of prothrombin time, partial thromboplastin time, international normalized ratio, fibrinogen level and fibrin degradation products to assess for possible consumptive coagulopathy.

Blood type and screen
- *Venomous snake bite*: to allow for crossmatch of blood in the rare event that coagulopathy or hemolysis mandates the need for transfusion. Both circulating snake venom and any administered antivenom can interfere with crossmatch as time progresses.
- *Presumed recluse spider bite with suspected systemic toxicity*: to prepare for transfusion if needed to treat hemolysis or thrombocytopenia.
- *Lonomia caterpillar sting*: to allow transfusion of packed red blood cells in the event of severe bleeding and anemia (whole blood and fresh frozen plasma should be avoided, as these may worsen the patient's clinical condition by feeding fuel to an ongoing consumptive coagulopathy).

Urinalysis
- *Venomous snake bite, presumed recluse spider bite, and Lonomia caterpillar sting*: point of care testing should be done on each voided specimen during the acute phase of envenomation to detect hematuria or myoglobinuria.

Electrocardiogram

An ECG should be obtained in an envenomation syndrome whenever the envenomation is severe, there are significant comorbidities, or the patient has chest pain or shortness of breath.

General treatment principles

Treatment may need to begin before a precise diagnosis is made, as in the case of a victim of hymenoptera-induced anaphylaxis. Any restrictive clothing or jewelry should be removed as soon as possible.

Patients presenting to an ED for evaluation of a possible envenomation syndrome can be divided into two major groups. The first, and most common, is the clearly stable victim of a bite or sting who is concerned about possible complications (e.g., a victim who presents with a painful papule that might be a spider bite). The second is the potentially unstable victim of an acute envenomation that may be progressing (e.g., a venomous snake bite victim).

In the first type of patient, treatment is directed at trying to limit complications of the bite or sting. This involves supportive, conservative care including sound wound care (cleansing, dressing, splinting, and tetanus immunization as appropriate) and symptomatic treatment (e.g., antihistamines for itching). If signs or symptoms of secondary infection occur, the patient should receive appropriate antibiotics with good *Staphylococcus* coverage (including coverage for possible MRSA in appropriate clinical scenarios). If a recluse spider bite is suspected, local ice treatment (every few hours over the course of 2–3 days) may be beneficial in slowing the action of venom enzymes. Ice should be avoided, however, in venomous snake bites due to the risk of compounding necrosis. If the victim has any systemic symptoms, laboratory studies should be obtained as outlined above (Table 47.7).

For potentially unstable patients (e.g., victim of serious widow spider bite, neurotoxic scorpion sting, or venomous snake bite), attention must first be directed at ensuring adequacy of the ABCs, in that order. Airway management must be aggressive if the patient was bitten by an elapid snake and presents with any evidence of respiratory depression or difficulty swallowing secretions. Oxygen should be started, and cardiac and pulse oximetry monitoring instituted. Two large-bore IV lines should be started if there is any evidence of hemodynamic instability. If the blood pressure is low or the patient is significantly tachycardic, a bolus of IV fluid (normal saline) should be administered (20–40 mL/kg in a child; 500–1,000 mL in an adult, depending on the patient's cardiovascular reserve).

Antivenom

After assessing the envenomation syndrome, it is important to determine whether an appropriate antivenom exists, if it is available, and whether it is necessary. Antivenoms exist for many of the world's venomous snakes, for widow spiders, and for some funnel-web spiders and scorpions. There is also a relatively new antivenom for *Lonomia obliqua* stings (though it appears ineffective in *Lonomia achelous* stings). Correct antivenom selection is important, particularly when dealing with a venomous snake bite, as there is generally little benefit to using an antivenom produced for a remotely related or unrelated species. For example, in North America, there would be no benefit using pit viper antivenom to treat a coral snake bite victim. Conversely, widow spider antivenom is effective regardless of which *Latrodectus* species inflicted the bite. There are no commercially available antivenoms outside of South America for recluse spiders. A trivalent antivenom against *Loxosceles* and *Phoneutria* spiders and *Tityus* scorpions is produced by Instituto Butantan in Brazil. Although the efficacy of many scorpion antivenoms continues to be debated, an appropriate product (if available) should be administered to victims with significant stings.

Antivenom is generally indicated if the victim demonstrates evidence of significant envenomation or evidence of progression. Because not all hospitals carry all available antivenoms even against locally indigenous venomous creatures, a search for a source of the appropriate antiserum should begin early. Poison control centers or local or regional zoos may need to be consulted for assistance in locating antivenom, particularly in cases of bites by exotic species.

Most antivenoms are produced by injecting horses or sheep with gradually increasing doses of the venom or venoms of interest. Once the animal has developed immunity to the venom(s), antibody-laden serum is obtained, processed, purified (to variable degrees) and packaged, in a liquid or lyophilized form. Many of the most recently developed antivenoms are produced using technology to cleave the antibodies into functional *Fab* or *F(ab)*$_2$ fragments and deleterious *Fc* fragments (which are discarded). Some believe these antibody fragment products are more effective and safer to use than whole antibody products, although this remains controversial; there are certainly very safe, effective whole immunoglobulin G antibody products available.

Administration of any antivenom (route, dose, timing) can be guided by specific instructions in the package insert for the product chosen. In some countries, the recommended starting doses may be inadequate, and it is prudent, if possible, to discuss management with someone familiar with managing envenomation victims. The initial volume of antivenom needed can vary from a single, 10-mL vial of widow spider antivenom, to six or more vials of rattlesnake antivenom. Additional doses may be required if signs, symptoms, or laboratory abnormalities progress or recur. Antivenoms should be administered in an unbitten extremity whenever possible, in order to ensure adequate systemic distribution.

Because all antivenoms are currently derived from heterologous animal serums, they carry some risk of adverse reactions. These reactions can take the form of acute anaphylactic (allergic or non-allergic) reactions, or delayed serum sickness (generally presents 1–2 weeks following antivenom administration with symptoms such as hives, fever, myalgias and arthralgias).

Antibiotics are usually not necessary for the treatment of venomous bites or stings, and should be reserved for cases with suspected secondary bacterial infection.

Additional treatment

Potential recluse spider bites

If local necrosis is severe, these victims may require judicious debridement and/or skin grafting. Skin grafts should be delayed for 4–6 weeks to allow resolution of any ongoing venom effects. Some experts believe the use of dapsone (a polymorphonuclear leukocyte inhibitor) may reduce the extent of necrosis following these bites. Although theoretically and anecdotally of value, dapsone is not approved for this purpose and has significant dose-dependent side effects, making its use controversial. Likewise, research results have been mixed on the use of hyperbaric oxygen (HBO) therapy to limit necrosis. If readily available and easily accessed, HBO can be tried for particularly severe wounds.

Neurotoxic spider bites

A combination of benzodiazepines and narcotic analgesics may be useful in the management of severe pain, muscle spasms and agitation often seen with this syndrome. Calcium gluconate, although mentioned anecdotally by some authors for the management of *Latrodectus* bites, is of little or no proven benefit.

Scorpion stings

In some countries, victims of severe scorpion stings with pulmonary edema are treated successfully with prazosin, often when antivenom has failed to produce improvement.

Caterpillar stings

Patients with significant pain refractory to standard analgesics may benefit from administration of long-acting local anesthetics (local injection or regional nerve blocks). Victims of *L. achelous* envenomation who develop severe coagulopathy may benefit from the administration of antifibrinolytic agents and cryoprecipitate, although whole blood and fresh frozen plasma appear to make the clinical situation worse. Any victim of *Lonomia* sting who develops renal insufficiency will need supportive care and possibly dialysis.

Venomous snake bites

Bites by viperid snakes often produce severe soft tissue swelling of the involved extremity. This swelling is usually restricted to the subcutaneous tissues, but in rare cases can occur within muscle compartments, resulting in possible compartment syndrome. Differentiating a compartment syndrome from severe, subcutaneous swelling without vascular compromise requires direct intracompartmental pressure measurement using a wick catheter, a needle and transducer, or a Stryker device. If pressures are found to be elevated (>30–40 mmHg), further antivenom should be administered while the limb is kept strictly elevated. If the victim's hemodynamic status is stable, a dose of IV mannitol (an osmotic diuretic) can be given in an effort to help reduce the intracompartmental pressure. If the pressure remains elevated over the next hour despite these treatments, a fasciotomy is required to ensure sustained blood flow to the muscles and nerves. Renal failure resulting from venomous snake bite requires supportive care and possibly hemodialysis. Although such renal failure is often temporary, chronic renal insufficiency can occur.

Special patients

As a general principle, victims at each end of the age spectrum may suffer more severe envenomation syndromes. Pediatric patients tend to receive the same venom load as an adult when bitten or stung, yet they have less body mass and circulating volume to buffer the venom's effects. In situations in which antivenom is required, pediatric doses meet or exceed those for adults due to this relatively greater venom load-to-body mass ratio. Elderly patients, likewise, may be more prone to severe venom effects in the face of comorbid conditions, and should be treated aggressively.

Disposition

Victims who are symptomatic but clearly stable following a bite or sting can be discharged from the ED after an appropriate period of observation (generally 6–8 hours). A victim of a hymenoptera sting who is asymptomatic but concerned about developing anaphylaxis (possibly due to a prior episode of anaphylaxis following a sting) should be observed for at least 2 hours in the ED. Victims of hymenoptera stings who experience a systemic reaction more severe than simple diffuse urticaria or mild bronchospasm should be admitted for observation, preferably to a monitored setting. Any patient who has experienced an anaphylactic reaction should, at the time of discharge from the hospital, receive a prescription for a self-administration epinephrine device (with instructions), instructions to obtain a Medic-Alert medallion, and referral to an allergist for further evaluation.

Patients who present with potential recluse spider bites can be discharged for daily wound checks (for 3 days) if they have no systemic findings (fever, flu-like symptoms). If systemic abnormalities are present (as manifested by signs, symptoms, or laboratory abnormalities), these patients should be admitted for IV fluids and monitoring for development of severe hemolysis and potential renal failure.

Victims of *Lonomia* caterpillar stings may develop coagulopathy many hours to days following envenomation, and should be warned of this possibility and told to return if they have evidence of bruising, bleeding, or other deterioration in their condition.

Victims bitten by a venomous snake who have no evidence of envenomation should be observed for at least 6 hours. If they remain asymptomatic and have normal laboratory tests and vital signs at 6 hours, they can be safely discharged with instructions to return if delayed signs or symptoms of envenomation appear. An exception to this 6-hour guideline exists if the biting snake was an elapid, due to the potential delay in onset of findings of envenomation. In these cases, victims should be admitted for 24 hours of observation. Any snake bite victim with signs or symptoms of envenomation should be admitted to the hospital. The patient should be in a monitored setting during any antivenom administration.

Pearls, pitfalls and myths

There are few topics in medicine as impacted by myths and folklore as the treatment of venomous bites and stings. Many anecdotal "remedies" still receive attention, particularly related to venomous snake bites. These include application of various poultices, use of extreme heat or cold, incision and suction, and electric shock therapy, to name several.

The keys to managing any acute envenomation syndrome are to be a keen observer, anticipate multi-system involvement, and promptly request specialty consultation from a regional expert or poison control center.

The major pitfalls in managing envenomations lie in:
- Performing an inadequate evaluation (particularly regarding the history and physical examination)
- Failure to periodically reassess the victim for progression of envenomation
- Failing to recognize the importance of abnormal vital signs
- Failing to aggressively secure the airway in a patient developing respiratory insufficiency
- Failing to secure and administer antivenom, when indicated, in a timely fashion
- Premature discharge of a victim of envenomation when the onset of systemic findings may be delayed (e.g., a victim of elapid snake bite)
- Diagnosing any lesion as a "brown recluse spider bite" in the absence of confirmatory evidence
- Failure to evaluate a victim of possible recluse spider bite with systemic symptoms for hemolysis or renal insufficiency
- Discharging a patient without appropriate follow-up and after-care instructions (e.g., failure to prescribe an epinephrine self-administration kit and referral to an allergist for a victim of hymenoptera-induced anaphylaxis)
- Failure to administer tetanus immunization when indicated, though these wounds can be considered low risk for tetanus unless secondary infection occurs
- Failure to suspect compartment syndrome and objectively measure intracompartmental pressures in a victim of viperid bite with severe extremity swelling
- Failure to seek early specialty consultation when needed

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48 Ethics and end-of-life issues

Michael A. Gisondi, MD

Scope of the problem

Challenging ethical issues may suddenly arise when caring for critically ill patients in the emergency department (ED). The rapid deterioration of such patients often prohibits lengthy deliberations about ethical dilemmas. Emergency physicians must possess a practical understanding of medical ethics and end-of-life care in order to address these cases in a thorough and efficient manner.

Terminology

Modern philosophy links the definitions of morality and ethics. In the simplest forms, *morality* is the difference between right and wrong, whereas *ethics* represents the critical study of morality. Individuals choose from a variety of sources of moral authority, such as religion, cultural norms, politics and law. As such, persons may regard situations or objects differently, based on the value systems espoused by their source of moral guidance. Ethics represents the cognitive evaluation of a principle or situation, acknowledging that individuals possess different moral backgrounds. Ethical dilemmas arise when there is a conflict of values between persons arguing for competing moral imperatives – when people cannot agree on what is right and what is wrong.

Medical ethics is a discipline that studies differences in value systems as they apply to clinical situations. Medical ethics is most commonly taught through classroom discussion, as a means to familiarize providers with common ethical principles. *Applied health care ethics* is the practical extension of such discussion, recognizing that like all clinical decision making, ethical dilemmas require action. The word "applied" then refers to the reality that physicians mediate ethical dilemmas and make tough decisions every day. They are not philosophers, but practitioners of medical philosophy.

Ethical theories

Most American physicians guide their ethical decision making from duty-based concepts known as the "principles of biomedical ethics." These principles include respect for autonomy, non-malfeasance, beneficence and justice. *Respect for autonomy* is demonstrated when the patient is given the ability to exhibit self-governance, or self-determination. Patients should be allowed to make choices regarding their own health care. *Non-malfeasance* is loosely translated into the statement "do no harm." Physicians have an ethical obligation to limit the risk of poor outcomes that may result from diagnostic or therapeutic interventions. *Beneficence* in health care refers to the fundamental challenge to optimize a patient's condition and well-being; this may be through treatment of disease or provision of comfort care. *Justice* refers to the fair and equal treatment of patients, both in access to and quality of health care. Justice is also manifest through systems and institutional ethics, which in today's marketplace must respond to the reality of limited health care resources.

A competing, but no less valid ethical theory is that of virtue ethics. Defined in medieval times, *virtues* were those character traits that shaped professional ethics in medicine for centuries. Among the most critical markers of character for the virtuous physician were fidelity, trust, compassion, temperance, integrity, prudence, justice and self-effacement. For patients, the virtues of love (of something, such as health or life) and faith (that life can continue or health be restored) translate into the modernly accepted virtue of hope. These latter virtues serve as the basis of value systems employed by some critically ill patients and their families, and as such, should be considered when making ethical decisions.

Making ethical decisions in the ED

The pace with which emergency physicians must make clinical and ethical decisions does not allow for extended discussions of ethical theory. Instead, physicians may benefit from a practiced, step-wise approach to ethical decision making. First, one must recognize that an ethical dilemma exists. Next, the physician must choose an ethical framework to guide the deliberation process in an organized manner. Finally, an action must be chosen and one's reasoning reflected in the medical record.

Recognizing the ethical dilemma

Ethical dilemmas must be recognized and characterized. They result from *conflicts* between values or the interpretation of values by patients, their families, physicians, staff, the hospital, society, cultural norms, the law and others. Once physicians recognize that tensions exist, the ethical conflict should be characterized as simply as possible. *"Mrs. A is in respiratory failure. Her daughter wants her intubated, despite a valid 'do-not-resuscitate' order in the patient's chart. The daughter's wishes (hope, beneficence) are at conflict with the physician's obligation to his patient (prudence, respect for autonomy)."*

Choosing an ethical decision-making framework

An ethical framework differs from a theory or Code of Conduct in that it represents a systematic, step-wise approach to addressing ethical dilemmas. It is impractical for emergency physicians to work through a detailed framework in an emergent situation, but familiarity with the steps of a given framework will make critical, time-sensitive decisions proceed more smoothly. Two widely accepted ethical frameworks are Thomasma's "Ethical Workup Guide" and Iserson's "A Rapid Approach to Ethical Problems."

The ethical workup guide (David C. Thomasma, PhD, 1978)

Step 1: What are the facts in the case?
Mrs. A is in respiratory failure following a long, progressive functional decline due to end-stage congestive heart failure. She has had ample time to consider the details of her living will, and apparently made an authentic choice to decline intubation. Her daughter has been estranged from her for many years, but now asks for "more time" in order to reconcile their differences.

Step 2: What are the values at risk in the case?
The daughter exhibits the virtue of hope. The physician realizes the futility of prolonging Mrs. A's life. The daughter is also genuinely beneficent, in that she believes intubation is the optimal treatment for her dying mother. The physician has a duty to respect his patient's autonomous choice to decline lifesaving procedures.

Step 3: Determine the conflicts between values and professional norms, and between ethical axioms, rules and principles.
At conflict are the daughter's beneficent hope and the physician's prudence and duty to respect his patient's autonomy.

Step 4: Determine possible courses of action, as well as the values and ethical principles each course of action would protect or infringe.
Intubation might temporarily extend Mrs. A's life, in accordance with the daughter's wishes. Noninvasive mechanical ventilation such as bilevel positive airway pressure (BiPAP) would be an option more respectful of Mrs. A's wishes not to be intubated and the physician's obligation to her. BiPAP may offer symptomatic relief for Mrs. A and provide some additional time with her daughter.

Step 5: Make a decision in the case.
Noninvasive ventilation instead of endotracheal intubation.

Step 6: Defend this course of action. Why is "X" better than "Y."
Respect for patient autonomy is more important than the perceived beneficence of the daughter's wishes. One might also view the physician as being more beneficent to his patient by honoring her living will.

A rapid approach to ethical problems (Figure 48.1) (Kenneth V. Iserson, MD, 1995)

Step 1: Is this a type of ethical problem for which you have already worked out a rule, or is it at least similar enough so that the rule could reasonably be extended?

- *Yes*: Follow the rule.
- *No:* Proced to Step 2.

Step 2: Is there an option that will buy you time for deliberation without risk to the patient?

- *Yes*: Take that option.
- *No*: Apply the tests of impartiality, universalizability and interpersonal justifiability.
- *Test of impartiality:* Would you be willing to have this action performed if you were the patient?
- *Test of universalizability:* Are you willing to have this action performed in all relevantly similar circumstances?
- *Test of interpersonal justifiability:* Are you able to provide good reasons to justify your actions to others? Will peers, superiors, or the public be satisfied with the answers?

Choose an action and document your reasoning in the chart

Applied health care ethics expects that an ethical decision (action) will occur in parallel with other clinical decisions. Once the physician chooses a course of action, the thought process should be documented in the medical decision making section of the medical record. *"Given the patient's choice to decline intubation as described in her living will, I will support her respirations with noninvasive ventilatory methods (BiPAP), which appear to help her sense of dyspnea."*

Palliative and end-of-life care

Emergency physicians regularly encounter patients who are dying – some may present with complications of a recently diagnosed terminal illness, others may be critically ill and quickly expire. One of the most meaningful expressions of the patient–physician relationship comes from the thoughtful and considerate treatment of dying patients and their loved ones. *Palliative care* is the active, comprehensive care of patients who present at any stage in the dying process. Palliative care addresses the physical, psychologic, social and spiritual needs of patients and their caregivers from the time of diagnosis to death, including bereavement care for families after death when needed. Principles of palliative care cross medical disciplines and can be applied to any patient with a life-threatening illness. Core cognitive domains of palliative care in emergency medicine are summarized in Table 48.1.

Death trajectories, prognostication and goals of care

Patients with terminal illness present to the ED along one of four global death trajectories that describe progressive inability to perform activities of daily living over time. These death trajectories, originally described by Lunney et al., include (1) *terminal illness*, characterized by prolonged illness with a sharp decline in function; (2) *organ failure*, described as sharp overall decline in function with intermittent severe exacerbations of symptoms; (3) *frailty*, a

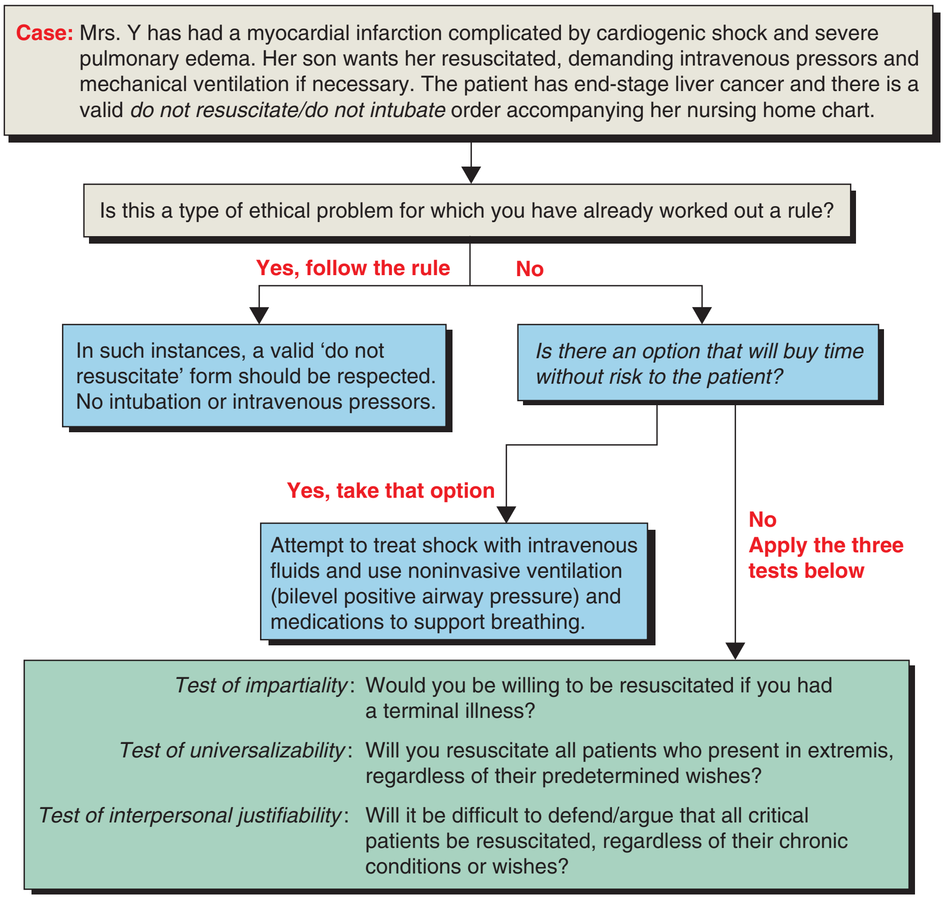
Case: Mrs. Y has had a myocardial infarction complicated by cardiogenic shock and severe pulmonary edema. Her son wants her resuscitated, demanding intravenous pressors and mechanical ventilation if necessary. The patient has end-stage liver cancer and there is a valid do not resuscitate/do not intubate order accompanying her nursing home chart.
Is this a type of ethical problem for which you have already worked out a rule?
Yes, follow the rule
No
In such instances, a valid 'do not resuscitate' form should be respected. No intubation or intravenous pressors.
Is there an option that will buy time without risk to the patient?
Yes, take that option
Attempt to treat shock with intravenous fluids and use noninvasive ventilation (bilevel positive airway pressure) and medications to support breathing.
No
Apply the three tests below
Test of impartiality: Would you be willing to be resuscitated if you had a terminal illness?
Test of universalizability: Will you resuscitate all patients who present in extremis, regardless of their predetermined wishes?
Test of interpersonal justifiability: Will it be difficult to defend/argue that all critical patients be resuscitated, regardless of their chronic conditions or wishes?

Figure 48.1
Using the rapid approach to ethical problems. Adapted from Iserson KV. An approach to ethical problems in emergency medicine. In Iserson KV, Sanders AB, Mathieu D (eds), *Ethics in Emergency Medicine*, 2nd ed., Galen Press, Tucson, AZ, 1995.

Table 48.1 Core palliative care domains in emergency medicine

- Understanding common death trajectories
- Prognostication
- Rapid assessment of palliative care needs
- Eliciting goals of care
- Advance directives
- Ethical and legal issues
- Caring for hospice patients
- Psychosocial aspects of care (including conflict resolution, spirituality, cultural considerations, and grief and bereavement)
- Withdrawing and withholding care
- Family-witnessed resuscitation
- Communicating bad news and death notification
- Treatment of common physical symptoms at the end of life
- Management of chronic pain
- Management of malignant pain
- Complications of cancer
- Care in the last hours of living

Adapted from Emanuel LL, Quest T (eds) for The EPEC Project. *The Education in Palliative and End-of-Life Care for Emergency Medicine (EPEC™-EM) Curriculum*. Northwestern University, Chicago, IL, 2008.

slow decline in function with death due to a complication of illness; and (4) *sudden death*, reserved for patients without a pre-existing diagnosis of terminal illness.

An understanding of death trajectories and their usual outcomes allows physicians to offer appropriate prognoses to patients, families and caregivers. Emergency physicians are often reluctant to offer quantitative prognoses, as they are generally evaluating their patients for the first time. However, a familiarity with the most common outcomes of terminal illness can guide physicians as they counsel patients and families regarding realistic goals of care. These family discussions can be especially useful in instances of severe trauma in elderly patients or prehospital cardiac arrest.

An accurate prognosis can better inform patients to set realistic goals of care for their immediate condition, as well as to express resuscitation preferences should their illness progress. Individualized goals help patients understand exactly what treatment options are available (i.e., "What are we doing, and why?"). Goal-directed assessments and plans ensure that providers avoid unwanted treatments, inappropriate use of resources, undue suffering and miscommunication.

The term Do Not Resuscitate (DNR) is a poor descriptor of a patient's detailed preferences for end-of-life care. For example, some patients may never want to be maintained on a ventilator for any amount of time, regardless of their prognosis, but would want to be defibrillated if they developed ventricular fibrillation. Physicians should approach discussions of resuscitation preferences not as an all-or-nothing DNR, but rather address separate questions of resuscitation within the context of an individual's illness and care goals (Table 48.2). The answers to specific questions should be listed in the chart following the DNR order (e.g., DNR includes: no compressions, no intubation, no defibrillation; yes to pressors, yes to intravenous [IV] fluids and artificial nutrition).

Table 48.2 Questions used to define resuscitation preferences

- If your heart were to stop beating, would you want chest compressions performed?
- If you were to stop breathing, would you want a breathing tube placed through your vocal cords into your windpipe so a ventilator could breathe for you?
- If your heart went into a life-threatening rhythm, would you want to be shocked out of it with electricity?
- If your blood pressure dropped to a dangerous level, would you want medications that might increase it or return it to normal?
- Is it acceptable to give you intravenous fluid and antibiotic medications in an attempt to treat your condition?

Identifying surrogate decision makers

Surrogate decision makers should be identified in the event a patient lacks decision-making capacity at a later time. Surrogates should have an appreciation of the patient's end-of-life preferences, so that they make future decisions in accordance with the patient's wishes. Surrogates can be named in a number of ways. *Next of kin* is a common method that states use to legally identify surrogates, if one has not already been named in an advance directive. *Advance directives* are lengthy, descriptive statements that provide a detailed discussion of patient preferences, including the identification of a surrogate decision maker. Advance directives are much more involved than DNR/Do Not Intubate (DNI) forms, which generally include answers only to specific resuscitation questions. *Living wills* can provide even more information, addressing preferences for potential organ donation and the handling of the body. A *health care proxy* form refers to the legal designation of a surrogate, only after prior consultation with an attorney. This proxy can be arranged alone, as part of an advance directive, or in conjunction with a living will.

Once resuscitation preferences and a surrogate decision maker are known, the medical team can better plan for future decisions to escalate or withdraw life-sustaining care. When such information is unknown, society expects physicians to fully resuscitate dying patients with two ultimate goals in mind: to save their life and to restore them to a state in which they can make autonomous decisions. If patients are later found to have valid DNR/DNI orders, it is ethically justified to withdraw life-sustaining care in accordance with their previous wishes. Although withdrawal of care can be emotionally upsetting to health care providers, it represents a profound demonstration of one's respect for patient autonomy.

Death notification

Death notification is an essential skill that must be practiced and refined by emergency physicians throughout their careers. Performed correctly, a culturally sensitive death notification can provide a supportive context for loved ones to begin grieving and processing their loss. When performed poorly, families and caregivers may remember the physician as uncaring, untrustworthy and poorly skilled – no matter how well the provider cared for the patient – and may not begin their grieving.

It is important to keep in mind that although the primary objective is death notification, there is certain information that must be obtained from family members as well (Table 48.3). Before you begin, prepare yourself for this discussion – gather your thoughts and make sure that your appearance is professional. When you enter the room, introduce yourself and ask the names and relationships of everyone present. Confirm that you will be discussing with them the correct deceased individual, and that they would have had permission to know the medical details of the case. Always notify the surrogate decision maker or next of kin before speaking to other family and friends. Explain exactly what you knew of the situation and what interventions were performed. Specifically state that the resuscitation was unsuccessful and that the patient *died*. Do not use euphemisms for death (e.g., “passed away, gone to the great beyond, passed on, didn't make it”), as this may be confusing to some family members. Pause for a moment and allow family members to process the information and develop their response. Use a sympathetic voice and offer physical gestures of comfort, such as hand-holding, if this seems appropriate.

Table 48.3 Important steps in death notification

- Prepare your thoughts and ensure that your appearance is professional.
- Introduce yourself as the physician in charge.
- Notify only those individuals who have permission to hear about the details of the case, after confirming that these are the correct individuals.
- Identify next of kin and notify these individuals before addressing other family and friends.
- Briefly obtain any relevant information about medical history, events leading up to the involvement of prehospital personnel or the emergency department.
- Succinctly describe the emergency department course and interventions.
- State that the resuscitation was unsuccessful and that the patient died. Do not use euphemisms.
- Pause and respond to grief with sympathetic gestures.
- Acknowledge efforts by prehospital staff, nurses and family members.
- Obtain necessary information about the primary care provider's name and contact information, and plans for organ donation, postmortem examination, and the body.
- Answer questions (including an offer to answer future questions), address the needs of those present, and leave the family in the care of a nurse, chaplain or social worker.

Acknowledge the efforts of the prehospital providers and nursing staff, especially if they had interacted directly with family members. Reassure loved ones that they should not blame themselves, as family members often feel the outcome may have been different had they intervened earlier. Reassure them that their loved one did not suffer. Offer to answer any questions, maintaining a sympathetic yet direct tone about the events and the outcome. Provide your business card or office number to allow families to contact you with future questions.

The death notification discussion should end by addressing the needs of those present. Utilize your support staff and have them remain after the notification; never leave a family member alone when you exit the room. Ask if they would like a nurse or social worker to assist in contacting a funeral home and making arrangements. Inquire about spiritual needs and offer to have someone contact a chaplain or spiritual advisor. Acknowledging cultural or religious needs may be the most comforting thing you can do for families at this time. Ask those present if they would like some time with the deceased. Prepare them for what they will see, and make certain someone from the healthcare team is available to be with them. Lastly, remember to ask if the surrogate requests a postmortem examination; this question is mandated by law in many states.

Special ethical issues in teaching hospitals

Unique ethical dilemmas arise in academic medical centers as a function of the training environment. The inherent nature of apprenticeship in medical education sometimes conflicts with patient expectations that their care be delivered by only the most experienced individuals. Ethical issues arising from the teaching environment require special attention by both the instructor and learner.

Informed consent

Legal and ethical principles mandate that physicians obtain informed consent for all treatments delivered. Patients are generally asked to consent to treatment when they arrive at the ED, indicating that they agree to be examined by the physician and receive "routine" care (i.e., blood tests, radiographs and medications). Although this initial consent implies that patients understand the benefits of such care, physicians should make every effort to review the risks of all significant interventions (i.e., new medications that may cause adverse reactions). More "invasive" procedures require additional information before a patient can truly give informed consent. At many hospitals, this process includes separate consent forms providing added documentation that a special discussion occurred.

There are four steps to obtaining informed consent:

1. The physician should assure that patients possess the capacity to make an informed choice. *Capacity*, which refers to the ability to understand one's options and make an authentic choice, differs from the legal term *competence*. Competence is a court-determined judgment of capacity. Emergency physicians may comment on a patient's decision-making capacity, but are not permitted to declare someone incompetent. If a patient cannot understand treatment options as a result of organic or psychiatric illness, a physician should describe them as "lacking decision-making capacity." Patients who lack capacity cannot consent to treatment, so a health care proxy should be identified (i.e., next of kin or predetermined surrogate decision maker). Such patients may still be able to agree to treatment; however, both the consent and assent should be documented.
2. The risks and benefits of proposed interventions should be reviewed in detail. This need not be an exhaustive list of all possible outcomes, but rather a discussion of the most common and serious complications. Physicians should make every effort to provide an honest risk–benefit analysis, including alternative treatments and the option to have no intervention. This step is critically important in teaching hospitals. Students and residents should not obtain consent for a procedure or treatment if they are unable to provide detailed risk–benefit information. Additionally, patients should understand which team member will be performing an invasive procedure, as well as the designated attending physician responsible for supervision.
3. The patient must comprehend the information discussed. One way to assess such understanding is to ask the patient to repeat the salient points from the conversation, in his or her own words. They should not simply repeat medical jargon. Patient questions should be elicited and answered as well.
4. Patients should confirm that they feel comfortable with their decision, acknowledging that their choice was voluntary and without duress. Both medical staff and family members can unduly influence patients to provide consent that is neither authentic nor autonomous. It is acceptable and ethically responsible to ask patients if they felt that they made their decision without feeling pressured.

Research ethics and the emergency exception to consent

Teaching hospitals often conduct research activities as part of their academic mission. Patients identified as potential research subjects for studies must first provide informed consent prior to their participation. It is sometimes difficult to obtain consent for emergency department-based studies, as patients often present gravely ill and alone. They may lack the decision-making capacity necessary to give consent and have no surrogate available to speak on their behalf. This complicates emergency medicine research, because it limits the ability to enroll subjects and conduct studies. Additionally, patients who might benefit from novel interventions are sometimes precluded from receiving such care if they are unable to provide consent.

The Food and Drug Administration (FDA) recognized these limitations and defined a set of guidelines termed the "waiver of informed consent for emergency research." This waiver of consent can be granted by human subjects committees or institutional review boards if:

1. There is a necessity for such research (study subjects have a life-threatening condition for which current treatments are unsatisfactory); or
2. There is a prospect for direct benefit to the subjects (risks are reasonable given the critical nature of the medical condition); or
3. Informed consent from patient representatives will be pursued (follow-up consent from surrogates or community notification of the ongoing study).

Although these guidelines offer researchers a method by which to conduct emergency medicine studies, they provide important ethical assurance that research subjects will be protected and respected.

Procedures on the newly dead

Recently deceased patients are occasionally used to teach lifesaving, invasive procedures to students and residents at teaching hospitals. An ethical dilemma exists between the need to respect the integrity and autonomy of patients and family members and the need to train health care providers. In response to this issue, the American Medical Association's (AMA) Council on Ethical and Judicial Affairs created a set of guidelines to help institutions ensure ethically responsible behavior in such learning environments. Prior to performing procedures on the newly deceased, the following considerations should be addressed:

1. The teaching of lifesaving skills should be the culmination of a structured training sequence, performed under close supervision and in accordance with the wishes and values of all involved parties.
2. Physicians should attempt to assess whether the deceased had expressed preferences for the handling of his or her body after death. If not, consent should be obtained from family members before proceeding. In the absence of expressed preferences or surrogate consent, physicians should *not* perform procedures for training purposes. These guidelines were incorporated into the 2003 AMA Code of Medical Ethics.

Pearls of wisdom: issues of professional ethics for physicians-in-training

- *Do not lie.* The pressure to perform often leads students and residents to misrepresent their knowledge or actions. Avoid the trap of embellishing history and physical examination details in order to appear more competent to your evaluators. Patient well being should always be the overriding priority.
- *Do not misrepresent yourself.* Emergency patients are acutely ill and vulnerable. They expect the best care possible and will assume that students or residents have more training than they actually do. Clearly describe your role on the health care team and be honest about your level of training. It is the attending physician's responsibility to supervise care and answer patient questions regarding the hierarchy of the teaching hospital.
- *Do not perform procedures if you feel uncomfortable.* Busy EDs often afford too much autonomy and responsibility to their trainees. Learners may be asked to perform procedures they have never seen or read about previously. Grades and evaluations are not as important as patient safety. Ask for supervision with procedures requiring skills you have not yet mastered, and refuse to perform them without assistance.
- *Do the right thing.* As a member of the health care team, you have an obligation to your patient. Sometimes you may feel that an ethical issue has arisen, but others on the team may be unaware or unconcerned. It is your duty to your patient to discuss such ethical issues with the team. It may result in a learning moment for you, as the attending physician may not have taken the time to explain his or her decision making. In other instances, you may feel it necessary to seek additional guidance elsewhere. Hospital ethics committees are available for consultation by any member of the health care team, including nurses, ancillary staff and physicians-in-training.

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49 Legal issues in emergency medicine

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Scope of the problem

Emergency physicians (EPs) interact with the legal system almost daily during the performance of their patient care and administrative duties in the emergency department (ED). Prominent legal issues in the ED include the patient's right to consent to or refuse medical care, or withdraw their consent for medical care; patients who are incapable of making decisions regarding their health care, such as minors and incapacitated individuals; state and federal regulations; proper documentation in the medical record; and criminal and civil law issues. EPs often interact with local, state and federal law enforcement personnel as they assess and treat trauma patients and battered children, spouses and elders, and participate in disaster scenarios. Over the past 20 years, state and federal governments have become increasingly involved in the oversight and regulation of medicine. Major federal statutes and administrative regulations, such as the Emergency Medical Treatment and Active Labor Act (EMTALA) and the Health Insurance Portability and Accountability Act (HIPAA), govern patient care in the ED. Furthermore, hospitals, health care networks, nursing homes and laboratories must be certified by the Joint Commission before they may participate in the Medicare program. Physicians must have a state license to practice medicine, and state and federal licenses to prescribe controlled substances. Likewise, most states have laws and regulations dealing with issues such as medical malpractice, advance directives, withdrawal of life support, informed consent, and treatment of minors.

This chapter examines common legal patient care issues, discusses the major federal statutes that govern patient care in the ED, details the essential elements of the medical record, and reviews how the criminal and civil law systems involve the ED.

Patient care issues

Decision-making capacity

All individuals with legal capacity have a constitutionally guaranteed right to accept or reject medical treatment and to withdraw consent for treatment at any time. Thus, any discussion of a patient's right to accept, refuse, or decline medical treatment in the ED begins with the premise that an EP knows whether a patient has decision-making capacity (DMC). That is, does the patient have the mental capacity to make informed decisions and to understand the consequences of accepting, refusing, or withdrawing consent for medical care?

As a general rule, all adults are presumed to have DMC. The EP must elicit and document objective data that prove a patient does not have it. Thus, a routine part of a patient's evaluation in the ED should include a determination of the patient's DMC. It is important to remember that EPs do not determine whether a patient is competent. *Competence* is a legal status determined by the courts. Furthermore, determining DMC is not a subjective determination. It is based on objective data obtained from the history and physical examination that demonstrate a patient has the mental capacity to understand and assimilate information and communicate a choice. A patient with DMC must be able to understand the information presented, deliberate the information, and present a choice and the reasons for it. The patient should also be able to state his or her values and goals that factored into the decision (Table 49.1).

EPs must employ the history and physical examination to determine whether a patient can comprehend the

Table 49.1 Determining decision-making capacity

Can the patient evaluate the information logically and rationally?

Does the patient realize his current medical condition or injury? If so, does he understand the suggested treatment?

Does the patient appreciate the consequences of refusing the recommended treatment?

Does the patient have personal values he can correlate with his decision to accept or reject the treatment?

Does the patient have a problem with language that would affect his ability to understand the information being presented?

Does the patient have trouble assimilating the information?

Does the patient forget the information shortly after the information is presented?

Suggested interview process:

- Does the patient understand the disclosed information?
  - Q: *"Tell me what you think is wrong with you."*
  - Q: *"How is the proposed treatment likely to affect you?"*
- Does the patient appreciate the consequences of her decision?
  - Q: *"What do you think will happen to you if you refuse the treatment?"*
  - Q: *"How would the benefits and risks of the treatment influence your daily activities?"*
- Does the patient use reasoning to make the choice?
  - Q: *"Explain how you reached your decision."*
  - Q: *"Help me understand how you decided to refuse the treatment."*
  - Q: *"Tell me what makes the treatment worse than no treatment."*

Adapted from Lo, B. *Resolving Ethical Dilemmas: A Guide for Clinicians*, 4th ed. Lippincott Williams & Wilkins, Philadelphia, PA, 2009.

immediate situation and its ramifications, and process the information in order to communicate a considered choice. The EP should pay attention to vital signs, level of consciousness, mental status and cognition. A mini-mental status examination assists in objectively demonstrating a patient's level of consciousness, orientation, mental acuity and comprehension. Patients who are unconscious, psychotic or demented are unlikely to understand their situation and related information, or communicate a deliberated choice. Furthermore, patients who are intellectually challenged, have language barriers, or have memory disturbances may have the same limitations.

If the physical and mental evaluations suggest that a patient has DMC, (s)he has the right to accept or refuse medical treatment. On the other hand, if the patient does not have DMC, then the EP may need to proceed with treatment in an emergency situation without consent or despite the patient's protests. In this situation, the EP should immediately contact a family member or the hospital's legal counsel to assist the patient in making health care decisions. Furthermore, if the patient lacks DMC, his demand to halt treatment or sign out against medical advice (AMA) should not be honored without further attention to the patient's condition.

It is important to remember that intoxication with alcohol or drugs does not automatically invalidate a patient's DMC. An intoxicated patient may have normal DMC, thereby maintaining the right to accept or refuse treatment. In cases of intoxication, the EP should note physical and mental findings and laboratory data that demonstrate that the level of intoxication has adversely affected the patient's DMC. Attention should be paid to vital signs, physical findings, mini-mental status examination, blood alcohol level and drug screens. The EP should use these data to substantiate that the severity of intoxication has adversely affected the patient's DMC, prompting the EP to render essential emergency treatment.

Finally, establishing the presence or lack of DMC is an ongoing process. It should be reassessed in patients without DMC and in those patients whom the EP suspects may lose or regain their DMC during the ED encounter.

Informed consent

Informed consent is a process whereby an EP obtains patient consent for a treatment or procedure through a discussion of the benefits, risks and alternatives to a proposed procedure or treatment. The doctrine of informed consent is judicially created based on fundamental concepts of autonomy, freedom from unwanted touching (battery), and privacy. It underscores a patient's right to participate in all decisions regarding his or her health care. The goals of informed consent are to foster communication between physician and patient, promote patient education, assure patient participation in determining the course of treatment, enhance rational decision making by physician and patient, protect the patient from unnecessary or unwanted treatment or procedures, and prevent fraud and duress.

Informed consent should be obtained during a formal conference where the EP, patient and interested family members discuss the patient's disease or injury and the procedure or treatment plan recommended. The conference allows the EP to educate the patient about his condition and the reasons why a certain course is recommended. The EP should complete all eight steps of the informed consent process and answer any questions the patient or family may have (Table 49.2). The patient's consent must be documented in writing, preferably on a preprinted form that includes space for the EP to note specific information about the procedure or treatment. The patient, the EP, and a witness who participated in the conference should sign the form. It is important to remember that, after being appropriately informed, the patient has the right to refuse the physician's recommendations. Accordingly, the conference acts as an informed refusal conference. The physician should detail the conference and the patient's decision in the medical record.

Table 49.2 The essential steps in obtaining informed consent

1. Nature of the patient's medical or surgical condition
2. Name, description, and purpose of the recommended treatment or procedure
3. Material risks[a] and potential complications of the recommended treatment or procedure
4. Chances of success and expected benefits of the recommended treatment or procedure
5. Risks of failing to undergo the recommended treatment or procedure
6. Alternative treatments or procedures
7. Risks, possible complications, and chances of success of the alternatives
8. Identity of the physician who will perform the treatment or procedure

[a]Material risks are those risks that a reasonable and prudent patient would want to know before deciding whether to accept or reject the proposed treatment or procedure. An easy way to determine material risks is based on *severity* and *numerosity*. Thus risks that are severe or occur frequently must be disclosed to the patient.

Express consent is the consent the patient gives in writing for evaluation and treatment of a specific injury or illness. All actions inherent and essential to the evaluation and treatment of the condition are implicitly included in the patient's express consent. Consequently, a repeat consent is not required for each action. For example, a patient presents to the ED seeking treatment for a laceration to his hand and gives express consent allowing the EP to evaluate and treat the laceration. The intrinsic steps involved include a detailed examination of the hand and forearm, cleaning the wound, updating tetanus (if indicated), obtaining an x-ray, initiating an intravenous line for antibiotics, and repairing and dressing the wound. All of the actions can be undertaken without the need of a separate consent for each. Another example is a patient who presents to the ED with fever, chills, headache and stiff neck suggestive of meningitis. The patient's initial consent allows the EP to take all necessary steps to evaluate the patient's signs and symptoms and provide treatment. Thus, the EP can perform a history and physical examination, perform necessary blood work, administer steroids and antibiotics, perform a lumbar puncture, obtain spinal fluid for analysis and cultures, and arrange for admission without the need of a separate consent for each of the actions. It is reasonable, however, for the EP to obtain a separate informed consent for the lumbar puncture (despite express consent), if the clinical situation allows.

The emergency exception to informed consent

In general, informed consent must be secured before an EP can institute any treatment or begin any procedure. However, emergency situations that meet certain criteria may be an exception to this rule, and the EP may initiate emergency treatment without prior informed consent. It is well accepted that an unconscious person with a severe injury would desire treatment despite the fact that they cannot consent to the treatment. The emergency exception becomes operative when (1) the patient has an injury that threatens life or limb and immediate treatment or intervention is required, and (2) the patient is unable to comprehend the situation because of unconsciousness, altered level of consciousness, mental status changes, acute psychosis, dementia, severe intoxication, or language barrier. Importantly, the treatment or procedures employed under the emergency exception are those necessary to save the patient's life or limb. Furthermore, the exception is only valid when both elements exist simultaneously. The exception covers both children and adults.

Although the emergency exception may allow the EP to initiate emergency treatment without prior consent, the EP must attempt to obtain consent from a family member as soon as possible. The emergency exception may not be utilized to avoid securing a patient's consent. It may only be invoked if the patient's condition meets the requisite criteria that give rise to the exception.

Signing out against medical advice

Patients with DMC have the right to refuse treatment and to withdraw their consent for evaluation and treatment. Patients with DMC have the right to desert or elope from the ED whenever they choose before or after they register for treatment. They are not required to obtain permission to leave the ED, nor are they required to sign specific forms to do so. Nevertheless, it is important that dissatisfied or unhappy patients not be allowed to elope from the ED after registering for treatment. Therefore, every ED should have a formal process by which the EP is notified immediately of any patient who desires to elope. Once notified, the EP should initiate a discussion with the patient regarding his desire to leave. Requiring a patient to sign out against medical advice (AMA) gives the EP an opportunity to discuss and address the patient's concerns, to update the patient on the progress of their evaluation and treatment, and to inform the patient of the risks of leaving the ED without completing their evaluation and treatment. In all cases, the EP must not allow a patient with diminished DMC to elope from the ED or to sign out AMA (Table 49.3).

Table 49.3 Process for addressing patients wishing to leave the ED prior to discharge

Patients should not be allowed to simply walk out of the ED.
The EP must be immediately informed of any patient who threatens to leave the ED.
It is the EP's responsibility to direct the AMA process and secure the patient's signature on the AMA form.
The EP should evaluate and document the patient's DMC.
The EP should determine whether there is a language or educational barrier affecting the patient's appreciation of what is transpiring in the ED.
The EP should explain to the patient the risks of leaving the ED before the evaluation and treatment are completed.
The EP should use all available resources to prevent the patient from leaving, including family, clergy, friends and other health care providers.
The EP should discuss reasonable alternatives.
The ED staff should apologize for long waits or inadvertent circumstances causing a delay in the patient's care.
The patient should sign a preformatted AMA form, witnessed by a health care professional who participated in or heard the discussion.
The entire AMA encounter must be documented in detail in the patient's medical record.
Any health care providers who participated in the AMA process should write ancillary notes detailing what transpired.
Patients with diminished or absent DMC should not be allowed to elope from the ED or sign out AMA.

AMA: against medical advice; DMC: decision-making capacity; ED: emergency department; EP: emergency physician.

Involuntary detainment

Occasionally, the EP may determine that a patient lacks DMC to sign out AMA. In such cases, the EP may be required to hold a patient against their will. This is referred to as *involuntary detainment*. Common situations involving involuntary detainment include patients who are a threat to themselves (suicidal), threats to others (homicidal), or a patient without DMC who requires urgent or emergent treatment. In situations in which the patient is suicidal or homicidal, the EP can initiate a physician's emergency certificate (PEC), a legal document that allows the EP to hold a patient against his will for further psychiatric evaluation and treatment. The PEC is typically limited to 72 hours, during which time the patient should receive necessary evaluation and treatment. Under a PEC, patients must also undergo a formal psychiatric evaluation to determine whether they require hospitalization for being a threat to themselves or to others, if further psychiatric intervention is required as an outpatient, or if they can be discharged from the ED. Importantly, a PEC may not be utilized to detain a patient whom the EP feels should not leave the ED without completing evaluation or treatment. An EP who invokes a PEC under false pretenses may be liable to the patient for assault, battery and false imprisonment. As a general rule, only physicians may initiate a PEC; they should not be invoked by nursing or prehospital personnel. However, in some situations, police and prehospital personnel may initiate a PEC in the field if they are directed by an EP during online medical control.

The other situation in which involuntary detainment may be necessary is when a patient has an organic disease process that adversely affects his mental status and DMC. The EP may need to hold the patient to provide urgent medical treatment necessary to stabilize his condition. In such a situation, the EP should document the patient's vital signs, level of consciousness, performance on a mini-mental examination, and factors that adversely affect the patient's DMC. An example is a middle-aged male presenting to the ED with acute delirium tremens and hallucinations screaming at everyone while the nurse attempts to establish an intravenous line. He states that he does not want treatment and wants to leave. His vital signs reveal hyperthermia, hypertension and tachycardia. He is confused and disoriented to time and place, and has generalized tremors. In this situation, the EP should detail the patient's lack of DMC. Furthermore, the patient should not be allowed to leave the ED because of the severity of his condition, its effect on his ability to appreciate and understand its severity, and the consequences of refusing treatment. Therefore, the patient's demand to leave the ED would be overridden in order to provide emergent treatment.

In all cases of involuntary detainment, the EP should explain to the patient and family why the patient is being held in the ED. The EP should explain the plan for evaluation and treatment. If no family member is present, the EP should contact the family and request that a family member or guardian come to the ED to be kept informed of the patient's condition and participate in treatment decisions. Hospital security personnel should prevent the patient from harming himself or others, or eloping from the ED. The entire encounter must be carefully documented by the EP and nursing staff in the patient's medical record. Ancillary hospital personnel involved in the detention should document their participation as well. It is important to continuously reexamine and assess vital signs, level of consciousness, mental status, response to treatment, and presence or absence of DMC.

Treatment of minors

Legally, minors are children and adolescents less than 18 years old. Adolescents who are 18 years and older are considered adults and have the rights and responsibilities of adults, including the right to accept, reject, or withdraw from medical treatment. However, most states have adopted the *mature minor doctrine*, which allows adolescents 16 years of age or older who possess a certain level of maturity and intelligence the right to consent to medical treatment. These adolescents are termed "mature minors." On the other hand, any minor, even if determined to be mature by the EP, may not refuse medical care authorized by their parents or legal guardian.

When considering the mature minor doctrine, the EP should verify that the minor is at least 16 years old, determine the severity of the procedure or treatment the minor requires, and ascertain whether the minor understands the benefits and risks. The EP should next determine whether the minor would be considered mature by considering the factors listed in Table 49.4. If the EP concludes that the minor is mature, the EP may render the treatment being sought. The EP should document the reasons for concluding that the minor met the maturity standard in the medical record.

Table 49.4 Factors to consider in determining maturity of a minor

Academic training
Age
Conduct
Economic independence
Intelligence
Parental control
Physical maturity
Work experience

Additionally, most states have laws that allow minors 16 years of age and older to seek treatment for sexually transmitted illness, pregnancy, drug and alcohol abuse, and mental health care without parental consent. Furthermore, most states consider minors legally emancipated from their parents if judicially determined to be so, or if they are on active duty in the armed forces, married, pregnant, or a parent.

Advance directives

Advance directives are documents or video-recorded statements generated by patients with the purpose of detailing medical treatment they desire in the event they become severely ill and lose their DMC. With an advance directive, a patient appoints a surrogate decision maker who has the authority and responsibility to make treatment decisions consistent with the patient's directions. The EP should remember that an advance directive is not operative until the patient loses DMC. Thus, the EP may not honor the surrogate's directions or decisions until the patient lacks DMC.

When an EP is informed that a patient has an advance directive, the EP should review and discuss the advance directive in an attempt to understand the patient's intentions through the advance directive. The EP should also speak with the surrogate to elicit whether the surrogate understands their authority and responsibilities, and whether the surrogate is willing to comply with the patient's directives. Any reservations on the part of the surrogate should be addressed before the surrogate has to act. When in doubt as to how to proceed, the EP should consult the hospital's ethics committee for direction from an objective third party.

There are several types of advance directives. The first is the *living will*, which is a written document formulated under state law. It contains specific language and terms describing actions to be taken by health care providers. Typically, living wills are effective only when the patient's illness, injury, or disease is terminal and irreversible, and the patient does not have the DMC to make health care decisions. Living wills do not appoint a surrogate. Instead, they inform health care providers not to proceed with life-sustaining treatment if the patient's condition is terminal and irreversible. Living wills are continuous and permanent, unless orally revoked by the patient or their actions. Finally, witnesses to the living will may not be heirs to prevent conflicts of interest.

A second type of advance directive is the *Durable Power of Attorney for Health Care Matters* (DPAHCM). The DPAHCM is a written or video document patients utilize to limit or restrict specific medical treatments or interventions in the case of certain anticipated medical conditions. It is drafted by the patient with the help of an attorney and the surrogate. With a DPAHCM, the patient appoints a surrogate decision maker and lists the actions that the surrogate should or should not take if the anticipated event occurs. Importantly, the DPAHCM is only operative if the patient lacks DMC, at which time the surrogate becomes the decision maker for health care matters only.

A *Do Not Attempt Resuscitation* (DNAR) advance directive is a written document initiated by a patient (or their surrogate) during a specific medical encounter. A DNAR is limited to the particular ED encounter or hospitalization during which it was invoked and automatically ends at the termination of the specific encounter that gave rise to the DNAR. For example, a patient makes himself DNAR after suffering a myocardial infarction, but survives and is discharged from the hospital. The DNAR terminates when the patient is discharged from the hospital. It is neither valid nor operative during subsequent hospitalizations. Thus if the patient seeks to initiate a DNAR status during a succeeding hospitalization, the patient and physician must complete the DNAR process again. It is important to remember that a DNAR only prevents the EP from initiating cardiopulmonary resuscitation and advanced life support. It does not allow the EP to deny food, fluids, medications, or comfort, nor does it allow the EP to withdraw life-sustaining treatments already in place.

Federal statutes

Patient privacy and confidentiality: The Health Insurance Portability and Accountability Act

Patient privacy and confidentiality are important aspects of the physician–patient relationship. EPs have legal, moral and ethical responsibilities to protect the privacy and confidentiality of their patient's health care information. In 1996, Congress enacted the Health Insurance Portability and Accountability Act (HIPAA), which governs health care providers and organizations that create, store, or transmit health care information for health care transactions. Providers and organizations governed by HIPAA are called *covered entities* (CE), and include health care providers, health care plans, health care clearinghouses and business associates. A *health care provider* is defined as a person or organization that furnishes, bills for, or is paid to provide medical care. All health care providers are required to have a 10-digit national provider number, which must be used in all transactions covered under HIPAA. Health care providers include individual physicians, faculty practice groups and hospitals. A *health plan* is an individual or group plan that provides or pays for medical care. A *health care clearinghouse* is an entity that acts as an intermediary in the exchange of health information between health care providers and payers. A *business associate* (BA) is defined as a person or organization that performs services for a CE through a contract, such as a supply or billing company, a collection agency, or legal counsel. If the BA's activity exposes it to an individual's health information, the activity is governed by HIPAA.

Under HIPAA, all health information that can identify an individual is considered protected health information (PHI). CEs have a legal responsibility to safeguard an individual's PHI from unauthorized access by others. HIPAA distinguishes between the *use* and *disclosure* of PHI. CEs may use an individual's PHI without their consent for treatment, billing purposes and health care-related operations. *Treatment* is defined as the provision, coordination, consultation, referral, or management of health care services. *Payment* is defined as all actions taken to obtain or provide reimbursement of medical fees. *Health care operations* are activities the CE conducts to oversee, monitor, or improve the provision of health care. Health care operations include quality assurance, performance improvement, credentialing, auditing and general administration.

On the other hand, a CE discloses PHI when it utilizes or releases an individual's PHI for any purpose other than treatment, payment, or health care operations (such as issuing PHI to the press or marketers). Under HIPAA, a CE must secure an individual's written authorization before it can disclose an individual's PHI. The authorization must state the purpose of the disclosure and to whom it will be disclosed. HIPAA grants individuals the right to limit or restrict disclosure of their PHI. Importantly, PHI may be disclosed without a patient's authorization if required by law for law enforcement purposes, public health activities, criminal investigations, and threats to the health or safety of the public.

HIPAA is not intended to impede reasonable communications required to provide appropriate health care, nor is it intended to alter daily operations and procedures. Its intent is to prohibit release of PHI to unauthorized individuals; to prevent inadvertent, indiscriminate and negligent disclosure of PHI in conversations in public areas, such as elevators and cafeterias; and prevent unauthorized viewing by third parties.

HIPAA also grants individuals specific rights. CEs are required to provide each patient with a Notice of Privacy Practices upon their first visit to the physician's office or hospital, or upon enrollment into a health plan. Patients have the right to access and copy their medical records, the right to request modifications or corrections of their medical record, the right to request restrictions on the disclosure of their PHI, and the right to an accounting of disclosures. Patients may file HIPAA complaints with the CE or with the Office of Civil Rights. Sanctions for violation of privacy and security standards include civil monetary fines and incarceration.

The Emergency Medical Treatment and Active Labor Act

Prior to 1986, a critically ill patient arriving in an ED faced the possibility of being refused care if unable to pay. In 1986, Congress enacted the Emergency Medical Treatment and Active Labor Act (EMTALA) as a way of protecting the medically indigent and preventing disparate emergency medical care between those with and without the ability to pay for it.

EMTALA applies to any individual who presents to the ED seeking emergency treatment. The statute encompasses all individuals, even if they are not citizens or residents of the United States. Furthermore, it specifically provides that an individual may seek emergency care or that someone may request it on their behalf. Under EMTALA, any individual who "comes to" an ED seeking medical care must undergo a medical screening examination (MSE) to determine whether the individual has an emergency medical condition (EMC). Importantly, the MSE must include all capabilities of the ED, including ancillary services. If an EMC exists, the individual must be treated and stabilized before they can be transferred. The evaluation and stabilization must be provided regardless of the individual's economic status or ability to pay for medical services. Medical care may not be delayed to determine whether the individual has health insurance. EMTALA also requires EDs to keep a log of individuals who come to the ED, to post signs in the ED informing individuals of their rights under EMTALA, to maintain physician on-call lists, and to document physicians who refuse to provide timely treatment.

EMTALA regulations and court decisions have established that an individual has "come to" the ED when (1) the individual physically arrives in the ED; (2) the individual arrives on hospital property within 250 yards of the ED; (3) the individual is on hospital property and a prudent layperson observer would believe that the individual needs examination or treatment for a medical condition; (4) the individual was picked up by a hospital-owned ambulance service, unless the ambulance is operating under community-wide protocol that directs the ambulance to another hospital; or (5) the individual is transported by a non–hospital-owned ambulance that arrives at the hospital's ED.

EMTALA does not define an MSE, but requires that the extent of the MSE be based on the capability of the ED. The MSE does not include triage. Instead, it should be reasonably intended to identify an EMC. The MSE may not be disparate; it must be performed to the same extent for individuals with insurance and those without. Furthermore, it may not be delayed in order to determine whether the individual has insurance coverage.

EMTALA defines an EMC as an acute medical condition of sufficient severity (including pain, psychiatric disturbances, and symptoms of substance abuse) for which the absence of immediate medical attention could result in placing the health of the individual, a pregnant woman, or unborn child in serious jeopardy; serious impairment to bodily functions; or serious dysfunction of any bodily organ or part. Importantly, a pregnant female need not be in labor to be an EMC. Instead, EMTALA provides that a female who is having contractions is in true labor until a physician certifies that she is in false labor.

EMTALA mandates that if an EMC exists, it must be stabilized before the individual can be transferred. Stabilization is defined as the provision of such medical treatment as necessary to assure that no material deterioration is likely to occur during the transfer of the individual from a facility. Stabilization of a pregnant woman having contractions includes delivering the child and placenta. Stabilization does not require that the EMC be "cured" in the ED. Transfer is defined as any movement of the individual outside of the hospital's facilities, including discharge of the individual. Transfer does not include an individual leaving the ED on their own volition.

A potential conflict under EMTALA arises when a minor's caregiver, who is not the minor's parent (babysitter, relative, or teacher) presents to the ED with the minor and requests emergency medical care for the minor. Under EMTALA, the minor would have come to the ED and a request would have been made on the minor's behalf for emergency care. Thus the EP would be obliged to perform an MSE in order to determine whether an EMC exists without delaying to secure parental consent. If an EMC does exist, the EP would be compelled to proceed with stabilization. However, if no EMC exists, the EP would not be required to continue without parental consent. Although the EP could not delay the MSE or stabilization because of a lack of prior parental consent, the EP should use all means possible to locate a parent or parents to inform them of the situation, secure their consent, and request them to proceed to the ED to participate in medical care decisions.

EMTALA provides two exceptions to the general rule that an individual with an EMC must be stabilized before they can be transferred. First, an individual may request, without coercion, transfer to another institution. This request must be documented in writing on a transfer consent form, which must be signed by the individual. Furthermore, the EP must explain the risks of the transfer to the patient. The second reason is if the benefits of transfer outweigh the risks of transfer. For example, an individual with an open femur fracture presents to an ED that has no orthopedic surgeon. The benefits of transferring the individual to a hospital with an orthopedic surgeon would outweigh the risks of attempting to treat the injury without proper resources. In both cases, the EP must contact the receiving hospital and obtain approval for the transfer, send pertinent medical records and the signed transfer consent form, and arrange the appropriate mode of transportation and personnel to insure the individual's health and safety during the transfer.

The Joint Commission

When Congress established the Medicare program in 1965, it authorized the Joint Commission (JC) to be responsible for developing standards to accredit health care organizations and participate in Medicare. These standards promote and mandate appropriate medical care, performance improvement activities, patient safety, pain management, proper use of restraints and seclusion, patient confidentiality, fire safety, inspection and repair of medical equipment, medication management and organizational staffing. In addition, JC publishes National Patient Safety Goals each year designed to promote patient safety and prevent adverse outcomes.

Most JC standards directly impact the ED. For example, JC requires an organization-wide, multi-disciplinary policy regarding procedural sedation. Physicians must demonstrate competency in performing procedural sedation and must be credentialed to perform it. In addition, policies and procedures must detail how it is to be employed and monitored. JC also requires hospitals to have a written restraint policy, and the order form must be completed each time a patient is restrained. Furthermore, JC requires an organization-wide approach to pain management and requires health care institutions to inform patients of their right to pain control. Health care institutions must educate physicians and nurses about the safe and effective use of analgesics. Additionally, they must collect data on pain management outcomes and staff competency in pain control.

JC has published a "Do Not Use" list that includes the abbreviations that should not be used in any handwritten patient-related material. JC has also proposed other abbreviations that must not be used in the medical record (Table 49.5). Furthermore, JC prohibits using trailing zeros for medication doses, because the decimal point may be missed and result in a dose 10 times greater than that ordered. On the other hand, if the dosage amount is less than one, a zero must precede the decimal point. Thus, instead of ordering ".3 mg" of a medication, the order must be written as "0.3 mg" to assure that the decimal point is not missed.

Table 49.5 The Joint Commission's mandatory "Do Not Use" abbreviations

- U (*write* unit)
- IU (*write* international unit)
- QD (*write* every day)
- QOD (*write* every other day)
- MS (*write* morphine sulfate *or* magnesium sulfate)
- MSO$_4$ and MgSO$_4$ (*write* morphine sulfate *or* magnesium sulfate)

Other abbreviations that should not be used:
- H.S. (*write* half-strength or at bedtime)
- TJW (*write* 3 times weekly or three times weekly)
- SC or SQ (*write* Sub-Q, subQ or subcutaneously)
- D/C (*write* discharge)
- cc (*write* milliliter or ml)
- µg (*write* mcg or microgram)
- AS, AD, and AU (*write* left ear, right ear, or both ears)
- OS, OD, and OU (*write* left eye, right eye, or both eyes)
- < and > (*write* greater than and less than)
- @ (*write* at)

Medical Record

The medical record has become the primary means of communication between physicians, nurses, paramedics and non-medical personnel who interact with, evaluate and treat patients. It is also critical to those who submit fees for and pay for medical care. Importantly, the medical record is an essential component of the physician–patient relationship. The medical record allows appropriate, timely, pertinent, continuous, and coordinated medical care. It serves as a medical and legal document that facilitates non-verbal communication between physicians and other health care professionals participating in the patient's care; verifies medical fees and hospital charges; assists in resolving controversies; refutes or substantiates medical malpractice claims; and provides data for internal hospital *auditing*, peer review activities, *quality assurance* and medical research.

The EP should use the patient's medical chart to record the encounter in detail. Unless electronic documentation is available, all documentation must be recorded in ink, written legibly, dated, timed and signed. Only approved abbreviations should be used. In prolonged ED visits, the patient's vital signs should be repeated and reassessed. Furthermore, interval progress notes should detail successive patient reassessments and changes in clinical parameters. All actions taken to address the patient's condition, as well as the patient's response to them, should be recorded. All conclusions regarding the patient's medical condition or injury should be confirmed with objective clinical and laboratory data. The EP should consider drawing and explaining important physical findings.

The EP should review nurse and paramedic personnel notes, and reconcile any discrepancies without demeaning or judgmental language. Instead, the EP should document pertinent facts and observations that support modifications or corrections of the information in the patient's medical record. In addition, the EP should reference all documents that are not an integral part of the medical record. For example, the EP should include a notation that a patient signed a consent form for a procedure or signed an AMA form before leaving the ED. Important statements made by the patient, family, or bystanders should be accentuated with quotation marks. Colloquialisms, slang, or street language should only be used when quoting the patient. The EP should refrain from attempting to make the medical record entertaining.

Discharge instructions should be written or typed on a preprinted form in language easy for a lay person to understand. The instructions should provide the patient with the diagnosis; specific instructions for home care, work and activity; and timely and appropriate follow-up care. Patients should always be instructed to seek care sooner than prescribed if needed. The EP and nurse should present the discharge instructions to the patient during a discharge conference. They should document that the information was explained to the patient, and that the patient acknowledged understanding. If the patient has a problem comprehending, the patient's representative should be included in the discharge conference. Finally,

Table 49.6 Information to include in the emergency department medical record

Patient's name and contact information
Patient's time of arrival
Chief complaint, physical presentation at triage, vital signs
Medications and allergies
Immunization history
Name of the patient's primary physician
Pertinent history and physical examination, including mental status and behavior
Written and verbal orders
Listing of laboratory and radiologic studies, including the time ordered, the time performed, and the results of each study
Differential diagnosis
Treatment plan
Treatment rendered and the patient's response to treatment
Final diagnosis and patient disposition
Appropriate discharge and follow-up instructions written in lay language

the patient (or patient representative) should sign the discharge instruction form verifying that the instructions were explained and provided (Table 49.6).

Charting errors must be addressed appropriately. White-out should never be used to correct the medical record, and the medical record should never be altered. The proper way to correct documentation errors is to draw a single line through the inaccurate statement and write "error" or "void" in the margin next to it. The EP should include his initials, date and time next to the correction. Inserting addenda after the fact into the medical record is discouraged. However, if necessary, the EP should note that the information is a late entry, record the date and time of the entry, and limit the entry to the specific issue being addressed. Finally, destroying or altering the medical record is called "spoliation of evidence," and may subject the EP to civil and criminal liability.

The criminal and civil justice systems

The two major legal systems in the United States are criminal and civil. Interacting with both systems is an inevitable part of EM practice. EPs interact with law enforcement personnel, district and criminal defense attorneys, prisoners, and assailants and victims of crime almost on a daily basis. Criminal offenses are acts by individuals or legal entities that municipal, state, or federal legislative bodies have declared illegal because of their detrimental effect on individuals and their property. Criminal offenses are prosecuted by governmental agencies, state or federal attorneys general, and municipal district attorneys. For a criminal defendant to be guilty of a criminal act, the prosecutor has the burden of proving that the criminal defendant completed all of the elements of a particular criminal offense "beyond a reasonable doubt." The standard requires that a prosecutor produce evidence to a judge or jury that demonstrates that the criminal defendant committed the criminal act with greater than 99% certainty. The punishment for committing a criminal act is imprisonment and/or a monetary fine.

EPs should be aware of their responsibilities under local, state or federal laws regarding reporting child, spousal and elderly abuse, and reporting communicable diseases. Some states require that the EP report all victims of violence from guns and knives. Additionally, EPs must understand their duties and limitations when collecting evidence from victims of violent crimes and sexual assault, and when collecting blood and bodily fluids for criminal investigations. The EP must follow the proper *chain of evidence* in collecting evidence for law enforcement personnel. The EP must avoid destroying forensic evidence during treatment of a perpetrator or victim, such as cutting through bullet holes in clothing, destroying or discarding personal belongings and evidence, or placing chest tubes through gunshot or stab wounds. All wounds or injuries should be described objectively, including the appearance of the wound and its dimensions. They should not be described as entrance or exit wounds. A description along with a diagram or drawing of the wound or evidence should be documented. Any photographs must be submitted within the chain of evidence. The existence of photographs should be noted in the medical record. EPs should cooperate with criminal investigations and should understand their duties when served with a subpoena to testify in a deposition or criminal trial.

Alternatively, the civil justice system involves noncriminal acts or failures-to-act that damage an individual's person or property, or a legal entity or its property. Civil claims are private matters in which an individual or legal entity claiming an injury files a lawsuit against another individual or legal entity that caused the injury. Civil actions are initiated and contested in civil courts.

A *tort* is a wrongful act perpetrated by a tortfeasor that results in injury or damage to an individual, a legal entity, or their property. The party claiming injury or damages is the *plaintiff*. The party who allegedly caused the injury or damages is the *defendant*. A plaintiff files a claim in civil court against a defendant to obtain restitution for the alleged injury or to deter further wrongful acts. The main thrust of civil law is to make the plaintiff "whole again." This concept is to place the plaintiff where he would have been if the tortious act had not occurred.

Civil law allows a plaintiff to file a claim for an injury and allows a jury to conclude (1) whether the plaintiff is truly injured, (2) if the defendant caused the injury, and (3) what compensation should be rendered to the plaintiff. Remedies for civil actions include monetary compensation for damages, injunctive relief, restitution, and forfeiture. Monetary compensation may include compensation for loss of earning capacity, loss of consortium, pain and suffering, present and future expected losses, and future medical expenses. In order to prevail in a civil claim, the plaintiff must convince the jury that the defendant committed the alleged action by "a preponderance of the evidence." Thus the plaintiff must present evidence to prove that there is greater than 50% certainty that the defendant committed or failed to perform the specific act, and that an injury resulted from the defendant's action or inaction.

Torts can be divided into intentional and unintentional torts. *Intentional torts* are wrongful actions where a defendant clearly intended to injure an individual or his property. *Unintentional torts* are actions where the defendant caused injury to the plaintiff either through negligently acting or failing to act. *Negligence* is a type of unintentional tort. It exists when a defendant fails to exercise the appropriate level of care that a reasonable person possessing the same knowledge would have exercised under similar circumstances. In a negligence claim, there is no intent by the defendant to cause harm. Instead, the failure of a defendant to act within a certain standard causes the injury or damages.

Medical malpractice is a claim of negligence against a physician or other health care provider. Physicians and other professionals are required to provide their services in conformity with nationally accepted standards for their profession. Failure to comply with the standard of practice for the particular profession can render the professional liable to a plaintiff for damages. When a plaintiff files a medical malpractice claim, he or she asserts, and must prove, that the EP's actions were (1) inconsistent with the national standard practiced by other reasonable, similarly trained EPs, and (2) a deviation from the standard of care that caused the injury. The most common medical malpractice allegations made against EPs are failure to diagnose, negligent performance of a procedure, misdiagnosis, substandard treatment, delay in diagnosis, and delay in treatment.

In instituting a medical malpractice claim, a plaintiff's attorney will file a complaint (termed a *petition for damages*) into the court of proper jurisdiction. The petition for damages details the injury suffered by the plaintiff and the alleged actions or omissions by the EP that caused the injury. When the complaint is served on the EP, the lawsuit is formally initiated. The EP is required to contact the malpractice insurance carrier, who will begin the defense of the claim by assigning an attorney to defend the EP. The defense attorney must file a response to the plaintiff's complaint, which is called an *answer*. The answer allows the EP to deny or admit to the plaintiff's allegations. Once the answer is filed, the formal process of discovery and litigation begins.

In a medical malpractice claim the plaintiff must prove, by a preponderance of the evidence, that the EP met all four elements of negligence. These are (1) the EP owed a duty to the plaintiff, (2) the EP breached that duty, (3) the plaintiff suffered an injury as a result of the breach of duty, and (4) the injury was directly related to the breach of duty by the EP. At trial, the plaintiff goes first and produces witnesses, experts, and documents to demonstrate that all four elements of the negligence claim exist. The EP then presents his version of the facts with the aim of refuting and contradicting the plaintiff's evidence. After presentations by both sides, the jury decides whether the plaintiff proved that the EP's act or failure to act met the four elements of negligence by a preponderance of the evidence. If the jury concludes that the EP was negligent, they establish the types and amount of compensatory damages to award the plaintiff. If the jury concludes there was no negligence, the claim is dismissed.

EPs can minimize their exposure to medical malpractice claims by practicing medicine consistent with the national standards of care. Because the medical record is the principal document used by parties to substantiate or repudiate the plaintiff's claim, the EP should complete the medical record with sufficient detail in a chronological manner. Furthermore, timely and courteous interaction between the EP and the patient and family is essential to ensure appropriate medical care, address potential conflicts, and clear up misunderstandings or misgivings. Likewise, patients expect honesty from the EP and do not tolerate being ignored. They insist that the EP keep them informed of the progress of their medical evaluation and treatment, and involve them in the decision-making process. Patients who believe that the EP acted professionally, genuinely cared about them, and did the best she could in dealing with their situation are less prone to file a malpractice claim, even when the outcome is suboptimal. Research has demonstrated that physicians who spend time with their patients, encourage a dialogue, seek their patient's opinions, discuss the patient's understanding of their medical condition and potential outcomes, keep their patients informed about the progress of their evaluation and treatment, and have an appropriate sense of humor are sued less often.

Conclusion

EPs are usually the first health care providers to treat patients with urgent and emergent traumatic, medical and psychiatric conditions. Furthermore, many patients utilize the ED for their primary care needs. In dealing with this variety of patients, EPs are continually faced with medical–legal issues. Therefore, EPs should have a broad appreciation of the potential issues that occur routinely in the ED, how to avoid or address them, and their responsibilities under state and federal law. EPs should investigate and understand specific legal obligations and responsibilities particular to the state in which they practice.

Pearls, pitfalls and myths

- EPs should assess all patients for decision-making capacity. Patients with decision-making capacity have the right to accept or refuse medical care.
- Informed consent is a formal process in which the EP and patient discuss recommended treatment or procedures, their risks and benefits, and potential alternatives.
- The emergency exception to the need to secure informed consent before rendering treatment exists if

the condition threatens the patient's life or limb and the patient is unable to understand the situation.

- Patients may be held against their will and treated in the ED if they lack decision-making capacity and require emergency treatment, or if they are a danger to themselves or others.
- Minors 16 years and older who meet certain criteria may be considered legally mature and capable of consenting for medical treatment. Most states have laws allowing minors to seek treatment for certain medical and psychiatric conditions without consent from their parents.
- Advance directives are legal documents created by a patient that delineate the medical care the patient desires or refuses if he or she lacks decision-making capacity. A surrogate decision maker is appointed to coordinate medical care.
- A patient's health information is protected by federal law and may only be used for treatment, payment, or health care operations. Any other sharing of patient's health information is considered disclosure and must be authorized by the patient.
- Individuals who "come to" the ED requesting or requiring medical care must be given an appropriate medical screening examination without regard to ability to pay for it, to determine whether an emergency medical condition exists.
- All emergency medical conditions must be stabilized as best as possible given the capabilities of the medical facility before an individual is transferred from the emergency department.
- Numerous Joint Commission regulations govern procedures, processes and patient safety in the emergency department.
- Appropriate medical record documentation is essential to coordinate and document proper patient care, substantiate medical fees, and resolve disputes or misunderstandings.
- EPs should understand their legal responsibilities and cooperate with law enforcement and other administrative agencies as required by local, state and federal law.
- Medical malpractice is a civil claim in which a patient asserts that they suffered an injury because the EP was negligent in evaluating or treating them. Most medical malpractice claims are not the result of negligent care. Instead, they are the product of a patient's dissatisfaction with their bill for medical care, or their interpersonal and professional interaction with the EP.
- Exposure to medical malpractice claims can be minimized by communicating clearly, effectively and respectfully with patients and their families, and by appropriate and timely creation of and documentation in the medical record.

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50 Patient safety in emergency medicine

Cherri D. Hobgood, MD

Scope of the problem

Errors in medicine are an issue of national concern. The Institute of Medicine's (IOM) report *To Err Is Human*, published in 2000, highlighted this concern and issued a warning that errors in medicine were much more common and costly than previously recognized. Following publication of this report, the medical community has developed a new focus on error, health care safety and quality. Much of what has been learned is the result of study and adaptation of systems from other complex organizations, such as the military and airline industry, as well as knowledge transfer from human factors engineering and organizational psychology. Human performance experts teach that *all* humans err. Therefore, errors are to be expected as a normal part of human behavior and function. Yet medical school and postgraduate training teach, and physicians ultimately swear an oath to the Hippocratic concept "above all, do no harm." This most noble of all aspirations has often been misconstrued to imply that physicians should practice error-free. Studies of human performance suggest that although we should always aspire to error-free performance, we must all come to the realization that medical errors are a regrettable but inevitable part of medical practice. Understanding medical error and being conversant in its nomenclature, processes and prevention is an important part of modern practice. The first step in this process is learning the taxonomy and language universally accepted and designed to enhance effective communication and mitigate blame. A set of definitions proposed by the IOM and adopted by the emergency medicine community is presented in Table 50.1.

In a professional setting, the causes of error are complex. Although human error may contribute to incidents, many errors may be beyond the control of the individual and occur as a function of human-system interaction. The high error rate of the emergency department (ED) has been largely ascribed to environmental features associated with the complexity inherent in delivering emergency care and the required variations in work needed to fit the dynamic fluxes occurring in such a versatile health care environment. Thus, practitioners who are aware of the inevitability of medical errors, possess knowledge of error types, and understand how systems are predisposed to error are better prepared to work safely within complex systems in many cases designed to fail.

Essentials of safety

To understand and improve patient safety, it is necessary to understand the type, frequency, severity and etiology of medical errors. What is known about the actual types and rates of error in the ED? Given that the ED has been described (along with the intensive care unit [ICU] and operating room [OR]) as one of the top three sites of hospital-based error, one would think these questions could be easily answered. Interestingly, a decade after the IOM report, error identification remains an elusive task in most clinical settings, particularly in the ED.

Table 50.1 Institute of Medicine's patient safety and adverse event nomenclature

Safety – The freedom from accidental injury
Patient Safety – Freedom from accidental injury; involves the establishment of operational systems and processes that minimize the possibility of error and maximizes the probability of intercepting errors when they occur
Accident – An event that damages a system and disrupts the ongoing or future output of the system
Error – The failure of a planned action to be completed as intended, or the use of a wrong plan to achieve an aim
Adverse event – An injury caused by medical management rather than by the underlying disease or condition of the patient
Preventable adverse events – Attributable to error
Negligent adverse events – A subset of adverse events that meet the legal criteria for negligence
Adverse medication event – An adverse event due to or caused by a medication or pharmacotherapy
Active Error – Errors that occur at the frontline and whose effects are felt immediately
Latent Error – Errors in design, organization, training, or maintenance that are often due to management or senior-level decisions. When expressed, these errors result in operator errors but may have been hidden, dormant in the system for lengthy periods of time prior to their appearance

One of the few prospective studies examining error in the ED was performed by Fordyce et al. This prospective, observational study conducted in western Massachusetts described approximately 18 errors per 100 ED patients along the entire continuum of emergency care. Fortunately, 98% of these errors resulted in no adverse outcomes to the patient. If these rates were extrapolated to all ED visits in the United States, this would represent more than 18 million errors and 360,000 adverse events annually.

Understanding these error types is an important next step. In this study, the authors categorized errors into six common areas of emergency care (Table 50.2). This categorization scheme reflects the complexity of systems existing in the ED environment. Review of this table suggests that error in the ED is ubiquitous and the result of many unique operating systems present in this complex environment.

Table 50.2 Categories of error

Category	Example of type of error
Diagnostic study	Mislabel specimens Wrong order ED organizational failure
Administrative procedure	Misidentification Wrong registration Lost paperwork
Pharmacotherapy	Wrong dose Wrong medication Improper order completion Incorrect prescription
Documentation	Incorrect documentation Documentation in wrong chart
Communication	Incomplete information transfer Miscommunication between staff Miscommunication between staff and patient Wrong person contacted
Environment	Incorrectly stocked equipment or supplies Unclean equipment Equipment malfunction Equipment incorrectly stored

Modified from Fordyce J, Blank FS, Pekow P, et al. Errors in a busy emergency department. *Ann Emerg Med* 2003;42(3):324–33.

The operating systems of the ED that predispose to error have been categorized as error-producing conditions (EPCs) and violation-producing behaviors (VPBs). The intrinsic properties of emergency medicine and the design of the system in which it is practiced yield EPCs. For example, high levels of diagnostic uncertainty associated with extremely high decision density and high cognitive clinician load result in an error-prone environment. Coupled with unknown, undifferentiated patients and short diagnostic windows, the ED environment's milieu becomes even more challenging. Add to this frequent interruptions, distractions, high patient volumes, overcrowded conditions, inadequate staffing and shift work fatigue, and the result is an environment in which errors are to be expected.

The other major sources of error are VPBs, which are individual characteristics rather than systems issues. Behavior in the workplace is a product of personality factors, willingness to take risks or avoid them, adaptation to adversity, rule following, confidence, authority gradients, institutional hierarchy and gender. Each of these interacts at both the individual and team level to create the human portion of the "work environment."

Both EPCs and VPBs result in errors that occur continuously in all EDs. As noted by Fordyce et al., very few of these result in patient harm. Most errors will be corrected as the work of patient care progresses; however, some of these errors will coalesce to form significant events resulting in patient harm. A visual representation of this is James Reason's Swiss cheese model (Figure 50.1), illustrating the defenses, barriers and safeguards against potential error. The holes in the cheese arise from *active failures* (mostly VPBs); when combined with fertile *latent conditions* (mostly EPCs), these "holes" may align. This allows an accident to navigate the system, resulting in a critical incident. Critical incidents that occur when the normal system of barriers and human recovery mechanisms fail are the gems of safety and performance improvement efforts. These must be addressed properly and carefully analyzed in a non-judgmental manner.

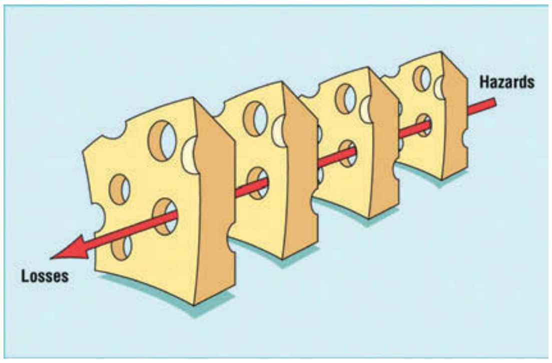

Figure 50.1
The Reason Swiss cheese model of error events: Lapses in patient safety culminate in error. Reproduced from Reason J, Human error: Models and management, *BMJ* 2000;320(7237):768–70. Used with permission, BMJ Publishing Group Ltd.

Defining error events

Errors that occur in the ED are not unique to this environment. Medicine has adapted a widely accepted cognitive performance and error classification scheme originated in the 1970s by cognitive psychologists Rasmussen and Jensen, since modified by Reason. This model elucidates three major types of cognitive performance: skill-based, rule-based and knowledge-based.

In *skill-based* cognitive performance, acts become automatic or habitual based on a previously acquired skill. Once the skill is acquired, these actions occur quickly with little conscious thought. One of the best examples of this is driving a car. The skill is learned; once acquired, one seldom thinks of the activities that comprise the "act of driving." Similarly, in clinical situations such as repairing a wound, the clinician thinks about how the wound will close, and makes decisions about the types of sutures to use and the types of knot to be tied, but does not think about the *act* of tying the knot. The act of tying the knot is therefore a skill-based cognitive performance. Once the decisions required for the process are made, the technical performance of the event occurs "automatically."

In *rule-based* cognitive performance, a guideline is used to direct the activity. In this type of performance, the context and content of the encounter are matched to a previously determined rule: "if X then Y." These rules may be based on a clinician's past experience, explicit instructions or protocols, clinical guidelines, or established criteria. The application of clinical decision rules (such as the Ottawa ankle rules) are excellent examples of this type of decision making. Other rules may be less formal and result from the experience of individual clinicians. For example, all patients with the chief complaint of chest pain should have an ECG performed; thus the internal rule is "chest pain = ECG assessment." Additional clinical decision rules and guidelines can be found in Appendix A.

The third type of cognitive performance is *knowledge-based*. When individuals are placed in new or unfamiliar situations without rules to guide behavior, they resort to knowledge-based cognitive performance. This is a conscious process, often referred to as analytic thought. In performance of this type, new data are interpreted in light of an existing knowledge set. Reasoning is goal-directed, incorporating data provided by the patient or the situation to knowledge acquired from all other sources. This type of clinical decision making is most commonly used in diagnostic reasoning (e.g., integrating the presenting symptoms, context, physical examination and diagnostic testing data to arrive at a diagnosis and treatment plan). As providers gain clinical experience, they are exposed to a wider array of presentations and problems. Consequently, their type of cognitive processing changes. Clinicians become less reliant on knowledge-based cognitive performance and spend greater amounts of time using rule- or skill-based cognitive performance strategies.

Table 50.3 Categories of diagnostic failures

No Fault – occur at any level in the system of care delivery.
Lack of complete information transfer from patient
Insufficient medical information available about a new disease
Patient noncompliance
Illness is indolent, silent or masked
Unusual presentation of illness
Inconsistent confusion or lack of clarity of symptoms by patient
System – occur at any level in the system of care delivery.
Technical
Insensitive, inaccurate, or inappropriately performed test
Equipment failure
Lack of correct equipment
Organizational
Absence of or inadequate policies
Lack of backup expertise
Inefficiencies in care delivery
Lack of training or supervision
Failure to coordinate care
Defective communication
Excessively stressful work environment
Cognitive – occur at the level of the diagnostician.
Inadequate knowledge
Incomplete data gathering or information processing
Faulty clinical reasoning

Adapted from Graber M, Gordon R, Franklin N. Reducing diagnostic errors in medicine: What's the goal? *Acad Med* 2002;77(10):981–92.

Differential diagnosis of error

Error classification

There are multiple ways to classify errors that occur in the ED. One such error classification system, derived from the model of cognitive performance, identifies skill-based errors (termed *slips* and *lapses*) or rule- and knowledge-based errors (*mistakes*). Slips often occur due to an interruption or disturbance that produces a breakdown in an action sequence. Memory failures that impair the implementation of a plan are known as lapses. During problem solving, a rule-based mistake occurs when the wrong rule is selected, applied, or applied incorrectly. Incomplete or incorrect knowledge, flawed analytic processing, or incorrect application of correct information results in knowledge-based mistakes.

Croskerry recently proposed a theme-based approach to error classification. He identified cognitive, procedural and affective errors as predominant error themes in the ED setting. *Cognitive errors* occur during decision making, or the thought-based sphere of clinical processes. This is in contradistinction to *procedural errors*, which occur during the performance of actions in the ED such as line placement or wound repair. *Affective errors* occur when emotions or the affective state of the provider disproportionately influences the clinical decision-making process. Another taxonomic system classifies errors of delayed or missed diagnoses. These are among the most significant ED errors because they are most likely to result in death or disability. These errors account for much of the litigation against emergency physicians. Three major types of diagnostic error include no-fault errors, system errors and cognitive errors (Table 50.3).

Error models

Traditionally, medicine has viewed error as the responsibility of the individual. According to this viewpoint, errors occur as a result of limited knowledge, inattention, or recklessness. Using this model, the individual is blamed or punished with retaliation or litigation. A *system-based* view of error posits that individuals are fallible, errors are inevitable, and errors should be expected as a normal part of human behavior. This system-based perspective has multiple advantages. First, it provides a useful approach to error prevention, because all parties have a shared mental model that anticipates error as a normal part of human cognition and performance. Individuals are not blamed for events, and specific changes and safeguards can be put into operation to prevent future errors. The overall result is improved systems operations that enhance organizational reliability.

Another method of classifying errors is to identify them as active or latent failures. *Active failures* occur at the provider–patient interface, and are perpetrated by individuals with direct patient contact. These span the gamut of error types, including cognitive, affective and procedural, or may be the result of provider-based VPBs. Systems errors that are not expressed directly lie "latent" within the system, awaiting expression during the right combination of human and system actions; these are known as latent failures. *Latent failures* are the result of conclusions and actions made by people not at the patient interface (e.g., managers, procedure writers, hospital administrators, architects). These design, strategic and management decisions are not in and of themselves mistakes, but can

Table 50.4 Active and latent failure

Active failures	Latent failures
Actions are immediate and have adverse consequences on direct patient care, typically through the actions of providers	Actions are delayed and effects are those that impact patient care indirectly through the actions of others
Cognitive Errors Types Slips Lapses Mistakes *Rule-based* *Knowledge-based* Violations Low morale Poor senior staff behavioral modeling Gradient of authority Overconfidence and under-confidence Risk-taking behaviors	Disproportionate workload, insufficient staff Knowledge, experience and training gaps Inadequate or absent supervision High-pressure environment Antiquated communication systems Poorly maintained work environment Culture gap between institutional mission and values Throughput pressure Overcrowded clinical environment Absent or insufficient feedback Poor design characteristics

Adapted from Reason J. Human error: Models and management. *BMJ* 2000;320(7237):768–70, and Vincent C, Taylor-Adams S, Stanhope N. Framework for analyzing risk and safety in clinical medicine. *BMJ* 1998;316(7138):1154–7.

create a fertile field for mistakes to occur. As a group, they represent weaknesses in the system and opportunities for failure. Examples of both active and latent failures are presented in Table 50.4.

Special patients

Just as systems can create a rich environment for errors to occur (i.e., EPCs), certain patient populations are associated with higher risk for error. When considering patients at risk for error, clinicians should consider any factor that might take the patient outside the norm. Such factors include intrinsic ones (e.g., patient age, gender, cultural barriers, social vulnerabilities), or those unique to the provider (e.g., bias).

Errors intrinsic to the patient include patients at the extremes of age, such as neonates and geriatric populations. These populations are particularly susceptible to medication errors. Pediatric patients experience a well-described medication error known as a "factor of 10" error. In these cases, medication doses are incorrectly ordered by the simple movement of a decimal place one point to the right, increasing the dose by a factor of 10. These errors are difficult for support personnel to identify because both doses may be accurate. An example of this is 1 mg morphine sulfate IV versus 10 mg morphine sulfate IV. Similarly, geriatric patients typically require downward dose adjustments because their capacity to metabolize medications may be impaired, and routine drug side effects may increase their risk for confusion and falls.

Patients with complicated medical conditions, compromised immune status, or medications initiated as a result of organ transplant are also vulnerable to errors. These patients may require isolation in the ED setting, and are at risk for medication interactions. Similar risks exist for patients with end-stage organ failure, such as renal disease. Patients with psychiatric illness, substance abuse, and/or homelessness are also at increased risk for error. They will often be unable to provide complete and accurate histories or take necessary medications regularly. Furthermore, these patients are more susceptible to our personal biases. For example, patients with known psychiatric illness or behavioral abnormalities are more likely to be assigned a psychiatric (functional) etiology for their presentation, without a careful search for a medical (organic) etiology. This may result in missed diagnoses, failure to stabilize the condition, or continued exacerbation of the current condition. Understanding risks of error in vulnerable patient populations is one mechanism to mitigate risk or identify errors early.

Error disclosure

The traditional culture of medicine has discouraged open disclosure of errors, and individuals have feared embarrassment, litigation and loss of prestige. These fears, although understandable within the context of medicine, directly conflict with a physician's primary duty to be truthful. Fortunately, our medical culture is changing. Growing evidence suggests that disclosure is a powerful action on the part of the physician that has broad impact not only on the doctor–patient relationship, but also on the system of practice. When providing error disclosure to a patient and/or their family, certain key components of information must be included in the discussion (Table 50.5). Multiple professional organizations and regulatory agencies have endorsed error disclosure and developed policies supporting both disclosure and error reporting. Despite this recent culture change regarding disclosure, understanding the barriers to its practice is important for systematic improvement in disclosure practices.

The literature has numerous studies demonstrating an overwhelming desire by patients and families for disclosure of any error events that occur during their clinical care. This desire for disclosure is even more pronounced

Table 50.5 Six components of successful error disclosure

Six KEY communication content items
1. Express that an error occurred
2. Describe the events resulting in the error
3. Explain the clinical implications of the error
4. Describe why the error occurred
5. Delineate how recurrences will be prevented
6. Apologize

(99%) among parents presenting with their children to a pediatric ED. This desire for disclosure by patients and families directly contrasts with provider behavior that frequently does not meet a 100% disclosure standard. Several barriers exist that limit provider disclosure, including lack of error recognition, uncertainty about what the patient needs or wants in such situations, minimization of error significance, and fear of losing personal authority, reputation, or status.

One of the most substantial barriers to effective disclosure is the fear of litigation. Almost all providers fear what a malpractice claim will mean to their lives and professional career. Disclosing errors to patients seems risky. Studies using both hypothetical and health center–based models demonstrate that litigation risk may be decreased with effective disclosure. For example, the Veterans Affairs (VA) Medical Center of Lexington, KY has a policy of aggressive disclosure. Although there were higher numbers of claims overall, including several settlements that likely would not have resulted in claims without such a disclosure policy, there was a much lower level of overall payments made compared with the similar VA hospital systems.

Legislative action has also attempted to encourage provider disclosure of errors. Currently, 29 states have statutes allowing expressions of sympathy to be used in courts as proof of liability. Five states have a mandatory disclosure law. North Carolina law states that "statements by health care providers apologizing for an adverse outcome in medical treatment, offers to undertake corrective or remedial treatment or actions, and gratuitous acts to assist affected persons shall not be admissible to prove negligence or culpable conduct by the health care provider."

Prevention of future error is one of the most positive effects of disclosure. For this to be effective, errors must be disclosed to the health care system, known as reporting. Although errors may not always harm patients or impact the system of care, reporting remains critically important in exposing system failures. As barriers to reporting disclosure include fear and enhanced exposure to litigation risk, reporting also includes the inconvenience of completing a report and a perceived lack of response by administrators. The federal government has created legislation that provides relief to providers who disclose and encourage reporting in a voluntary and confidential manner through the Patient Safety and Quality Improvement Act of 2005 (PSQIA). The goal of PSQIA is to prohibit reports of error disclosure from being used in civil and criminal legal cases.

Developing a culture of safety

Reducing incidents and adverse events is the goal of any safety program. Universal strategies for achieving this goal include prevention, recognition and mitigation. Error prevention results from systematic identification and eradication prior to their occurrence. Recognition is a primary goal of education regarding error, and is intended to make events more transparent and easier to recognize. With improved recognition, the opportunity for mitigation and cure is present before significant patient harm occurs. Mitigation has an emphasis on the ability of the system to recover from errors by decreasing their damage.

Over the past decade, reducing errors in the ED has taken on greater importance from both a research and administrative focus. There are three major approaches to error reduction: continuous quality improvement (CQI), clinical research, and feedback. Each of these is useful in certain situations, but all have significant limitations, including under-reporting of errors, variability in the definition of errors, and challenges of system change. It is important to note that a new innovation designed to reduce error in one area may have the unintended consequence of increasing error in another. For example, innovations such as computerized physician order entry (CPOE) may reduce errors due to drug interactions or illegible writing, yet may create hazards if the system is sluggish, produces excessive warnings, or detracts from patient care in other ways. This example highlights the need to study changes designed to improve patient care, because the changes themselves may have important consequences impacting safety.

Other important considerations for improving patient safety in the ED focus on a preoccupation with failure. Organizations that focus on various ways systems fail, and acknowledge that human fallibility can combine with systems events to produce failures, constantly plan ways to improve. In this quest for excellence, organizations must investigate more than the events immediately preceding a critical incident. Identification of EPCs, active and latent failures of systems, and VPBs allows an organization to break the cycle of error at its root and make meaningful systems improvements.

Conclusions

Errors are inevitable and will remain a constant in medicine. The ED provides a unique and challenging setting for clinical practice. It is an area of the hospital demonstrated to be error-prone. As emergency patient visits increase and the demands on the ED expand, there will be an increasing need to identify and reduce errors in this environment. Emergency medicine has taken a proactive approach to error through enhancing our reporting system, engaging in multi-disciplinary quality improvement, and error reduction strategies. Emergency medicine practitioners should openly and honestly acknowledge and disclose errors as a basic part of clinical practice that provides comfort to patients, reduces their risk of litigation, and assists in the prevention of future similar errors.

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51 Occupational exposures in the emergency department

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Scope of the problem

Exposure to blood, body fluids or respiratory secretions that may be contaminated with infectious agents is a common occupational hazard for health care workers (HCWs). HCWs in the emergency department (ED) or prehospital setting are at especially high risk for such exposures. In addition, non-emergency HCWs are frequently referred to EDs for immediate treatment of occupational exposures. Emergency physicians should be familiar with the prevention and management of blood, body fluid and respiratory exposures, both for their own protection and to ensure the successful management of exposed HCWs in the ED.

Anatomic essentials

Infectious pathogens may be present in patients, body fluids, contaminated equipment, or they may be airborne. *Direct inoculation* may occur when a microbe is transmitted by direct contact between a health care provider and an infected patient. *Indirect inoculation* may occur when a microbe is transmitted to a health care provider following contact with an inanimate object (e.g., needle). *Respiratory transmission* may occur when exhaled droplets or aerosols reach mucous membranes of a susceptible host.

History and physical examination

Following known or suspected exposure, a focused history and physical examination of the exposed person allows for risk assessment and initiation of an appropriate plan of care. Specific information regarding the approximate time and route of exposure should be documented. With sharps exposures, the type of instrument including needle size, whether a needle was hollow, whether it was used for arterial or venous puncture, whether visible blood was present on the instrument, and depth of penetration are all important factors in risk assessment. With regard to mucocutaneous exposures, integrity of skin or mucous membranes at the exposure site should be assessed. The approximate volume of exposure should also be determined. A detailed immunization history should be obtained from the exposed person.

A medical history from the source patient, if available, may be extremely useful to determine the risk factors for or known infection with bloodborne pathogens, such as human immunodeficiency virus (HIV), hepatitis B virus (HBV), or hepatitis C virus (HCV). If the source patient has known HIV, information regarding viral load and antiretroviral history should be obtained. Physical examination of source patients is of low yield in most circumstances, but findings that suggest immune suppression (e.g., oral thrush or Kaposi's sarcoma) might indicate HIV infection in those with unknown status.

Prevention of exposures

The Healthcare Infection Control Practices Advisory Committee at the Centers for Disease Control and Prevention (CDC) has developed guidelines for infection control in hospitals. Precautionary measures are divided into two categories: *standard precautions*, followed in the care of all patients, and *transmission-based precautions*, used for patients who are known or suspected to be infected or colonized with certain infectious agents. Examples of transmission-based precautions include contact precautions, droplet precautions, and airborne precautions (described below). The Occupational Safety and Health Administration (OSHA) mandates use of protective equipment by HCWs to prevent blood and body fluid exposures (Figure 51.1). Despite regulations and educational programs promoting use of universal precautions, compliance is poor in many hospital settings (including the ED), and HCWs remain at risk for occupational exposures.

Standard precautions

Standard precautions apply to all patients regardless of diagnosis or presumed infection status. Handwashing between patient contacts and following any contact with blood, bodily fluids, or other contaminated items is recommended whether or not gloves are worn. Simple hand hygiene has tremendous potential to reduce nosocomial infections, yet physicians often fail to adhere to it consistently. Clean gloves should be worn when touching mucous membranes, nonintact skin, blood, bodily fluids, or contaminated items. Masks, eye protection, face shields, and gowns are recommended during procedures and other activities likely to generate splashes or sprays of bodily substances. Three elements have recently been added to standard precautions primarily for patient protection: respiratory hygiene/cough etiquette, safe injection practices, and use of masks during spinal/epidural procedures. Cough etiquette involves education about covering the mouth and nose when coughing, proper disposal of tissues, use of surgical masks, appropriate hand

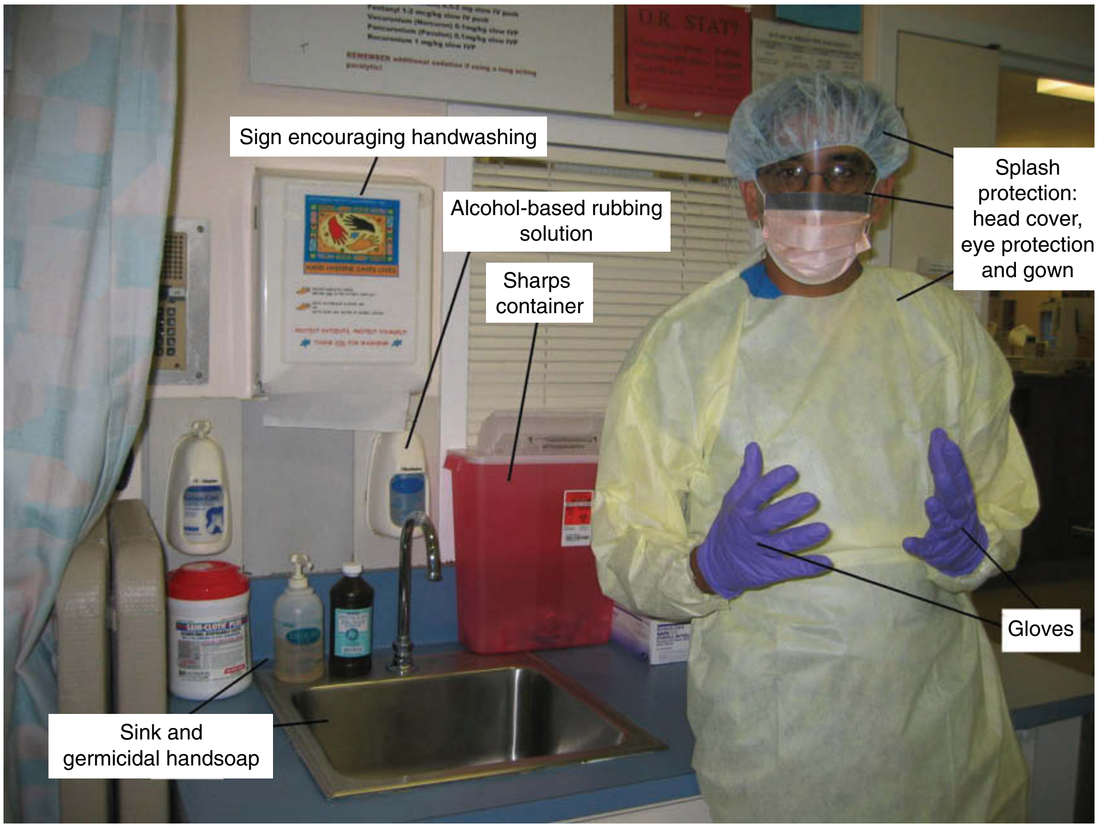
Sign encouraging handwashing
Alcohol-based rubbing solution
Sharps container
Splash protection: head cover, eye protection and gown
Gloves
Sink and germicidal handsoap

Figure 51.1
Health care worker with precautions in place. Courtesy: S.V. Mahadevan, MD.

hygiene following contact with respiratory secretions, and spatial separation (>3 feet) of persons with respiratory infections. Safe injection practices include use of a sterile, single-use, disposable needle and syringe for each injection, and prevention of contaminating injection equipment and medications in multi-dose vials. Additional recommendations regarding handling and cleaning of environmental surfaces, linens and patient care equipment, as well as sharps handling procedures and patient placement, are available from the CDC.

Contact precautions

Contact precautions are intended to prevent transmission of infectious agents that spread by direct or indirect contact with the patient or the patient's environment. In addition, contact precautions also apply when the presence of excessive wound drainage, fecal incontinence, or other discharge from the body suggests an increased potential for environmental contamination. A single patient room is recommended for patients requiring contact precautions. Gloves and gowns should be worn for all interactions that may involve contact with the patient or potentially contaminated areas of the patient's environment. All objects and surfaces within a contact isolation room should be considered contaminated. Gowns and gloves should be removed and discarded before exiting a contact isolation room, and hands should be washed or otherwise sanitized. Of note, alcohol-based hand sanitizer solutions are not effective against *Clostridium difficile*; hands should be washed with soap and water following contact with a patient known to have or suspected of carrying this organism.

Droplet precautions

Droplet precautions are intended to prevent transmission of pathogens that spread by contact of respiratory secretions with respiratory or mucous membranes. Certain infections (e.g., *Bordetella pertussis*, influenza virus, *Neisseria meningitis*, and group A streptococcus) are transmitted via respiratory droplets (5 μm) that can be projected approximately 3 feet upon coughing, sneezing, talking, or during procedures. Special air handling or ventilation is not necessary for these patients. However, they should be placed in a private room when possible, roomed with other patients with the same organism, or kept at least 3 feet from other patients if separate rooms are unavailable. A standard surgical mask should be worn while working within 3 feet of patients with known or suspected infections that are transmitted by droplets.

Airborne precautions

Airborne precautions are intended to prevent transmission of agents that remain infectious when suspended in aerosolized particles (generally <5 μm in size). Examples of pathogens requiring airborne precautions include tuberculosis (TB), varicella, measles, and (possibly) severe acute respiratory syndrome (SARS) corona virus. Patients requiring airborne precautions should be placed in an airborne infection isolation room (also known as a negative pressure room). When caring for patients placed in airborne precautions, a mask or respirator (depending on the disease-specific recommendations) should be worn at all times within the isolation room. N95 respiratory masks should be worn by anyone entering a room of a patient with known or suspected tuberculosis. Nonimmune HCWs should not care for patients with vaccine-preventable airborne diseases when providers with known immunity are available.

General principles of prophylaxis and treatment

Prevention remains the best method for avoiding occupationally acquired infections. Despite the use of standard precautions and safety devices, exposures to infectious blood, body fluids, respiratory droplets and aerosols continue to occur. Table 51.1 summarizes key characteristics of important pathogens that may pose a threat to HCWs. Table 51.2 summarizes steps for management of exposures to blood or body fluids. Following exposure to potentially infectious materials, exposed persons should receive counseling regarding risks, as well as appropriate testing and postexposure prophylaxis (PEP) when indicated.

Bloodborne pathogens

Human immunodeficiency virus

HIV can be transmitted via exposure to blood, tissue, semen, vaginal fluids, cerebrospinal fluid, synovial fluid, pericardial fluid, amniotic fluid, and any other body fluid that might contain blood. The average risk associated with occupational exposure to HIV through percutaneous injuries involving needles or other contaminated devices is approximately 0.3% (95% confidence interval [CI], 0.13% to 0.70%). Risk of HIV transmission following percutaneous exposure depends on the amount of blood to which the person is exposed and the amount of HIV in the source blood. Factors associated with greater blood exposure (e.g., deep injury, visible blood on the device, needles placed directly into an artery or vein) have been associated with increased likelihood of HIV transmission. With regard to the source, body fluids of terminal patients are thought to be more infectious, possibly reflecting higher viral loads. The risk of HIV transmission associated with mucous membrane exposures is estimated to be 0.09% (95% CI, 0.006 to 0.5), and transmission via human bite has been reported rarely. A study of 2,712 exposures to intact skin found no cases of HIV transmission; however, the CDC recommends that any exposure to concentrated virus (i.e., from a laboratory sample) be evaluated.

Numerous studies have provided evidence that use of antiretroviral drugs reduces the likelihood of HIV transmission. Because a very small proportion of occupational exposures result in HIV transmission, the potential benefit

Table 51.1 Summary of selected agents with potential for occupational exposure within the emergency department

Organism	Route of transmission	Recommended precautions	Vaccine available	Diagnostic testing	Postexposure prophylaxis
Clostridium difficile	Fecal/oral	Contact (hand sanitizer not effective)	No	Stool culture or rapid toxin assay	Not recommended
Corona virus (including SARS)	Respiratory droplet (possibly airborne)	Airborne	No	Not commercially available	No
Hepatitis B virus	Bloodborne	Standard	Yes	Serology for antibody or antigen	Yes (see text)
Hepatitis C virus	Bloodborne	Standard	No	Serology for antibody	No
HIV	Bloodborne	Standard	No	Serology for antibody or viral load	Yes (see text)
Influenza virus	Respiratory droplet	Droplet	Yes	Rapid influenza tests only moderately reliable	Yes (see text)
MRSA	Direct contact	Contact	No	Standard bacterial culture	Not recommended
Mycobacterium tuberculosis	Respiratory droplet Aerosol	Airborne	BCG vaccine not highly effective	Sputum for AFB smear and culture	Not recommended
Neisseria meningitidis	Respiratory droplet	Droplet	Yes	Culture and gram stain of blood or CSF	Yes (see text)
Varicella	Respiratory droplet Aerosol	Airborne	Yes	Viral culture or fluorescent antibody testing	Yes (see text)

AFB: acid-fast bacillus; BCG: bacillus Calmette-Guérin; CSF: cerebrospinal fluid; HIV: human immunodeficiency virus; MRSA: methicillin-resistant *Staphylococcus aureus*; SARS: severe acute respiratory syndrome.

Table 51.2 Outline for management of exposures to blood or body fluids

Expedite triage of exposed person.
Irrigate exposed areas with sterile saline solution or tap water.
Obtain history regarding exposure circumstances, source patient, and vaccination history of exposed.
Obtain baseline HIV, HBV and HCV serologies from exposed person, as well as CBC, serum creatinine, AST, ALT and bilirubin if antiretroviral drugs are being considered.
Obtain urine pregnancy test for women of childbearing potential.
If source patient is known, obtain serologies for HIV (rapid if available), HBV and HCV.
Determine need for tetanus immunization if there is a wound.
Determine need for HIV PEP (see Table 51.3).
Determine need for HBV PEP (see Table 51.5).
Counsel exposed person regarding risk of specific bloodborne pathogens; discuss risks/benefits of available treatment options.
Review dosing and side effects of recommended treatments.
Arrange follow-up in 3 days through employee health clinic or other resource.

ALT: alanine aminotransferase; AST: aspartate aminotransferase; CBC: complete blood count; HBV: hepatitis B virus; HCV: hepatitis C virus; HIV: human immunodeficiency virus; PEP: postexposure prophylaxis.

Adapted from Moran GJ. Emergency department management of blood and body fluid exposures. *Ann Emerg Med* 2000;35(1):47–62.

of HIV PEP must be balanced against the cost and toxicity of PEP drugs. Current United States Public Health Service guidelines for the management of PEP are summarized in Table 51.3. For exposures with very little or no risk for transmission, the marginal benefit of HIV PEP may not outweigh the risks. For most cases in which HIV PEP is given, a two-drug regimen is recommended. For the highest-risk exposures, the additional cost and toxicity of adding a third drug to the regimen appear justified. PEP is most likely to be effective if implemented as soon after the exposure as possible (preferably within hours of exposure). When prophylaxis is indicated, a 4-week course is recommended. Given the complexity of choosing and administering PEP, consultation with an infectious disease specialist is recommended (especially when there is concern for resistance because of prior antiretroviral use in the source patient). However, specialist consultation should not delay timely initiation of PEP. When uncertain, the first dose can be promptly given in the ED, with a more deliberate decision made regarding ongoing treatment. The toxicity of one dose of PEP drugs is minimal. The availability of rapid HIV tests allows more selective use of PEP. HIV PEP is not necessary if the source patient tests negative for HIV and is not at very high risk of recent infection (rapid tests detect antibody to HIV, so may not detect recent HIV infection). Current basic (two-drug) and expanded (three-drug) regimens are summarized in Table 51.4.

Follow-up should be arranged within 72 hours to review results of baseline serologies from the source and exposed patient, and to determine whether continuation of PEP is appropriate. Exposed HCWs should be advised of symptoms of PEP toxicity and potential drug interactions, and referred for laboratory monitoring based on the regimen prescribed. All patients exposed to HIV should receive counseling regarding symptoms of acute retroviral syndrome. Exposed individuals should use precautions to prevent secondary transmission via blood or tissue donation, pregnancy, breastfeeding, and unprotected intercourse until they are medically cleared with follow-up serologies at 6 weeks, 12 weeks and 6 months after exposure.

Hepatitis B virus

The risk of HBV transmission depends on the amount of virus in the source fluid, which in turn correlates with the presence of hepatitis B e antigen (HBeAg) in the blood. Among HCWs sustaining percutaneous exposure to hepatitis B, the risk of developing serologic evidence of infection was found to be 19% if the source was positive for HBeAg, but only 2.5% if the source was positive for anti-HBe. If Hepatitis B surface antigen (HBsAg) is not detected in blood from the source, transmission of hepatitis B is extremely unlikely. The risk of transmission of HBV from other body fluids appears to be less than from blood exposure, although transmission of the virus via human bites has been documented.

Fortunately, HBV transmission can be prevented with vaccination. All HCWs not known to be immune to HBV should receive a vaccine series consisting of three doses over a 6-month period. A Federal Standard issued under the OSHA mandates that hepatitis B vaccine be made available at the employer's expense to all health care personnel who may be occupationally exposed to blood or other infectious materials. All HCWs should have antibody titers tested 1–2 months after completing the hepatitis B vaccine series to verify immunity. Those who do not develop a protective titer should receive a second series. Those who do not respond after a second series should be counseled regarding susceptibility to HBV, and the need for hepatitis B immune globulin

Table 51.3 Recommended HIV postexposure prophylaxis following occupational exposure

	HIV status of body fluid source				
	HIV positive, Class 1[a]	HIV positive, Class 2[b]	Unknown status[c]	Unknown source	HIV negative
Percutaneous injuries					
Less severe[d]	Recommend 2-drug PEP	Recommend expanded ≥ 3-drug PEP	Generally, no PEP warranted; *consider* 2-drug PEP for source with HIV risk factors	Generally, no PEP warranted; *consider* 2-drug PEP if HIV exposure considered likely	No PEP warranted
More severe[e]	Recommend expanded 3-drug PEP	Recommend expanded ≥ 3-drug PEP	Generally, no PEP warranted; *consider* 2-drug PEP for source with HIV risk factors	Generally, no PEP warranted; *consider* 2-drug PEP if HIV exposure considered likely	No PEP warranted
Mucous membrane or nonintact skin					
Small volume	*Consider* 2-drug PEP	Recommend 2-drug PEP	Generally, no PEP warranted	Generally, no PEP warranted	No PEP warranted
Large volume	Recommend 2-drug PEP	Recommend expanded ≥ 3-drug PEP	Generally, no PEP warranted; *consider* 2-drug PEP for source with HIV risk factors	Generally, no PEP warranted; *consider* 2-drug PEP if HIV exposure considered likely	No PEP warranted

HIV: human immunodeficiency virus; PEP: postexposure prophylaxis.

[a] HIV positive, class 1: asymptomatic HIV infection or known low viral load (e.g., <1,500 ribonucleic acid copies per mL).

[b] HIV positive, class 2: symptomatic HIV infection, AIDS, acute seroconversion, or known high viral load.

[c] For sources with unknown status, rapid HIV test should be obtained to direct PEP. If rapid test is not available and source has high HIV risk, then PEP can be initiated and stopped if source is found to be HIV negative.

[d] Less severe percutaneous injuries include those involving a solid needle or superficial injury.

[e] More severe percutaneous injuries include those involving deep injury, large bore or hollow needles, needles used for arterial or venous puncture or objects with visible blood.

The recommendation to "consider PEP" indicates that PEP is optional and decision to initiate PEP should be made based on discussion between exposed person and treating clinician with regard to risks and benefits of prophylaxis.

Adapted from Centers for Disease Control and Prevention. Updated U.S. Public Health Service Guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. MMWR 2005;54(RR-9):1–17.

Table 51.4 Basic and expanded HIV postexposure prophylaxis regimens

Preferred basic two-drug regimens	
Zidovudine + lamivudine	Available as combination tablet (Combivir®) twice daily
Zidovudine + emtricitabine	Available as combination tablet (Truvada®) once daily
Preferred drugs to add for expanded regimens	
Lopinavir + ritonavir	Available as combination tablet (Kaletra®) twice daily
Atazanavir + ritonavir	Ritonavir added to boost level of atazanavir
Darunavir + ritonavir	Ritonavir added to boost level of darunavir

Dosages may require adjustment for renal disease.

(HBIG) for protection following HBV exposures. HCWs who test positive for anti-HepB antibody are considered immune and do not require further testing or treatment after exposure.

HBV infection can also be prevented by PEP with a combination of vaccination and/or HBIG for those who are not previously immune. Decisions regarding PEP for HBV are based on the infection status of the source and the vaccination status of the exposed. The source is tested for HBsAg and HBeAg to determine infectivity; if the exposed person is vaccinated but with unknown immune status, he is tested for anti-HBsAg. Exposed HCWs with pending test results should have follow-up within 72 hours. At that time, results of serologic studies from the exposed person and the source can be used to determine the potential need for treatment (it is acceptable to wait a few days before beginning PEP for HBV).

Recommendations regarding HBV PEP use are outlined in Table 51.5. HBIG and hepatitis B vaccine can be given simultaneously but at separate injection sites. Persons vaccinated after exposure should be tested for anti-HBsAg 1–2 months after completing the series. Exposed HCWs given appropriate prophylaxis are extremely unlikely to become infected with HBV.

Hepatitis C virus

HCV is a bloodborne pathogen that causes chronic liver disease in most of those infected, although only a fraction of those infected will develop complications such as cirrhosis or hepatocellular carcinoma. The risk of

Table 51.5 Guidelines for postexposure prophylaxis following exposure to HBV

	Source status		
Vaccination status and antibody response of exposed individual	**HBsAg positive**	**HBsAg negative**	**Source unknown or status unknown**
Unvaccinated	Initiate Hep B vaccine series, HBIG ×1	Initiate Hep B vaccine series	Initiate Hep B vaccine series
Previously vaccinated			
Known responder[a]	No treatment	No treatment	No treatment
Known non-responder[b]	HBIG ×2 or HBIG ×1 and initiate revaccination	No treatment	If high-risk source, treat is if source were HBsAg positive
Antibody response unknown	Test exposed person for anti-HBs: *if adequate:* no treatment. *if inadequate:* HBIG ×1 and vaccine booster	No treatment	Test exposed person for anti-HBs: *if adequate:* no treatment. *if inadequate:* administer vaccine booster, recheck titer in 1–2 months

Anti-HBs: antibody to hepatitis B surface antigen; HBsAg: hepatitis B surface antigen; HBIG: hepatitis B immune globulin; HBV: hepatitis B virus.

[a] Responder is defined as a person with adequate levels of serum antibody to hepatitis B surface antigen (i.e., anti-HBs ≥10 mIU/mL).

[b] Non-responders have anti-HBs <10 mIU/mL.

Adapted from Centers for Disease Control and Prevention. Guidelines for the management of occupational exposures to HBV, HCV, and HIV and recommendations for postexposure prophylaxis. *MMWR Morb Mortal Wkly Rep* 2001;50(RR-11):1–53.

HCV transmission following hollow-bore needle stick from an HCV-positive source is estimated at 1.8% (range 0–7%). Transmission of HCV via mucous membrane exposure occurs rarely, and transmission through intact skin has not been reported. No vaccine or prophylactic regimen has been demonstrated to prevent HCV transmission. The strategy for management of HCV exposure emphasizes identification and follow-up of those infected. Following potential exposure, baseline alanine aminotransferase (ALT) and anti-HCV titers should be obtained, with repeat testing in 4–6 months. People with percutaneous exposure to HCV-infected blood should be tested for HCV RNA at 4–6 weeks to allow early identification of infection. Early treatment of these individuals with an interferon regimen may prevent HCV-related complications. Exposed persons should receive counseling regarding prevention of secondary transmission until results of follow-up serologies are obtained.

Droplet and airborne pathogens

Tuberculosis

The ED is a high-risk area for TB transmission because patients with active pulmonary TB may not initially be recognized and placed in respiratory isolation. The key to preventing transmission of TB to HCWs is early recognition of patients who may have active TB and rapid respiratory isolation. Factors independently associated with pulmonary TB include history of TB, immigrant status, homelessness, history of incarceration, recent weight loss, chest radiograph with an apical infiltrate, and chest radiograph with cavitary lesion.

Triage protocols may facilitate the rapid isolation of patients at risk of TB. Patients with cough and epidemiologic risk factors for TB should immediately have a surgical mask placed, be isolated from others (if possible), and have an expedited chest radiograph. Those with a chest radiograph compatible with active TB should be placed in airborne precautions.

Antimicrobial prophylaxis is generally not warranted following exposure to a patient with TB. However, follow-up with a symptom screen and tuberculin skin test (or blood assay for *Mycobacterium tuberculosis* antibodies) should be arranged 8–10 weeks after exposure, and exposed persons should be counseled to seek medical attention immediately should they develop clinical evidence of active TB infection. Prior bacillus Calmette-Guérin (BCG) vaccination is not highly effective in preventing infection following TB exposure.

Meningococcal meningitis

Antimicrobial chemoprophylaxis is recommended for close contacts of patients with invasive disease caused by *Neisseria meningitidis*. The attack rate for household contacts with sporadic meningococcal disease has been estimated at 4 in 1,000. Health care providers directly exposed to oral secretions of an infected patient (those performing endotracheal intubation or managing endotracheal tubes) should receive PEP. In addition, emergency physicians may be called on to provide PEP to close contacts of source patients (household members, child-care center contacts, and anyone directly exposed to a patient's oral secretions via kissing, mouth-to-mouth resuscitation, or other activities). Anyone seated directly next to an index patient on a flight greater than 8 hours should receive PEP as well. PEP is thought to be most beneficial if administered as soon as possible after exposure has been identified (preferably within 24 hours); administration more than 14 days after exposure is probably of limited value. Exposed persons should receive a single dose of azithromycin, ciprofloxacin, or ceftriaxone; alternatively, rifampin may be given for 2 days. Ciprofloxacin should not be used in areas where fluoroquinolone-resistant *N. meningitidis* has been reported.

Varicella

Documentation of prior varicella infection, prior immunization, or laboratory evidence of immunity is considered evidence of immunity to varicella. Because of the prolonged incubation period, vaccination may be effective within 3–5 days of exposure in susceptible individuals. Acyclovir is not recommended for prophylaxis in otherwise healthy persons following varicella exposure. Administration of varicella zoster immune globulin (VZIG) is recommended following exposure in susceptible immunocompromised or pregnant individuals. Exposed susceptible HCWs should be temporarily relieved of patient care duties and monitored closely on days 10–21 following exposure, corresponding to the time period during which they might be contagious. Persons with varicella may be infectious 2 days prior to onset of rash. HCWs who develop skin lesions should not return to duty until their last lesions have crusted over. Recipients of VZIG should be monitored closely for evidence of varicella infection for 28 days, because VZIG has the potential to prolong the incubation period by 1 week.

Influenza

Influenza virus infection is most effectively prevented with annual vaccination for all health care personnel. Antiviral medications including oseltamivir, zanamivir, amantadine and rimantadine are adjuncts to vaccination; they may be effective when used for chemoprophylaxis following exposure to influenza virus, or may attenuate symptoms when administered as treatment. Resistance to one or more antiviral agents is periodically noted among circulating influenza strains. Current recommendations for treatment and prophylaxis from the CDC or World Health Organization (WHO) reflect resistance patterns in the most commonly circulating strains. HCWs are at increased risk when novel strains of influenza emerge to which there is less cross-immunity from previous infection or vaccination (as with the pandemic H1N1 strain in 2009). Basic infection control measures, such as masks on patients and HCWs and frequent handwashing, are critical to minimize the risk of influenza and other respiratory viruses. Regional and national recommendations regarding prevention of influenza transmission in the health care setting will continue to evolve over time.

Severe acute respiratory syndrome

SARS was first identified in Asia in 2003 and is caused by a coronavirus, now known as SARS-associated coronavirus (SARS-CoV). Because SARS may be transmitted via an airborne route, patients with known or suspected SARS should be placed in negative pressure isolation rooms, and N95 respirators should be worn by all HCWs entering the room. Because active transmission of SARS is typically limited to certain geographic areas during outbreaks, the necessity for special screening and isolation of patients depends on WHO recommendations at the time of outbreaks. HCWs exposed to SARS may require monitoring for illness and exclusion from duty; these decisions will be made by infection control personnel in consultation with public health authorities.

Special populations

Pregnant providers

Pregnant providers and women of reproductive age who may become pregnant should make efforts to limit or avoid contact with patients who may have communicable infections that would be harmful in pregnancy. These include varicella, influenza, cytomegalovirus and SARS-CoV. Recommendations for PEP are sometimes more aggressive for pregnant women (e.g., VZIG for varicella exposure), because pregnancy-associated changes in the immune system put pregnant women and the developing fetus at increased risk for severe illness.

Immunocompromised providers

Immunocompromised providers should take particular care to stay current with immunizations for which they are eligible. When possible, immunocompromised health care providers should avoid contact with patients harboring infections such as influenza, varicella, *N. meningitidis*, and active *Mycobacterium tuberculosis*, as infection with these pathogens poses increased risk for individuals with compromised immune systems.

Pearls, pitfalls and myths

Pitfalls

- Failure to stay up-to-date with the most current guidelines and recommendations for prevention and treatment of occupational exposures
- Failure to maintain current immunizations and regular screening (e.g., TB skin tests)
- Failure to strictly adhere to standard and special isolation precautions to prevent occupational exposures
- Failure to screen patients on arrival for symptoms and risk of respiratory infections (e.g., TB, influenza), exposing HCWs and other patients to risk of infection
- Failure to immediately and copiously irrigate skin and mucous membranes following exposures
- Failure to immediately report and seek treatment for any occupational exposure
- Failure to arrange appropriate and close follow-up after even the most minor occupational exposures

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Appendix A Clinical decision rules and guidelines

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Airway

Sedation scores

	ASA Levels of Sedation			
	Minimal sedation (anxiolysis)	**Moderate sedation/ analgesia (“conscious sedation”)**	**Deep sedation/ analgesia**	**General anesthesia**
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response after repeated or painful stimulation	Unarousable, even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

The American Society of Anesthesiologists (ASA) definition of moderate or conscious sedation: The goal is a decreased level of consciousness with purposeful response (more than just withdrawal to pain) to verbal command, either alone or accompanied by light tactile stimulation. No interventions are necessary to maintain a patent airway; ventilatory and cardiovascular functions are unaffected.

Reference

American Society of Anesthesiologists. ASA Standards, Guidelines and Statements, October 2007. Available at: https://ecommerce.asahq.org/p-365-asa-standards-guidelines-and-statements.aspx (accessed February 22, 2011).

ASA levels for risk stratification for sedation

The ASA developed a physical status classification system to risk-stratify patients receiving sedation for surgical procedures (see box).

Class 1 – A normal healthy patient
Class 2 – A patient with mild systemic disease
Class 3 – A patient with severe systemic disease
Class 4 – A patient with severe systemic disease that is a constant threat to life
Class 5 – A moribund patient who is not expected to survive with or without the operation
E – Emergency status: In addition to indicating underlying ASA status (1–5), any patient undergoing an emergency procedure is indicated by using the suffix “E.”

ASA class 3 or higher is proven to be an independent risk factor for adverse outcome in patients undergoing general anesthesia. Procedural sedation in the ED has been studied most extensively in patients who are ASA class 1 and 2. These patients are at low risk for peri- and post-procedural complications. Most clinicians would opt not to give sedation to a patient in ASA class 3 or greater, given a risk of morbidity or mortality.

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Assessment for difficult airway: LEMON, MOANS, RODS and SHORT

Difficult Intubation?		**Difficult Bag-Mask?**	
L	**Look:** obesity, micrognathia, head and neck surgery or irradiation, traumatic deformity, facial hair, poor dentition, dentures, large teeth, narrow face, high and arched palate, short, thick neck	**M**	Mask seal: facial hair or traumatic deformity
E	**Evaluate:** 3–3–2 rule (see Chapter 2, Airway Management)	**O**	Obesity, obstructive sleep apnea
M	**Mallampati:** limited visualization of uvula and soft palate (see Chapter 2, Airway Management)	**A**	Age >55 years
O	**Obstruction:** stridor, voice changes, drooling, dyspnea	**N**	No teeth
N	**Neck mobility:** degenerative or rheumatoid arthritis, presence of a cervical collar	**S**	Stiff lungs: asthma, chronic obstructive pulmonary disease, pulmonary edema
Difficult Combitube or LMA?		**Difficult Surgical Airway?**	
R	Restricted mouth opening	**S**	Past surgery, distorted anatomy
O	Obstruction	**H**	Hematoma
D	Distorted anatomy	**O**	Obesity
S	Stiff	**R**	Radiation
		T	Tumor

Assessment for signs of a difficult airway should be performed prior to initiation of any airway maneuvers. LEMON evaluates for potential problems with intubation. If any are present, the physician should then evaluate for potential problems with bag-mask ventilation (BMV), laryngeal mask airway (LMA) and surgical airway, using MOANS, RODS and SHORT.

Reference

Walls RM, Murphy MF, Luten RC. *Manual of Emergency Airway Management*, 3rd ed. Lippincott Williams & Wilkins, Philadelphia, PA, 2008.

CNS

Glasgow Coma Scale (GCS) = E + V + M

Adult			**Pediatric**	
Spontaneously	4	**Best Eye Opening**	Spontaneously	4
To verbal stimuli	3		To verbal stimuli	3
To painful stimuli	2		To painful stimuli	2
No eye opening	1		No eye opening	1
Oriented	5	**Best Verbal Response**	Appropriate coo & cries	5
Confused	4		Irritable cry	4
Inappropriate words	3		Inconsolable crying/screaming	3
Incomprehensible	2		Grunts	2
No verbal response	1		No verbal response	1
Obeys commands	6	**Best Motor Response**	Normal spontaneous	6
Localizes pain	5		Withdraws to touch	5
Withdraws to pain	4		Withdraws to pain	4
Flexion to pain	3		Flexion to pain	3
Extension to pain	2		Extension to pain	2
No motor response	1		No motor response	1

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ABCD2 Score for TIA

Age ≥60 years		1
Blood pressure ≥140/90 mmHg at initial evaluation		1
Clinical features of the TIA symptoms	Unilateral weakness Speech deficit only	2 1
Duration of symptoms	10–59 min ≥60 min	1 2
Diabetes history		1
TIA: transient ischemic attack		

The ABCD2 score predicts which patients with transient ischemic attack (TIA) are at high risk for stroke within 7 days. Risk of stroke was less than 1% in patients with a score less than 5, approximately 10% in patients with a score of 5, and approximately 30% in patients with a score of 6.

References

Johnston SC, Rothwell PM, Nguyen-Huynh MN, et al. Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack. *Lancet* 2007;**369**(9558):283–92.

Josephson SA, Sidney S, Pham TN, et al. Higher ABCD2 score predicts patients most likely to have true transient ischemic attack. *Stroke* 2008;**39**(11):3096–8.

NIH Stroke Scale

Level of Consciousness		alert	0	
		not alert	1	
		obtunded	2	
		unresponsive	3	
Questions		answers both correctly	0	
		answers one question correctly	1	
		answers neither question	2	
Commands		performs both tasks correctly	0	
		performs one task correctly	1	
		performs neither task correctly	2	
Gaze		normal	0	
		partial gaze palsy	1	
		total gaze palsy	2	
Visual Fields		no visual loss	0	
		partial hemianopsia	1	
		complete hemianopsia	2	
		bilateral hemianopsia	3	
Facial Palsy		normal	0	
		minor paralysis	1	
		partial paralysis	2	
		complete paralysis	3	
Motor Left Arm	0	no drift	0	**Motor Right Arm**
	1	drift before 10 seconds	1	
	2	falls before 10 seconds	2	
	3	no effort against gravity	3	
	4	no movement	4	
Motor Left Leg	0	no drift	0	**Motor Right Leg**
	1	drift before 5 seconds	1	
	2	falls before 5 seconds	2	
	3	no effort against gravity	3	
	4	no movement	4	
Ataxia		absent	0	
		one limb	1	
		two limbs	2	
Sensory		normal	0	
		mild-to-moderate loss	1	
		severe loss	2	
Language		normal	0	
		mild-to-moderate aphasia	1	
		severe aphasia	2	
		mute or global aphasia	3	
Dysarthria		normal	0	
		mild-to-moderate	1	
		severe	2	
Extinction/ Inattention		normal	0	
		mild	1	
		severe	2	

The level of stroke severity as measured by the National Institutes of Health (NIH) Stroke Scale scoring system:

0 = no stroke
1–4 = minor stroke
5–15 = moderate stroke
15–20 = moderate/severe stroke
21–42 = severe stroke

Reference

Brott T, Adams HP, Olinger CP, et al. Measurements of acute cerebral infarction: A clinical examination scale. *Stroke* 1989;**20**:864–70.

Modified Rankin Scale

SCORE	
0	No symptoms at all
1	No significant disability despite symptoms: able to carry out all usual duties and activities
2	Slight disability: unable to carry out all previous activities, but able to look after own affairs without assistance
3	Moderate disability: requiring some help, but able to walk without assistance
4	Moderately severe disability: unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability: bedridden, incontinent, requiring constant nursing care and attention
6	Dead

The Modified Rankin Scale measures the degree of disability after a stroke, and has become the most widely used clinical outcome measure for stroke clinical trials.

References

Rankin J. Cerebral vascular accidents in patients over the age of 60. II. Prognosis. *Scott Med J* 1957;**2**(5):200–15.

Wilson JL, Hareendran A, Hendry A, et al. Reliability of the Modified Rankin Scale across multiple raters: Benefits of a structured interview. *Stroke* 2005;**36**:777–81.

ENT

Modified Centor Score

Parameter	Points
Age	
3–14 years	1
>44 years	–1
Exudate or swollen tonsils	1
Tender/swollen anterior cervical lymph nodes	1
Fever >38°C	1
Absence of cough	1

The original Centor score provides an estimate for the probability of a group A beta-hemolytic streptococcal infection in patients with a sore throat. Patients with a score of 0 have approximately a 2% risk of bacterial infection; a score of 1 correlates with approximately a 7.5% risk; a score of 2 correlates with approximately a 15% risk; a score of 3 correlates with approximately a 30% risk; and a score of ≥4 correlates with approximately a 50% risk.

References

Centor RM, Witherspoon JM, Dalton HP, et al. The diagnosis of strep throat in adults in the emergency room. *Med Decis Making* 1981;**1**:239–46.

McIsaac WJ, Kellner JD, Aufricht P, et al. Empirical validation of guidelines for the management of pharyngitis in children and adults. *JAMA* 2004;**291**(13):1587–95.

Wessels MR. Streptococcal pharyngitis. *N Engl J Med* 2011;**364**(7):648–55.

Westley Croup Score

Clinical parameter		**Points**
Level of consciousness	Normal (including sleep)	0
	Disoriented	5
Cyanosis	None	0
	Cyanosis on agitation	4
	Cyanosis at rest	5
Stridor	None	0
	Stridor when agitated	1
	Stridor at rest	2
Air entry	Normal	0
	Decreased	1
	Markedly decreased	2
Retractions	None	0
	Mild	1
	Moderate	2
	Severe	3

Using the Westley scale, a score of <3 represents mild disease; a score of 3–6 represents moderate disease; and a score >6 represents severe disease.

Reference

Westley CR, Cotton EK, Brooks JG. Nebulized racemic epinephrine by IPPB for the treatment of croup. *Am J Dis Child* 1978;**132**:484–7.

Sinusitis Probability Criteria

Maxillary toothache
History of colored nasal discharge
No improvement with decongestants
Abnormal transillumination
Purulent secretions on examination

If one criterion is present, the risk of acute bacterial sinusitis is approximately 20%; if two are present, the risk is approximately 40%; if three are present, the risk is approximately 60%.

Reference

Williams JW Jr, Simel DL, Roberts L, Samsa GP. Clinical evaluation for sinusitis. Making the diagnosis by history and physical examination. *Ann Intern Med* 1992;**117**(9):705–10.

Cardiac

TIMI Risk Score for UA/NSTEMI

14-day event rate in patients with UA/NSTEMI	
Age ≥65 years	+1
Positive cardiac enzymes	+1
Known stenosis >50%	+1
ASA use in past 7 days	+1
>1 episode of angina past 24 hrs	+1
ST changes >0.5 mm	+1
>2 CAD risk factors (i.e., family history, hypertension, high cholesterol, diabetes or tobacco smoker)	+1

ASA: acetylsalicyclic acid; CAD: coronary artery disease; NSTEMI: non–ST-segment elevation myocardial infarction; UA: unstable angina.

The Thrombolysis in Myocardial Infarction (TIMI) risk score for unstable angina (UA) or non–ST-segment elevation myocardial infarction (NSTEMI) was developed to assist physicians in determining which patients would benefit from intensive care unit (ICU) admission. The event rate (death, infarct, or need for urgent revascularization) in patients with UA/NSTEMI was <5% if score was 0–1; approximately 10% if score was 2–3; approximately 20% if score 4–5; and >40% if score 6–7.

Reference

Antman EM, Cohen M, Bernink PJ, et al. The TIMI risk score for unstable angina/non-ST elevation MI: A method for prognostication and therapeutic decision making. *JAMA* 2000;**284**(7):835–42.

GRACE ACS Risk Score

Parameter		Points
Age (years)	40–49	18
	50–59	36
	60–69	55
	70–79	73
	80–89	91
	≥90	100
History of congestive heart failure		24
History of myocardial infarction		12
Resting heart rate	50–69	3
	70–89	9
	90–109	14
	110–149	23
	150–199	35
	>199	43
Systolic blood pressure	<80	24
	80–99	22
	100–119	18
	120–139	14
	140–159	10
	160–199	4
ST-segment depression		11
Initial creatinine (mg/dL)	<0.4	1
	0.4–0.79	3
	0.8–1.19	5
	1.2–1.59	7
	1.6–1.99	9
	2–3.99	15
	≥4	20
Elevated cardiac enzymes		15
Percutaneous transluminal coronary angioplasty *not* performed		14

A Global Registry of Acute Coronary Events (GRACE) score in patients with acute coronary syndrome (ACS) of >100 predicted 3% mortality at 6 months and 5% at 1 year; a score >150 predicted 15% mortality at 6 months and 20% mortality at 1 year.

Reference

Eagle KA, Lim MJ, Dabbous OH, et al. A validated prediction model for all forms of acute coronary syndrome: Estimating the risk of 6-month post-discharge death in an international registry. *JAMA* 2004;**291**:2727–33.

PURSUIT ACS Score

Parameter		Points
Age (years)	50	0
	60	2
	70	4
	80	6
Male gender		1
Angina at rest or with ordinary activity previous 6 weeks		2
Resting heart rate	80	0
	100	1
	120	2
Systolic blood pressure	120	0
	100	1
	80	2
Signs of heart failure (rales)		3
ST-segment depression initial ECG		3

ECG: electrocardiogram.

The PURSUIT score predicts 30-day outcomes in patients with UA or NSTEMI acute coronary syndromes. Risk of death or re-infarction within 30 days is associated with the following scores: score of <4 has <2% risk; score of 8 has approximately 5% risk; score of 12 has approximately 10% risk; score of 16 has approximately 25% risk.

Reference

Boersma E, Pieper KS, Steyerberg EW, et al. Predictors of outcome in patients with acute coronary syndromes without persistent ST-segment elevation. Results from an international trial of 9461 patients. The PURSUIT Investigators. *Circ* 2000;**101**:2557–67.

Killip Classification of CHF

Killip Classification of CHF after MI		30-day mortality
Class I	No clinical signs of heart failure	6%
Class II	Rales or crackles, gallop, elevated jugular venous pressure	17%
Class III	Frank acute pulmonary edema	38%
Class IV	Cardiogenic shock	81%

CHF: congestive heart failure; MI: myocardial infarction.

The Killip classification, which originally classified congestive heart failure (CHF) in post-myocardial infarction patients, has proved to be a powerful independent predictor of all-cause mortality in patients with non–ST-segment elevation acute coronary syndromes. Thirty-day mortality was <3% in ACS patients with a Killip class I, approximately 8% in Killip class II, and approximately 14% in Killip class III/IV.

References

Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit. *Am J Cardiol* 1967;**20**:457–64.

Khot UN, Jia G, Moliterno DJ, et al. Prognostic importance of physical examination for heart failure in non–ST-elevation acute coronary syndromes: The enduring value of Killip classification. *JAMA* 2003;**290**:2174–81.

Pneumonia

CURB-65

Parameter	Points
Confusion	1
Urea >7 mmol/L (Blood Urea Nitrogen >19 mg/dL)	1
Respiratory rate ≥30 breaths/min	1
Blood pressure <90 mmHg systolic or ≤60 mmHg diastolic	1
Age ≥**65** years	1

CURB-65 is a clinical prediction tool that has been validated for predicting mortality in community-acquired pneumonia and infection of any site. The score is an acronym for each of the risk factors measured. The risk of death is >10% with a score of 2; >15% with a score of 3; >40% with a score of 4; and >50% with a score of 5.

Reference

Lim WS, Lewis S, Macfarlane JT. Severity prediction rules in community acquired pneumonia: A validation study. *Thorax* 2000;**55**(3):219–23.

PORT (Patient Outcomes Research Team)

Step 1: In the absence of all of the following Risk Class I criteria, the patient may be treated as an outpatient with oral antibiotics

- >50 years of age
- Altered mental status
- Pulse ≥125/min
- Respiratory rate ≥30/min
- Systolic blood pressure <90 mm Hg
- Temperature <35°C or ≥40°C
- History of:
  - Neoplastic disease
  - Congestive heart failure
  - Cerebrovascular disease
  - Renal disease
  - Liver disease

If any are present proceed to ***Step 2***

Step 2: Stratify to inpatient care, Risk Class II – V

Demographics	**Points assigned**
If male	+ Age (years)
If female	+ Age (years) – 10
Nursing home resident	+10
Comorbidity	
Neoplastic disease	+30
Liver disease	+20
Congestive heart failure	+10
Cerebrovascular disease	+10
Renal disease	+10
Physical examination findings	
Altered mental status	+20
Respiratory rate >30/min	+20
Systolic blood pressure <90 mm Hg	+20
Temperature <35°C or ≥40°C	+15
Pulse ≥125/min	+10
Lab and radiographic findings	
Arterial pH <7.35	+30
Blood Urea Nitrogen ≥30 mg/dL (9 mmol/L)	+20
Sodium <130 mmol/L	+20
Glucose ≥250 mg/dL (14 mmol/L)	+10
Hematocrit <30%	+10
Partial pressure of arterial O_2 <60 mmHg	+10
Pleural effusion	+10
<70 = Risk Class II	
71–90 = Risk Class III	
91–130 = Risk Class IV	
>130 = Risk Class V	

The pneumonia severity index or PORT score calculates the probability of morbidity and mortality among adult patients with community-acquired pneumonia. The first part of the PORT score determines whether a patient is appropriate for treatment as an outpatient. The second part determines whether the patient should be admitted as a full inpatient or if the patient may be a candidate for 23-hour observation. A Risk Class II–III pneumonia patient may be sent home after IV antibiotics or treated and monitored for 24 hours in hospital. Patients with Risk Class IV–V pneumonia should be hospitalized for treatment.

Reference

Fine MJ, Auble TE, Yealy DM, et al. A prediction rule to identify low-risk patients with community-acquired pneumonia. *N Engl J Med* 1997;**336**:243–50.

Pulmonary embolism/ deep vein thrombosis

PERC (Pulmonary Embolism Rule-out Criteria)

Age <50 years
Heart rate <100
O_2 sat on room air >94%
No prior history of DVT/PE
No recent trauma or surgery
No hemoptysis
No exogenous estrogen
No clinical signs suggesting DVT
DVT: deep venous thrombosis; PE: pulmonary embolism.

If all of the above eight criteria are met, the patient has a <2% risk of PE. A further study showed that a negative PERC rule plus a negative D-dimer reduced the risk of PE to <1%.

References

Kline JA, Mitchell AM, Kabrhel C, et al. Clinical criteria to prevent unnecessary diagnostic testing in emergency department patients with suspected pulmonary embolism. *J Thromb Haemost* 2004;**2**:1247–55.

Kline JA, Runyon MS, Webb WB, et al. Prospective study of the diagnostic accuracy of the simplify D-dimer assay for pulmonary embolism in emergency department patients. *Chest* 2006;**129**(6):1417–23.

Wells Score for PE

Parameter	Points
Signs and symptoms of DVT	3
Alternative diagnosis less likely than PE	3
Heart rate >100 beats/min	1.5
Immobilization or surgery in past 4 weeks	1.5
History of DVT/PE	1.5
Hemoptysis	1
Malignancy within past 6 months	1
DVT: deep venous thrombosis; PE: pulmonary embolism.	

Risk of PE is low (approximately 2%) in patients with a score <2, moderate (approximately 15%) in patients with a score 2–6, and high (<30%) in patients with a score >6. The negative predictive value of a negative D-dimer combined with a low Wells score is 99.5%.

References

Wells PS, Ginsberg JS, Anderson DR, et al. Use of a clinical model for safe management of patients with suspected pulmonary embolism. *Ann Intern Med* 1998;**129**: 997–1005.

Wells PS, Anderson DR, Rodger M, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: Management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and D-dimer. *Ann Intern Med* 2001;**135**:98–107.

PESI (Pulmonary Embolism Severity Index)

Parameter		Points
Demographics	Age (years)	Age
	Male sex	10
Comorbid disease	Active or history of cancer	30
	Heart failure	10
	Chronic lung disease	10
Clinical findings	Pulse ≥100/min	20
	SBP <110 mmHg	30
	RR ≥30/min	20
	Temperature <36°C	20
	Altered mental status	60
	Arterial oxygen saturation <90%	20
SBP: systolic blood pressure; RR: respiratory rate.		

The PESI (age plus score of each criteria present) corresponds with the following risk classes: <66, class I; 66–85, class II; 86–105, class III; 106–125, class IV; and <125, class V. Patients in risk classes I and II are defined as low risk, and low-risk patients have a risk of PE-related mortality of <1%, with a negative predictive value of 99%.

Reference

Aujesky D, Perrier A, Roy PM, et al. Validation of a clinical prognostic model to identify low-risk patients with pulmonary embolism. *J Int Med* 2007;**261**(6):597–604.

Simplified Revised Geneva Score for PE

Parameter	Points
Age >65 years	1
Previous DVT or PE	1
Surgery or fracture (lower limb) within 1 month	1
Active malignant condition (or cured <1 year)	1
Unilateral lower limb pain	1
Hemoptysis	1
Heart rate:	
75–94 beats/min	1
95 or more beats/min	1
Pain on deep palpation of lower limb and unilateral edema	1
DVT: deep venous thrombosis; PE: pulmonary embolism.	

Patients with a low score (0–1) are considered unlikely (7%) to have a PE. The post-test probability of a PE in patients with a combination of a low simplified revised Geneva score and a normal D-dimer was found to be 1–2%, depending on the sensitivity of the D-dimer test used.

Reference

Klok FA, Mos IC, Nijkeuter M, et al. Simplification of the revised Geneva score for assessing clinical probability of pulmonary embolism. *Arch Intern Med* 2008;**168**(19):2131–6.

Wells Criteria: Simplified clinical model for assessment of DVT

Parameter	Points
Active cancer	1
Paralysis, paresis or recent plaster immobilization of lower extremities	1
Recent bedridden >3 days, or surgery within 4 weeks	1
Local tenderness along deep venous system	1
Entire leg swelling	1
Calf swelling 3 cm more than asymptomatic leg (measured 10 cm below tibial tuberosity)	1
Pitting edema qreater in symptomatic leg	1
Collateral superficial veins present (non-varicose)	1
Previous DVT	1
Alternative diagnosis at least as likely as DVT	–2

DVT: deep venous thrombosis.

Patients with a Simplified Wells score of zero were considered low risk (3%); a score of 1–2 was considered intermediate risk (approximately 20%). The combination of a Wells score <2 and a normal D-dimer had a 99.1% negative predictive value.

References

Wells PS, Anderson DR, Bormanis J, et al. Value of assessment of pretest probability of deep-vein thrombosis in clinical management. *Lancet* 1997;**350**(9094):1795–8.
Wells PS, Anderson DR, Rodger M, et al. Evaluation of D-dimer in the diagnosis of suspected deep-vein thrombosis. *N Engl J Med* 2003;**349**(13):1227–35.

Tuberculosis (TB)

Need for Isolation

Parameter	Points
TB risk factors: recent immigrant, institutionalization, history of exposure to TB, or 3 or more months of significant weight loss, malaise, weakness or night sweats	4
Positive PPD	5
Shortness of breath	–3
Crackles	–3
Upper lobe consolidation on chest X-ray	6
Temperature 38.5–39.0°C	3
Temperature >39.0°C	6

PPD: purified protein derivative; TB: tuberculosis.

Among adult patients with suspected respiratory TB, a score of 1 or higher indicates the need for respiratory isolation with 98% sensitivity.

Reference

Wisnivesky JP, Kaplan J, Henschke C, et al. Evaluation of clinical parameters to predict mycobacterium tuberculosis in inpatients. *Arch Intern Med* 2000;**160**(16):2471–6.

Gastrointestinal

Appendicitis

<table>
<tr><th colspan="2">MANTRELS Score - Adults</th><th colspan="2">Pediatric Appendicitis Score</th></tr>
<tr><td>1</td><td colspan="2">Migration of pain: umbilicus to right lower quadrant</td><td>1</td></tr>
<tr><td>1</td><td colspan="2">Anorexia</td><td>1</td></tr>
<tr><td>1</td><td colspan="2">Nausea or vomiting</td><td>1</td></tr>
<tr><td>2</td><td colspan="2">Tender right lower quadrant</td><td>2</td></tr>
<tr><td>1</td><td>Rebound tenderness</td><td>Cough/Percussion/ Hopping tenderness</td><td>2</td></tr>
<tr><td>1</td><td>Elevated temperature >37.3°C</td><td>Elevated temperature >38°C</td><td>1</td></tr>
<tr><td>2</td><td>Leukocytosis >10,000</td><td>Leukocytosis >10,000</td><td>1</td></tr>
<tr><td>1</td><td colspan="2">Shift of WBC: PMNs >7500 cells/mm^3</td><td>1</td></tr>
</table>

PMN: polymorphonuclear neutrophil; WBC: white blood cell.

Using the MANTRELS score in adults, the two most important factors (tenderness in the right lower quadrant and leukocytosis) are assigned two points; the six other factors are assigned one point each for a possible total score of 10 points. A score above 6 indicates a probable appendicitis.

A Pediatric Appendicitis Score (PAS) modified several of the MANTRELS criteria. Instead of rebound, the PAS uses cough/percussion/hopping tenderness, which earns two points, and the definition of elevated temperature differs (38°C, not 37.3°C). A leukocytosis in adults earns two points, but only one point in children. A PAS ≥6 indicates high risk for appendicitis.

References

Alvarado A. A practical score for the early diagnosis of acute appendicitis. *Ann Emerg Med* 1986;**15**:557–64.
Goldman RD. The Paediatric Appendicitis Score (PAS) was useful in children with acute abdominal pain. *Evid Based Med* 2009;**14**:26.

Ranson's Criteria for Pancreatitis Mortality

Parameter	Points
On admission:	
Age >55 years	1
WBC >16,000/mm^3	1
Glucose >200 mg/dL (>10 SI)	1
LDH >350 IU/L	1
AST >250 IU/L	1
48 hours after admission:	
HCT drop <10%	1
BUN increase >5 mg/dL (>1.79 SI)	1
Calcium <8 mg/dL (<2 SI)	1
Arterial $p0_2$ <60 mmHg	1
Base deficit (24 – HCO_3) >4 mEq/L	1
Fluid sequestration >6L	1

AST: aspartate aminotransferase; BUN: blood urea nitrogen; HCT: hematocrit; LDH: lactate dehydrogenase; WBC; white blood cell.

A score of <2 is associated with 1% mortality; 3–4, 16% mortality; 5–6, 40% mortality; and >7, 100% mortality. Some studies have found the APACHE II to be a better prognosticator than Ranson's criteria, but APACHE calculation is very cumbersome to use at the bedside.

References

Ranson JHC. Etiological and prognostic factors in human acute pancreatitis: A review. *Am J Gastroenterol* 1982;**77**:633–8.

Yeung Y, Lam B, Yip A. APACHE system is better than Ranson system in the prediction of severity of acute pancreatitis. *Hepatobiliary Pancreat Dis Int* 2006;**5**(2):294–9.

Trauma

Revised Trauma Score

Glasgow Coma Scale	Systolic blood pressure	Respiratory rate	Points
13–15	>89	10–29	4
9–12	76–89	>29	3
6–8	50–75	6–9	2
4–5	1–49	1–5	1
3	0	0	0

The Revised Trauma Score is a physiologic scoring system in which the initial Glasgow Coma Scale (GCS), systolic BP and respiratory rate correlates highly with mortality.

Reference

Champion HR, Sacco WJ, Copes WS, et al. A revision of the trauma score. *J Trauma* 1989;**29**(5):623–9.

Simplified Motor Score in Trauma

Parameter	Points
Obeys commands	2
Localizes pain	1
Withdrawal to pain or less	0

Derived from GCS, the Simplified Motor Score has been shown to be as accurate as the GCS in predicting brain injury outcomes.

References

Gill M, Windemuth R, Steele R, Green SM. A comparison of the Glasgow Coma Scale score to simplified alternative scores for the prediction of traumatic brain injury outcomes. *Ann Emerg Med* 2005;**45**(1):37–42.

Haukoos JS, Gill MR, Rabon RE, et al. Validation of the simplified motor score for the prediction of brain injury outcomes after trauma. *Ann Emerg Med* 2007;**50**(1):18–24.

Radiologic imaging criteria

Minor head injury: Adults

New Orleans Criteria	Canadian CT Head Rule	NEXUS 2 (BEAN BASH[a])
Headache	Mechanism of injury was a dangerous activity or event	**B**ehavior abnormal
Emesis	≥2 episodes of vomiting after the injury	**E**mesis intractable
Age >60 years	Patient >65 years	**A**ge ≥65years
Drug or alcohol intoxication	Glasgow Coma Score <15 at 3 hrs post injury	**N**eurologic deficit **B**leeding disorder
Convulsion (seizure)	Amnesia for events 30 min prior to injury	**A**ltered mental status
Trauma visible above clavicles	Possible open or depressed skull fracture	**S**kull fracture
Short-term memory deficits	Any sign of basal skull fracture	**H**ematoma scalp

[a]Acronym developed by S.V. Mahadevan, HEADCTS acronym for New Orleans criteria developed by M.J. Haydel

All three minor head injury guidelines have proven to be very sensitive (>98%) in identifying patients with intracranial injury. Each includes some form of vomiting, advanced age, altered mental status and signs of head trauma on physical examination. The New Orleans Criteria limits patients to those with a loss of consciousness, a GCS of 15, and a normal neurologic examination. The Canadian Rule includes patients with a GCS of 13–14. NEXUS 2 includes patients with a GCS 13–15 and neurologic deficits, with or without a loss of consciousness.

References

Haydel MJ, Preston CA, Mills TJ, et al. Indications for computed tomography in patients with minor head injury. *N Engl J Med* 2000;**343**(2):100–5.

Stiell IG, Wells GA, Vandenheen K, et al. The Canadian CT Head Rule for patients with minor head injury. *Lancet* 2001;**357**:1391–6.

Mower WR, Hoffman JR, Herbert M, et al. Developing a decision instrument to guide computed tomographic imaging of blunt head injury patients. *J Trauma* 2005;**59**(4):954–9.

Stein SC, Fabbri A, Servadei F, Glick HA. Critical comparison of clinical decision instruments for computed tomographic scanning in mild closed traumatic brain injury in adolescents and adults. *Ann Emerg Med* 2009;**53**(2):180–8.

Minor head injury: Children–PECARN Criteria (“SPLASH[a]”)

Age <2 years	Age ≥2 years
Severe mechanism of injury	**S**evere mechanism of injury
Parent says “Acting abnormal”	**P**uking
Loss of consciousness >5 seconds	**L**oss of consciousness
Altered mental status	**A**ltered mental status
Skull fracture (palpable)	**S**kull fracture (basilar)
Hematoma of scalp	**H**eadache severe

[a]Acronym developed by S.V. Mahadevan

The PECARN criteria (Pediatric Emergency Care Applied Research Network) identify children at very low risk for clinically important head injury, obviating the need for CT. The negative predictive value and sensitivity (respectively) of the PECARN criteria are 100% and 100% in children less than 2 years of age, and 99% and 97% in children 2 years and older if none of the criteria is present.

Reference

Kuppermann N, Holmes JF, Dayan PS, et al. and the Pediatric Emergency Care Applied Research Network (PECARN). Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. *Lancet* 2009;**374**(9696):1160–70.

Indications for plain radiographs of the cervical spine after blunt trauma

NEXUS criteria
Midline C-spine tenderness
Abnormal level of alertness
Focal neurologic deficits
Evidence of intoxication
Painful distracting injury

NEXUS: National Emergency X-Radiography Utilization Study

Canadian C-Spine Rule

1. Presence of high-risk factor that mandates radiography
   - A. Age ≥65 years
   - B. Dangerous mechanism
     - Fall from >3 feet/5 stairs
     - Axial load to head (e.g., diving)
     - High-speed MVC (>100 km/hr), rollover, ejection
     - Motorized recreational vehicles
     - Bicycle collision
   - C. Paresthesias in the extremities
2. Absence of any *one* low-risk factor that allows for safe assessment of range of motion
   - A. Simple rear-end MVC
     Excludes:
     - Being pushed into oncoming traffic
     - Hit by bus/large truck
     - Rollover
     - Hit by high-speed vehicle
   - B. Sitting position in the ED
   - C. Ambulatory at any time
   - D. Delayed onset of neck pain
   - E. Absence of midline c-spine tenderness
3. Inability to actively rotate the neck 45 degrees to the left and right

ED: emergency department; MVC: motor vehicle collision.

References

Hoffman JR, Mower WR, Wolfson AB, et al. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. *N Engl J Med* 2001;**343**:94–9.

Stiell IG, Wells GA, Vandemheen KL, et al. The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA* 2001;**286**(15):1841–8.

Indications for plain radiographs of the knee after acute injury

Ottawa Knee Rules	Pittsburgh Knee Rules
Tenderness of patella	Fall or blunt trauma
Tenderness at head of fibula	*plus*
Inability to flex 90 degrees	Inability to ambulate 4 steps
Inability to bear weight both immediately and in the ED for 4 steps (regardless of limping)	*or* Age <12 or >50 years
Age ≥65 years	

ED: emergency department.

Plain radiographs of the injured knee are indicated if any findings of the Ottawa or Pittsburgh knee rules are present.

References

Seaberg DC, Jackson R. Clinical decision rule for knee radiographs. *Am J Emerg Med* 1994;**12**:541–3.

Stiell IG, Greenberg GH, Wells GA, et al. Prospective validation of a decision rule for the use of radiography in acute knee injuries *JAMA* 1996;**275**:611–15.

Ottawa Ankle and Foot Rules

	Ankle x-ray indicated if there is complaint of pain in malleolar zone	Foot x-ray indicated if there is complaint of pain in midfoot
Inability to bear weight both immediately and in the ED (4 steps)		
Plus any of the following:		
Bone tenderness	Posterior edge or tip of lateral malleolus	Navicular bone
	Posterior edge or tip of medial malleolus	Base of 5th metatarsal

ED: emergency department.

Reference

Stiell IG, Greenberg GH, McKnight RD, et al. A study to develop clinical decision rules for the use of radiography in acute ankle injuries. *Ann Emerg Med* 1992;**21**:384–90.

Syncope

San Francisco Syncope Rule

C	History of **C**HF
H	**H**ematocrit <30%
E	Abnormal **ECG**
S	**S**hortness of breath
S	Initial **S**ystolic BP <90 mmHg

BP: blood pressure; CHF: congestive heart failure; ECG: electrocardiogram.

The presence of one or more of the criteria is associated with high risk for serious outcome, including death, myocardial infarction, dysrhythmia, pulmonary embolism, stroke, subarachnoid hemorrhage and significant hemorrhage. The mnemonic **FED 30–90**[a] (**F**ailure [cardiac], **E**CG changes, **D**yspnea, hematocrit <**30**% and systolic BP <**90**) helps remember the elements of the San Francisco Syncope Rule.

References

Quinn JV, Stiell IG, McDermott DA, et al. The San Francisco Syncope Rule vs physician judgment and decision making. *Am J Emerg Med* 2005;**23**(6):782–6.

Mattu A. Syncope. In Mahadevan SV, Garmel GM (eds). An Introduction to Clinical Emergency Medicine. Cambridge University Press, Cambridge, UK. 2005.

[a] FED 30–90 mnemonic developed by Gus M. Garmel, MD.

Fever

Modified Duke Criteria for infective endocarditis

Major criteria	Minor criteria
Blood culture growing typical IE microbes	IVDU or predisposing heart conditions: valvular diseases Fever >38°C
Echocardiographic findings: vegetations or abscesses	Vascular: major arterial emboli, septic pulmonary conjunctival hemorrhages and Janeway lesions Immunologic: glomerulonephritis, Osler nodes, Roth spots and rheumatoid fever Echocardiographic findings consistent with IE, but not meeting major criteria

IE: infective endocarditis; IVDU: intravenous drug use.

Clinical criteria for infective endocarditis: two major criteria, or one major and three minor criteria, or five minor criteria.

Reference

Durack DT, Lukes AS, Bright DK. New criteria for diagnosis of infective endocarditis: Utilization of specific echocardiographic findings. Duke Endocarditis Service. *Am J Med* 1994;**96**(3):200–9.

Rochester and Philadelphia Criteria for febrile infants

Rochester Criteria for febrile (>38°C) nontoxic, previously healthy, full-term infants 28–60 days old with no current antibiotic use or focus of infection:	**Philadelphia Criteria** for febrile (>38.2°C) well-appearing, previously healthy, full-term infants 29–60 days old with no current antibiotic use or focus of infection:
	Normal Chest X-ray and stool WBC (if obtained)
WBC count 5,000–15,000 with ≤1,500 band forms/μL	WBC count <15,000, with band:neutrophil ratio of <0.2
WBC count in spun urine sediment ≤10/hpf	WBC count in spun urine sediment <10/hpf
WBC count in stool ≤5/hpf, if diarrhea present	CSF WBC <8 and negative Gram stain

CSF: cerebrospinal fluid; hpf: high-power field; WBC: white blood cell.

The presence of all of the Rochester Criteria identifies febrile infants at low risk (<1%) for serious bacterial infection and who can be managed as outpatients if parents are reliable. The Philadelphia Criteria identifies febrile infants at low risk (<2%) for serious bacterial infection and who can be managed as outpatients without antibiotics if parents are reliable. In the post-pneumococcal immunization age, both sets of criteria were found to have maintained their previously reported negative predictive values.

References

Dagan R, Powell KR, Hall CB, Menegus MA. Identification of infants unlikely to have serious bacterial infection although hospitalized for suspected sepsis. *J Pediatr* 1985;**107**:855–60.

Baker MD, Bell LM, Avner JR. Outpatient management without antibiotics of fever in selected infants. *N Engl J Med* 1993;**329**:1437–41.

Garra G, Cunningham SJ, Crain EF. Re-appraisal of criteria used to predict serious bacterial illness in febrile infants less than 8 weeks of age. *Acad Emerg Med* 2005;**12**(10):921–5.

Kawasaki syndrome

Fever for at least 5 days: typically high-spiking with fevers ≥40°C (104°F), *plus*	
At least 4 of the following 5 signs	Bilateral conjunctival injection
	Oral mucosa: erythematous, dry and fissured lips; strawberry tongue; erythema of the pharynx
	Hands and feet: erythema and edema in the acute phase; desquamation in the subacute phase
	Rash: polymorphic truncal exanthem
	Cervical lymphadenopathy: node diameter ≥1.5 cm
No other explanation for disease process	

Kawasaki syndrome is an acute vasculitic illness of early childhood.

Reference

American Heart Association Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease. Diagnostic guidelines for Kawasaki disease. *Am J Dis Child* 1990;**144**:1220–29.

Morbidity and mortality scores

APGAR

	Score +2	Score +1	Score +0
Activity/tone	Active	Some extremity flexion	Limp
Pulse	>100 bpm	<100 bpm	Absent
Grimace	Sneeze/cough	Grimace	None
Appearance/color	All pink	Blue extremities, pink body	Blue/pale
Respirations	Good/crying	Irregular/slow	Absent

bpm: beats per minute.

The APGAR score is used to determine which neonates require medical intervention or resuscitation. Scores ≤3 are generally regarded as critically low, 4–6 fairly low, and 7–10 generally normal.

Reference

Apgar V. A proposal for a new method of evaluation of the newborn infant. *Curr Res Anesth Analg* 1953;**32**(4):260–7.

Mortality in Emergency Department Sepsis (MEDS) – adults

Parameter		Points
Terminal illness	Metastatic cancer or chronic illness with >50% likelihood of fatality within 30 days	6
Tachypnea/hypoxia	Respiratory rate >20 or pulse oximetry <90%	3
Septic shock	Sepsis plus a systolic blood pressure <90 mmHg despite a 20–30 mL/kg fluid bolus	3
Platelet count <150,000/mm^3		3
Bands >5%		3
Age >65 years		3
Lower respiratory tract infection	Based on clinical findings	2
Nursing home resident		2
Altered mental state	Glasgow coma score <15	2
Total possible score: 27		

The risk of death according to the MEDS score is very low (0–4), low (5–7), moderate (8–12), high (12–15), and very high (>15).

Reference

Shapiro NI, Wolfe RE, Moore RB, et al. Mortality in Emergency Department Sepsis (MEDS) score: A prospectively derived and validated clinical prediction rule. *Crit Care Med* 2003;31:670–5.

Modified Early Warning Score (MEWS)

Heart Rate	Respiratory Rate	Systolic BP	Temp (°C)	Alertness (AVPU)	Score
		<70			3
<40	<9	71–80	<35		2
41–50		80–100			1
51–100	9–14	101–199	35.0–38.4	**A**lert	0
101–110	15–20			Responds to **V**erbal	1
111–129	21–29	≥200	≥38.5	Responds to **P**ain	2
≥130	≥30			**U**nresponsive	3

A value is determined for each of 5 variables. A MEWS >5 is associated with an increased risk of death and indicates a need for ICU admission.

Reference

Burch, VC, Tarr G, Morroni C. Modified early warning score predicts the need for hospital admission and in-hospital mortality. *Emerg Med J* 2008;**25**:674–8.

APACHE-II

APACHE-II		Points
Chronic organ insufficiency or immune compromised?	Yes	5
	Yes + emergent post-op	5
	Yes + elective post-op	2
Creatinine (mg/dL)	With ARF	
	<0.6	4
	0.6–1.4	0
	1.5–1.9	4
	2.0–3.4	6
	>3.5	8
	Without ARF	
	<0.6	2
	0.6–1.4	0
	1.5–1.9	2
	2.0–3.4	3
	≥3.5	4
Age (years)	<44	0
	45–54	2
	55–64	3
	65–74	5
	≥75	6
Temperature (rectal)	≤29.9°C	4
	30.0–31.9°C	3
	32.0–33.9°C	2
	34.0–35.9°C	1
	36.0–38.4°C	0
	38.5–38.9°C	1
	39.0–40.9°C	3
	≥41°C	4
Mean arterial pressure (mmHg)	≤49	4
	50–69	2
	70–109	0
	110–129	2
	130–159	3
	≥160	4
Heart rate (beats/min)	≤39	4
	40–54	3
	55–69	2
	70–109	0
	110–139	2
	140–179	3
	≥180	4
Respiratory rate (breaths/min)	≤5	4
	6–9	2
	10–11	1
	12–24	0
	25–34	1
	35–49	3
	≥50	4
Sodium (mEq/L)	≥180	4
	161–179	3
	155–159	2
	150–154	1
	130–149	0
	120–129	2
	111–119	3
	≤110	4
Potassium (mEq/L)	≥7.0	4
	6.0–6.9	3
	5.5–5.9	1
	3.5–5.4	0
	3.0–3.4	1
	2.5–2.9	2
	<2.5	4
Hematocrit (%)	≥60	4
	50–59.9	2
	46–49.9	1
	30–45.9	0
	20–29.9	2
	<20	4
WBC count	≥40,000	4
	20,000–39,900	2
	15,000–19,900	1
	3,000–14,900	0
	1,000–2,900	2
	<1,000	4
GCS	score equals 15 – actual GCS	
PaO_2 (mmHg) [use if F_iO_2 <50%]	<55 (≥500)	4
	55–60 (350–499)	3
	(200–349)	2
A–a gradient [use if F_iO_2 >50%]	61–70	1
	>70 (<200)	0
Arterial pH	≥7.70	4
	7.60–7.69	3
	7.50–7.59	1
	7.33–7.49	0
	7.25–7.32	2
	7.15–7.24	3
	<7.15	4

A–a: Alveolar–arterial; ARF: acute renal failure; F_iO_2: Fraction inspired Oxygen; GCS: Glasgow coma scale; PaO_2: partial pressure of oxygen; WBC: white blood cell.

The Apache II score provides an estimate of ICU mortality using least favorable values from the initial 24 hours in the ICU. For each 5-point increase in Apache II score, there is a significant increase in mortality. Patients with a score of 0–4 have a <2% mortality rate, and patients with a score >40 have a mortality rate >80%.

Reference

Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: A severity of disease classification system. *Crit Care Med* 1985;**13**:818–29.

CPR futility in medical arrest: Indications to stop resuscitation in normothermic adults (all must be met)

Remain pulseless during 10 min of advanced cardiac life support (ACLS)
Initial rhythm not shockable
Unwitnessed arrest

It is acceptable to stop resuscitation efforts in the setting of an unwitnessed non-shockable arrest in patients who remain pulseless after 10 minutes of CPR.

Reference

van Walraven C, Forster AJ, Stiell IG. Derivation of a clinical decision rule for the discontinuation of in-hospital cardiac arrest resuscitations. *Arch Intern Med* 1999;**159**:129–34.

Appendix B Common emergency procedures

George Sternbach, MD

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Introduction

Performing procedures in the emergency department is often challenging, as such activity may take place under less than ideal conditions. Time may be a consideration because certain procedures are lifesaving or crucial to a patient's well-being. Circumstances may require that a procedure be performed without undue delay. The need to perform a procedure expeditiously should never lead one to rush the task, because because the potential for error is magnified by haste. Attention must also always be paid to proper preparation and technique. Some procedures are time-consuming; adequate time should be allotted for their completion.

Contraindications exist for most of the procedures in this section, and an effort should be made to elicit a patient history of illness or medications that could constitute a contraindication or warrant extra precaution. In the unconscious, intoxicated or uncooperative patient, such medical history may be incomplete or difficult to obtain.

Some of the procedures described must be performed utilizing sterile technique. Adherence to these procedures is very important because infection is an undesirable and sometimes dangerous complication. In all instances, assume that there is a risk of contracting infectious disease by contact with the patient's blood, secretions or other bodily fluids. Utilize universal precautions to avoid such contact, including gloves, eye protection, masks and surgical gowns, as appropriate.

Be aware of the complications of various procedures, and assess the patient for signs of their appearance. Not all complications manifest immediately, but some, such as pneumothorax consequent to central venipuncture, appear shortly after they are caused. In such instances, obtaining post-procedure diagnostic tests (e.g., a chest radiograph following subclavian or internal jugular cannulation) or instituting appropriate monitoring is imperative.

There are variations in the performance of some of the procedures described here, as well as "short cuts" known to experienced practitioners. Until you have mastered a particular procedure via standard technique, avoid the use of alternate methods. When performing a procedure as a member of a resuscitation team, focus on the task and do not be distracted by other management activities being carried out simultaneously. Be aware of the materials required for the procedure and assemble these beforehand, so you do not have to break sterile technique or interrupt performance to ask an assistant for additional items.

Reference

Rosen P, Chan TC, Vilke GM, et al. *Atlas of Emergency Procedures*. Mosby, St. Louis, MO, 2001.

Peripheral venous cannulation

Indications

The indications include the need for vascular access for the administration of intravenous (IV) fluids, blood products or medications. Even stable patients in whom such administration is anticipated are likely to benefit from having an IV line in place. For patients who require rapid volume resuscitation, short, large-bore peripheral IV catheters allow more rapid flow of fluids than do longer central venous catheters.

Contraindications

Whenever possible, avoid entry through skin that shows signs of infection or burns. Do not use veins that have previously been involved with phlebitis or thrombosis, or extremities affected with lymphatic insufficiency. Insertion of a peripheral venous catheter may be difficult in patients who have venous collapse (as a result of hypovolemia), obesity, edema or a history of IV drug abuse.

Equipment

- Gloves
- Povidone–iodine antiseptic solution

- Tourniquet
- Over-the-needle venous catheter (angiocatheter)
- IV tubing set and fluid bag
- Sterile dressing
- Arm board
- Tape

Technique

This procedure is described on the arm (Figure B.1), where it is most commonly performed, although the leg, scalp, or external jugular veins are sometimes utilized. Prepare the IV setup by attaching IV tubing to the solution bag and running solution to fill the tubing. Place a tourniquet around the upper arm and search for a prominent vein. These are usually found in the antecubital fossa or the dorsum of the hand. If no prominent veins are apparent, apply a warm towel to the skin to induce venodilation. Tapping a vein can also cause reflex dilation of the vascular wall. Having the patient open and close his fist several times will also distend the vein.

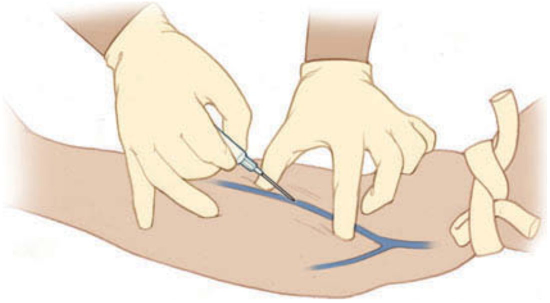

Figure B.1
Peripheral venous cannulation. © Chris Gralapp.

The tourniquet should be placed about 3–4 cm proximal to the puncture site. It should be tight enough to impede venous flow, but not so constricting as to curtail arterial circulation. Tie the tourniquet in a single loop in such a way that it can be released with one hand.

Although anesthesia for venipuncture is not usually necessary for adults, the procedure is painful for children. Topical application of a eutectic mixture of local aesthetics (EMLA) at the venipuncture site can be used, but a period of at least 30 minutes is needed between EMLA application and the onset of effect. Powdered lidocaine can also be delivered into the epidermis at the venipuncture site utilizing a pressurized needle-free system. This provides a local anesthetic effect within 3–5 minutes.

Prepare the puncture site in sterile fashion. With the non-dominant hand, pull the skin taut and stabilize the vein. Puncture the skin with the needle/catheter unit, entering at about a 30° angle to the skin. Keep the bevel of the needle facing upward. Puncture the vein. When the vein is entered, blood will appear in the flash chamber of the angiocatheter. Holding the unit steady, advance the catheter over the needle and into the vein. Remove the needle, holding the catheter in place.

Attach the IV tubing to the catheter. Remove the tourniquet and simultaneously initiate flow by opening the valve on the tubing. Apply a sterile dressing and tape the catheter in place. Taping the tubing to the skin in a U-shaped loop will reduce the likelihood of the catheter's accidental dislodgement. It may be advisable to affix an arm board if the cannulation site overlies a joint.

Fluid should flow freely into the vein. Immediate subcutaneous swelling around the catheter site indicates extravasation of fluid from the vein. If this occurs, stop the infusion, withdraw the catheter, apply pressure over the area, and attempt cannulation at a different site.

Ultrasound allows the clinician to visualize peripheral veins to aid in cannulation. This is a particularly useful technique in patients whose veins are difficult to identify, such as those who are obese or have extremity edema.

Complications

Local bleeding is a common complication. Hematomas are usually produced when the posterior venous wall is punctured during cannulation. Such bleeding is almost always minor and can be controlled readily by application of direct pressure.

Accidental puncture of the posterior wall or displacement of the catheter will result in subcutaneous extravasation of IV fluid. This produces swelling and pain. The condition must be recognized early, and the infusion discontinued. Subcutaneous infiltration of certain high-osmolality solutions (e.g., potassium chloride, sodium bicarbonate) is particularly toxic to soft tissue and may lead to tissue necrosis.

Phlebitis (inflammation of the vein) occurs frequently at IV sites utilized for several days or longer, but is not an immediate complication. Local subcutaneous infection is also a delayed complication; its incidence can be diminished by careful skin preparation and technique.

Reference

Sweeney MN. Vascular access in trauma: Options, risks, benefits and complications. *Anesthesiol Clin North Am* 1999;**17**:97–106.

Central venous cannulation

Indications

Catheterization of the central venous system may be performed for a number of reasons. One common reason is the inability to obtain peripheral venous access in a patient who requires urgent administration of IV fluid, blood products or medications. The need to infuse medications that are irritating to smaller peripheral veins also mandates cannulation of a central vein. Access to the central circulation is also necessary for measurement of central venous pressure, as well as for passage of a temporary transvenous pacemaker or pulmonary artery catheter.

Contraindications

There are no absolute contraindications to performing central vein cannulation. A relative contraindication is the presence of a coagulation disorder. This is particularly true in subclavian venipuncture. Relative contraindications include a markedly obese patient with poorly defined anatomical landmarks, an uncooperative patient or an overlying skin infection.

Equipment

- Sterile gloves
- Commercial central venous access kit, including:
  - Povidone–iodine antiseptic solution
  - Sterile drapes
  - Anesthetic solution, syringe and needles
  - Introducer needle
  - Metal guide wire
  - Catheter or sheath introducer
  - Semi-rigid dilator catheter
  - Gauze pads
  - Scalpel with No. 11 blade
  - 5-mL and 10-mL syringes
  - 3–0 or 4–0 non-absorbable suture
  - Antibiotic ointment
- IV solution and tubing

Technique

General

The Seldinger guide wire method (Figure B.2) is the recommended technique for insertion of a central venous catheter. This technique allows placement of a large-bore catheter over a wire inserted through a smaller bore needle. Identify the appropriate landmarks according to the vessel to be cannulated. Ultrasound should be used to identify the femoral and internal jugular veins to aid central venous cannulation.

Prepare the skin of the involved area with povidone–iodine solution. Surround the field with sterile drapes. Infiltrate the skin and underlying subcutaneous tissue to be entered with about 5 mL of 1% lidocaine. Using the external landmarks and ultrasound to identify the puncture site, locate the vessel with an introducer needle (an 18-gauge, 2.5-inch needle) attached to a syringe. Aspirate while advancing the needle until blood flows freely into the syringe. This indicates that the vessel has been entered. If no blood is returned, withdraw the needle to the skin edge and redirect.

Stabilizing the needle in place, remove the syringe and cover the hub of the needle with your thumb to prevent air from entering the vein. Pass the metal guide wire through the needle. The wire should pass smoothly through the needle into the vein. If you encounter resistance, withdraw the wire together with the needle and attempt the procedure again.

Once most of the wire been passed through the needle, withdraw the needle over the wire, leaving the wire in place. Be careful not to insert the entire length of the wire through the needle. Allow enough of the wire to protrude through the skin to allow passage of the catheter over it.

With a scalpel, make a small superficial incision of the skin at the point of entry of the guide wire. Pass the dilator over the wire and through the skin with a twisting motion. This creates a passage in the subcutaneous tissue that will allow easier admittance of the catheter. Remove the dilator, leaving the wire in place. Be careful not to remove or lose the wire inadvertently.

Pass the catheter over the wire in a manner similar to passing the dilator. The catheter should advance smoothly, requiring no force to pass. Remove the guide wire through the catheter and attach the IV tubing to the catheter. As you withdraw the guide wire, be sure the catheter is not removed inadvertently.

Suture the catheter to the skin, first injecting a wheal of anesthetic into the area into which the suture will be placed. Lower the IV bag below the level of the bed for a few seconds. Flow of blood into the tubing is an indication of intravascular placement. Apply topical antibiotic ointment and a sterile dressing to the venipuncture site. When using the internal jugular or subclavian approaches, obtain a chest radiograph to ascertain that the catheter is in proper position and that no pneumothorax has been produced.

Internal jugular vein

Position the bed with the patient's head down at an angle of 10–15 degrees (Trendelenburg position). Turn the patient's head away from the side of planned insertion. The right side is preferred because of the straighter course of the vein to the superior vena cava. In critically ill patients in whom simultaneous airway management is being performed, the subclavian or femoral approaches may be preferable.

There are several approaches to the internal jugular vein. A widely used one is entry in the triangle formed by the clavicle and the sternal and clavicular heads of the sternocleidomastoid muscle. The internal jugular vein runs lateral to the carotid artery in this triangle. Ultrasound allows direct visualization of the internal jugular vein and carotid artery; the vein is compressible, the artery is not. Anesthetize the skin and soft tissue in the apex of the triangle. Insert the introducer needle at the apex at an angle of 30–45 degrees to the skin and advance the needle toward the ipsilateral nipple (Figure B.3). Aspirate while advancing the needle. Brisk flow of blood into the syringe indicates entry into the internal jugular vein. Proceed with insertion of the guide wire and catheter using the Seldinger technique. Obtain a chest radiograph to verify correct catheter position and the presence of any complications.

Subclavian vein

Position the bed in the Trendelenburg position as described for internal jugular cannulation. Prepare the skin overlying the clavicle, sternum and neck to the angle of the mandible. A rolled towel placed between the patient's shoulder blades to accentuate the sternoclavicular joint is helpful. Anesthetize the skin and soft tissue overlying

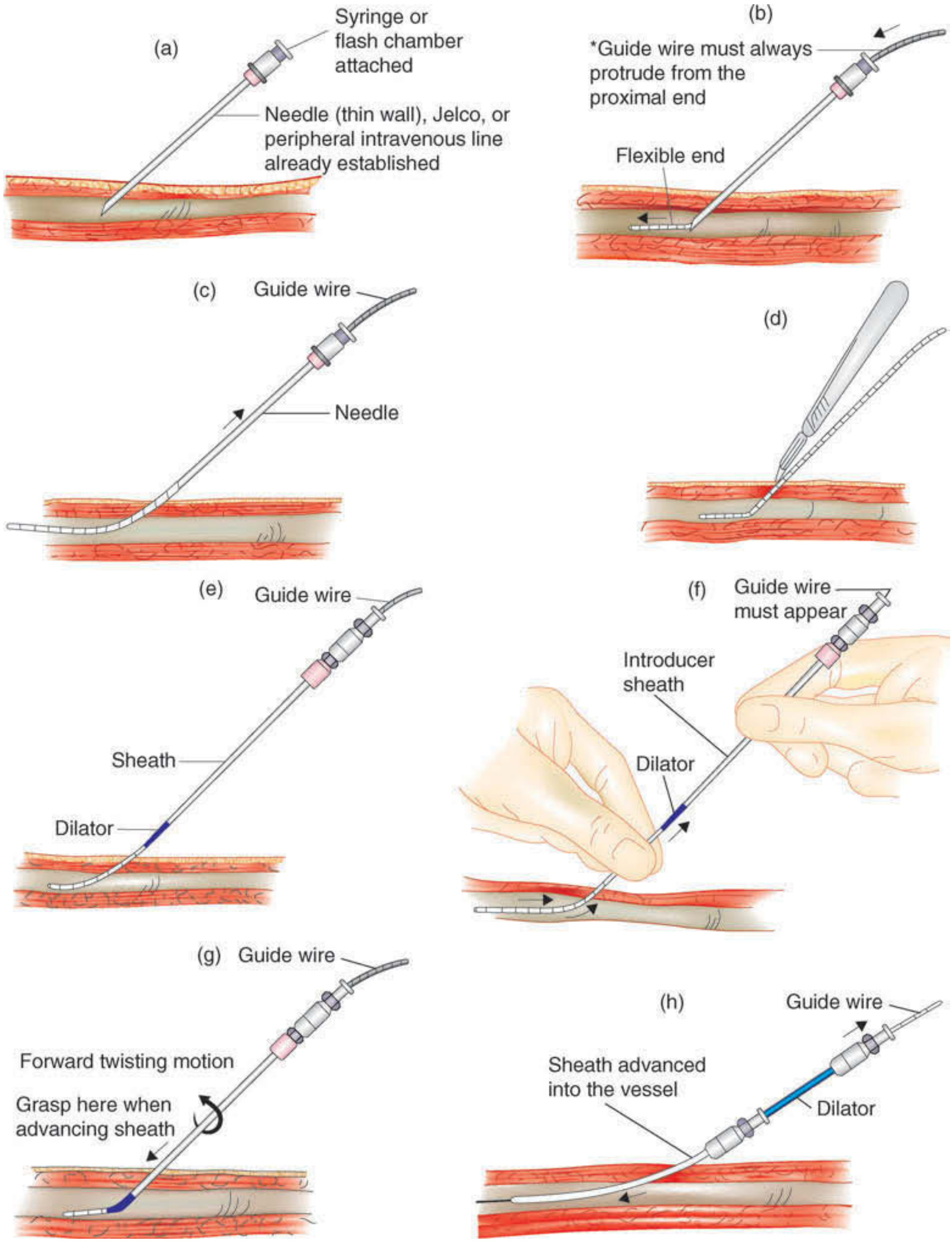
(a)
Syringe or flash chamber attached
Needle (thin wall), Jelco, or peripheral intravenous line already established
(b)
*Guide wire must always protrude from the proximal end
Flexible end
(c)
Guide wire
Needle
(d)
(e)
Guide wire
Sheath
Dilator
(f)
Guide wire must appear
Introducer sheath
Dilator
(g)
Guide wire
Forward twisting motion
Grasp here when advancing sheath
(h)
Guide wire
Sheath advanced into the vessel
Dilator

Figure B.2
Procedure for placement of Seldinger-type guidewire catheter. (a) The selected vessel is cannulated with a thin-walled needle, or an existing IV catheter is changed using the wire technique. (b) The guidewire is threaded through the needle, with the flexible end first, into the lumen of the vessel. (c) The needle is removed so that only the wire now exists from the vessel. (d) The skin entry site is enlarged with a No. 11 scalpel. (e) The catheter sheath and the dilator are threaded over the wire and advanced to the skin. The wire must be visible through the back of the device. (f) If the proximal wire is not visible, it is pulled from the skin through the catheter until it appears at the back of the catheter. (g) The sheath and the dilator are advanced as a unit into the skin with a twisting motion. It is best to grasp the unit at the junction of sheath and dilator to prevent bunching up of the sheath. The wire (at the back of the catheter) must be held while the sheath and dilator are advanced as a unit. (h) Once the sheath and the dilator are well within the vessel, the guidewire and the dilator are removed. Reprinted from Roberts JR, Hedges J (eds), *Clinical Procedures in Emergency Medicine*, 5th ed., Copyright 2010, with permission from Elsevier.

and just inferior to the junction of the lateral and middle thirds of the clavicle.

Insert the introducer needle 1 cm inferior to the clavicle, at a point at the junction of the lateral and middle thirds of the clavicle. Direct the needle medially and cephalad, aiming for the suprasternal notch (Figure B.4). Use a shallow angle to the skin and advance the needle just posterior to the clavicle. Once the vein is entered, blood should flow briskly into the syringe. Proceed with insertion of the guide wire and catheter. Obtain a chest radiograph to verify correct catheter position and detect the presence of any complications.

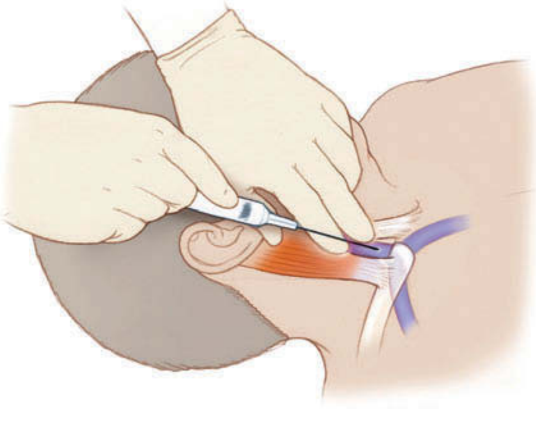

Figure B.3
Internal jugular vein cannulation. © Chris Gralapp.

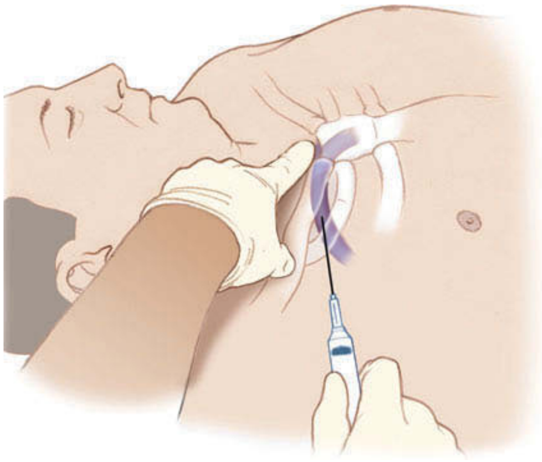

Figure B.4
Subclavian vein cannulation. © Chris Gralapp.

Femoral vein

Femoral vein cannulation has the advantage of being performed distant to the neck and thorax, and may therefore be more accessible in the patient undergoing simultaneous airway management or chest compressions. In addition, pneumothorax is not a concern as with the other two approaches. In cases of intra-abdominal injury, though, use of sites above the diaphragm is generally preferred to the femoral site.

Place the patient supine, with the hip slightly externally rotated. Feel the femoral arterial pulsation in the groin. The femoral vein lies just medial to the artery. When there is no palpable pulse, the artery can be expected to lie approximately at the midpoint of a line between the anterior–superior iliac crest and the pubic tubercle. Ultrasound is very useful for femoral vein identification, particularly when the femoral vein runs inferior to the artery; ultrasound also allows needle insertion under direct vision and minimizes the risk of accidental arterial puncture.

Prepare and drape the groin area in a sterile fashion. Anesthetize the skin and soft tissue in the area of the femoral pulsation. Insert the introducer needle approximately 2–3 cm distal to the inguinal crease and 1–2 cm medial to the femoral arterial pulsation (Figure B.5). The needle should be angled at 45 degrees to the thigh. Aspirate as you advance the needle. Venous blood should flow briskly into the syringe when the femoral vein is entered. Reduce the angle to 20 degrees and advance the needle an additional 2–3 mm to ensure that the entire bevel lies within the femoral vein. Proceed with insertion of the guide wire and catheter as described previously.

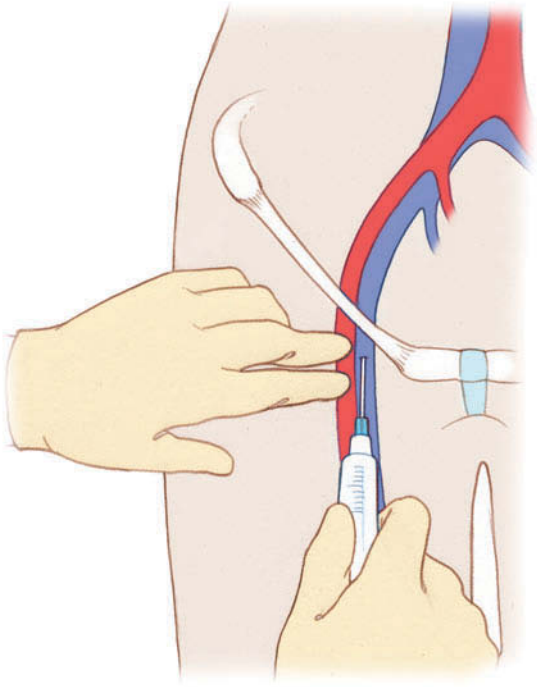

Figure B.5
Femoral vein cannulation with fingers on the pulse of the femoral artery. © Chris Gralapp.

Complications of central venous cannulation

Air embolism may occur if air enters the central circulation when the needle or catheter aperture is uncovered. The patient is particularly at risk when the syringe is removed for passage of the guide wire and when the catheter is attached to the IV apparatus. The risk of air embolism can be reduced by occluding the hub of the needle and catheter with the thumb at these times.

Catheter position should be checked by obtaining a chest radiograph immediately following the procedure. Malposition may be due to the catheter entering the wrong vein (e.g., the internal jugular vein rather than the superior vena cava in the subclavian technique), or knotting or kinking of the catheter.

Arterial puncture is probably the most common complication of central venous cannulation. This can be recognized by the appearance of bright red blood in the syringe and the presence of pulsatile flow. In puncture of the carotid artery during attempted internal jugular cannulation, a neck hematoma may develop that can cause tracheal compression. However, when either the femoral or internal carotid artery is inadvertently punctured, the area around these vessels can be readily compressed. This is not true of the subclavian vessels, and bleeding from the subclavian artery or vein can be particularly problematic, producing a hemothorax. If a subclavian catheter is inadvertently placed in the thoracic cavity, the fluid infused will extravasate into the thorax. Appropriate catheter location should always be assured prior to initiating infusion of IV fluids.

If an insufficient length of guide wire is maintained outside the skin puncture site, the wire can migrate into the vessel and be lost in the venous system. The guide wire can also break, especially if it is sheared against the needle when the needle is withdrawn. Insertion of the guide wire may irritate the right ventricle, especially with the internal jugular or subclavian vein approaches. This may provoke ventricular premature beats (VPBs). If VPBs appear on the cardiac monitor, the wire should be withdrawn a few centimeters until the ectopy ceases.

Pneumothorax is a hazard of the internal jugular and subclavian vein approaches. It is more likely to occur on the left side, because of the higher location of the left pleural dome. After performing the procedure, examine the patient for dyspnea, subcutaneous emphysema, tracheal shift or unilateral reduction of breath sounds. In addition, obtain a post-procedural chest radiograph. If an existing pneumothorax is known to be present, perform the procedure on the same side as this pneumothorax. Performing it on the opposite side places the patient at risk for bilateral pneumothoraces.

Infection is a possible complication of any venipuncture technique, but is more likely to lead to sepsis in central than peripheral venipuncture. Careful attention to sterile technique is therefore particularly important.

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Intraosseous infusion

Indications

Infusion of fluid or medications via an intraosseous (IO) line is usually reserved for acute life-threatening situations such as cardiac arrest, especially when IV access cannot be obtained. Fluid that is infused into the bone marrow enters the systemic circulation via nutrient and emissary veins. Crystalloid solutions, blood products and many drugs can be infused with nearly immediate absorption into the circulation. All drugs typically administered in cardiac arrest can be delivered via the IO route.

This procedure is most often performed in children under 5 years of age, though it may be used in adults as well. However, due to the nature of the mature marrow, infusion is not as effective in adults.

Equipment

- Sterile gloves and drapes
- Sandbag or rolled towel
- Povidone–iodine solution
- Lidocaine 1% local anesthetic, needles and syringe
- Bone marrow aspiration needle (16- or 18-gauge) with trocar (if not available, use a lumbar puncture needle with stylet)
- Tape and gauze pads
- Clear medicine cup
- IV solution bag with tubing
- Pressure infusion pump

Technique

The procedure can be performed at a number of sites (distal femur, distal tibia, medial malleolus, iliac crest and sternum in adults), but is described here at the proximal tibia, the site most often used. The insertion site is 1–2 cm distal to the tibial tuberosity on the medial tibial surface. Prepare the skin in a sterile fashion with povidone–iodine solution. Support the leg by placing a sandbag or rolled towel behind the knee. If time and clinical conditions permit, anesthetize the skin with lidocaine solution.

Stabilize the leg with your non-dominant hand while holding the needle/trocar in the other. Insert the needle into the skin nearly perpendicular to the tibia but angled slightly away from the knee (Figure B.6). A commercial Jamshidi or other bone marrow needle is preferred, but 18- or 20-gauge lumbar puncture needles may be used in children younger than 18 months.

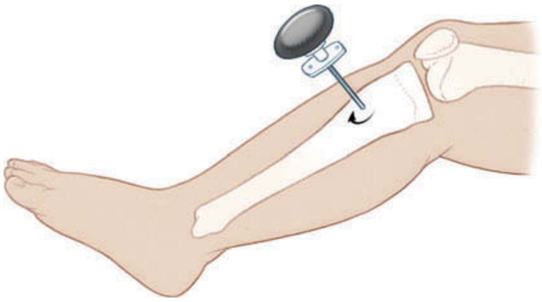

Figure B.6
Intraosseous cannulation of proximal tibia. © Chris Gralapp.

Use a rotatory motion with constant pressure to advance the needle into the bone. There is usually a sudden release of resistance when the needle enters the marrow cavity. When the needle is in the marrow, it should stand perpendicularly without support. Remove the trocar and confirm the position of the needle by aspirating a small amount of marrow, or by injecting a small volume of saline to determine that there is minimal resistance and no subcutaneous extravasation. Occasionally, marrow may not return with aspiration, but placement is correct if fluid infuses without extravasation.

Attach the IV solution apparatus. Re-examine the area for signs of extravasation. Place gauze pads around the needle, tape these down and apply a clear medicine cup to protect the needle. If necessary, apply a pressure pump to assist infusion.

Mechanical needle drivers and battery-powered drills have been developed to insert the intraosseous needle. Theses devices are safe and easy to use, but the operator should be trained prior to utilizing them.

Complications

There are few absolute contraindications to the procedure, but IO insertion should not be performed on patients with osteogenesis imperfecta, into fractured bone, or through infected skin. The most common complication is osteomyelitis, but this is rare, being described in less than 1% of most series.

Other complications include sepsis, cellulitis, subcutaneous abscess, subcutaneous or subperiosteal infusion of fluid, bone fracture and growth plate injury. The needle may be bent during insertion. There have been a few reported cases of compartment syndrome following IO infusion, but this appears to be a rare complication.

The likelihood of osteomyelitis can be reduced if the needle is left in place only long enough for resuscitation and stabilization, and by avoiding the procedure in bacteremic children. Subcutaneous infiltration (and the risk of subsequent compartment syndrome) can be minimized by careful observation of the infusion site. Infectious complications can be reduced by proper sterile technique. Adherence to bony landmarks avoids damage to the epiphyseal plate.

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Arterial puncture

Indications

Arterial blood samples can provide important information regarding respiratory and acid–base status, including arterial pH, pCO_2, pO_2 and bicarbonate levels. Such information is often sought in patients to assess respiratory and metabolic status, in those with significant respiratory compromise, and in others who are severely ill.

Contraindications

Although there are no absolute contraindications, arterial puncture should be performed with extreme care in patients with the following: bleeding disorders or anticoagulation, severe arterial disease in the area (as evidenced by diminished pulse or audible bruit), evidence of absent collateral flow in areas where it normally exists, and previous vascular surgery in the area. Do not perform arterial puncture through infected skin. When frequent blood sampling is anticipated, it may be preferable to insert an indwelling arterial catheter rather than performing repeated arterial punctures.

Equipment

- Prepackaged arterial blood gas kit including:
  - Antiseptic sponge or solution
  - Heparinized 5-mL syringe with 20- or 22-gauge needles
  - Syringe stopper
  - Gauze pads
- Syringe, and 25- or 27-gauge needles for anesthesia
- Local anesthetic solution

Technique

The blood sample is obtained with a 5-mL syringe, the barrel of which has been coated with heparin. If a prepackaged kit is used, the syringe already contains heparin. When preparing the syringe yourself, draw 2 mL of heparinized saline solution (1,000 IU/mL) into the syringe. Draw back the plunger to coat the barrel and needle, and then eject the remaining heparin.

Select an arterial puncture site. Common sites are the radial, brachial and femoral arteries. The radial artery at the wrist is the most commonly used location (Figure B.7),

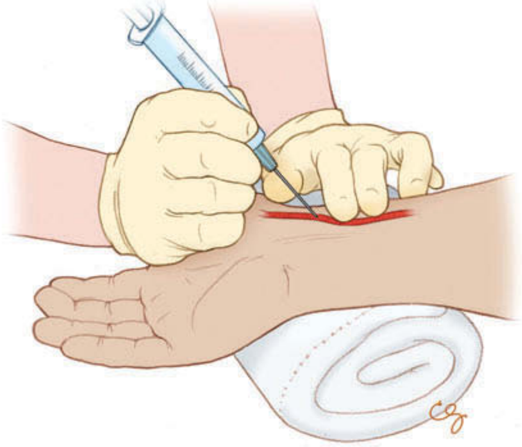

Figure B.7
Radial artery puncture at the wrist. © Chris Gralapp.

although the femoral artery is often preferred in patients in circulatory shock. For the radial artery, palpate the pulse at the wrist, placing the hand in approximately 60 degrees of dorsiflexion. Avoid hyperextending the wrist, as this may place excessive traction on the artery, making the pulse more difficult to feel. The brachial pulse can be felt on the flexor aspect of the elbow, just proximal to the antecubital fossa. The femoral artery enters the thigh after passing beneath the inguinal ligament; the pulse can be felt in the groin, midway between the anterior-superior iliac spine and the pubic symphysis.

When the radial artery is considered, the *Allen test* should be performed to ascertain the adequacy of collateral ulnar flow. Perform the Allen test as follows:

1. Palpate the radial and ulnar pulses at the wrist;
2. Compress both the arteries while having the patient repeatedly make a tight fist;
3. Instruct the patient to release the fist, and observe for blanching of the palm;
4. Release your compression of the ulnar artery, noting the time it takes for blanching to resolve. This should normally occur within 5–10 seconds.

When return of normal color to the palm is delayed, the adequacy of ulnar collateral flow is questionable, and radial artery puncture should not be performed. The Allen test requires a cooperative patient. Moreover, even a normal Allen test does not guarantee the adequacy of collateral circulation.

Prepare the skin overlying the puncture area with antiseptic solution. In an awake patient, the skin may be anesthetized by introducing a small volume of 1% lidocaine via a 25- or 27-gauge needle to make a small wheal. A large wheal may obscure the pulse.

Palpate the pulse with the index and middle fingers of the non-dominant hand. Puncture the skin over the artery between these two fingers. Advance the needle at approximately a 45-degree angle to the skin, parallel to the vessel. When the artery is entered, allow the syringe to fill with the force of arterial pressure. Obtain at least 3 mL of blood for analysis. If no blood is encountered or the blood does not readily advance the syringe's piston, withdraw the needle and redirect it.

Once blood sampling is completed, withdraw the needle and apply pressure over the puncture site for at least 5 minutes. If the patient has a coagulopathy or is on anticoagulation therapy, apply pressure for 10–15 minutes. Expel any air bubbles present in the sample through the needle holding it upright, then plug the needle or cap the syringe to maintain anaerobic conditions.

Complications

Hematoma formation is the most common complication. This can be avoided by conscientious application of pressure after the procedure is completed. In any event, such bleeding is usually minor.

Infection at the site is another potential complication. Serious infections, however, are uncommon. Although it has been postulated that the femoral site is at particular risk for infection because of proximity to the groin and perineum, no studies substantiate this.

Puncture may induce arterial spasm, which in turn can produce ischemia and thrombus formation. Such spasm usually causes transient ischemia, without significant sequelae in most cases.

Nerve or venous injury from the needle is a potential complication. The femoral vein and nerve lie immediately medial and lateral to the artery, respectively. The median nerve lies just to the ulnar side of the brachial artery at the antecubital crease.

Reference

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Nasogastric intubation

Indications

Passage of a nasogastric (NG) tube is performed for a variety of indications. An NG tube is commonly used to evacuate the stomach of air (gastric distention), gastric contents (intestinal obstruction, pancreatitis), blood (gastrointestinal hemorrhage) or ingested material (certain toxic ingestions). An NG tube should be placed to decompress the stomach of a trauma patient prior to performing a diagnostic peritoneal lavage. The tube may also be utilized as a conduit for administration of medication to the patient who is unable to swallow (e.g., the obtunded poisoned patient who needs activated charcoal or an antidote).

A 16- or 18-French tube is appropriate for most purposes in adults. A larger tube may be needed for evacuation of particulate material or blood clots, but it may be necessary to pass such a tube via the oral rather than the nasal route.

Levine and Salem sump tubes are most commonly used. The Levine tube has a single lumen at the tip and is adequate for instillation of material into the stomach or diagnostic aspiration. The Salem sump has a second vent lumen that attaches to a blue pigtail extension. This vent allows outside air to be drawn into the stomach, thereby permitting continuous flow through the tube. The Salem sump is preferred for continued suction or lavage.

Contraindications

The presence of injury to the mid-face, with possible fracture of the cribriform plate, constitutes an absolute contraindication to the passage of an NG tube. Attempting to insert an NG tube under these conditions could lead to passage of the tube into the cranium (Figure 7.20). In this setting, use an orogastric tube instead. Do not attempt passage of an NG tube in a comatose patient with an unprotected airway, as this risks aspiration. Coagulopathy and severe thrombocytopenia are relative contraindications, as significant nasal hemorrhage can be induced in these patients.

Equipment

- Gloves, gown and mask
- NG tube
- Topical anesthetic/vasoconstricting liquid
- Lubricant jelly
- Water and a flexible drinking straw
- 50-mL plain-tipped syringe
- Surgical tape
- 5-in-1 tapered adapter

Technique

Wear gloves, a gown and a face mask when performing the procedure. Position the patient in the sitting position when feasible, and elevate the back of the bed so that the patient does not withdraw his head during insertion. Flex the neck slightly.

Estimate the distance that the tube will have to traverse as follows: measure the distance from the tip of the nose to the earlobe and add the distance from the earlobe to the xiphoid process. The tube has graduated markings at various distances from the distal end. Make note of the distance you have estimated relative to these markings. This distance is approximately 50–60 cm in the typical adult. Underestimating the distance will result in failure to pass the tube past the gastroesophageal junction.

Check the nostrils for patency by inspection and by asking the patient to inhale while occluding each nostril in turn. When time permits, you may anesthetize and constrict the nostril by applying a topical liquid agent (e.g., 4% cocaine solution or neosynephrine spray) to the nasal mucous membrane. You may also spray a topical anesthetic (e.g., benzocaine spray) into the posterior oropharynx to diminish gagging. Lubricate the most patent naris by injecting 5–10 mL of water-soluble lubricant jelly or xylocaine jelly via a syringe. Coat the tip of the NG tube with this lubricant to a distance of about 6 cm from the tip. A common alternative is administering nebulized (or atomized) lidocaine to anesthetize the nares and oropharynx prior to insertion.

Insert the tip of the tube into the inferior portion of the nostril and aim it directly backward, perpendicular to the axis of the face (Figure B.8a). Slide the tube along the nasal floor beneath the inferior turbinate; do not direct it upward. Apply gentle pressure to advance the tube. Slight resistance may be encountered at the posterior nasopharynx. Have the patient sip some water through a straw to facilitate the tube's passage through the esophagus. If the tube twists or kinks in the mouth, withdraw it to the level of the nasopharynx to reattempt passage; it should not be removed entirely.

Once you have advanced the tube to the distance previously determined, confirm its appropriate position in the stomach. Rapidly insufflate 20–50 mL of air into the tube via a syringe, simultaneously listening with a stethoscope over the epigastrium (Figure B.8b). A rush of air will be audible as the air bolus enters the stomach. The egress of gastric contents through the tube is also an indication that the stomach has been entered.

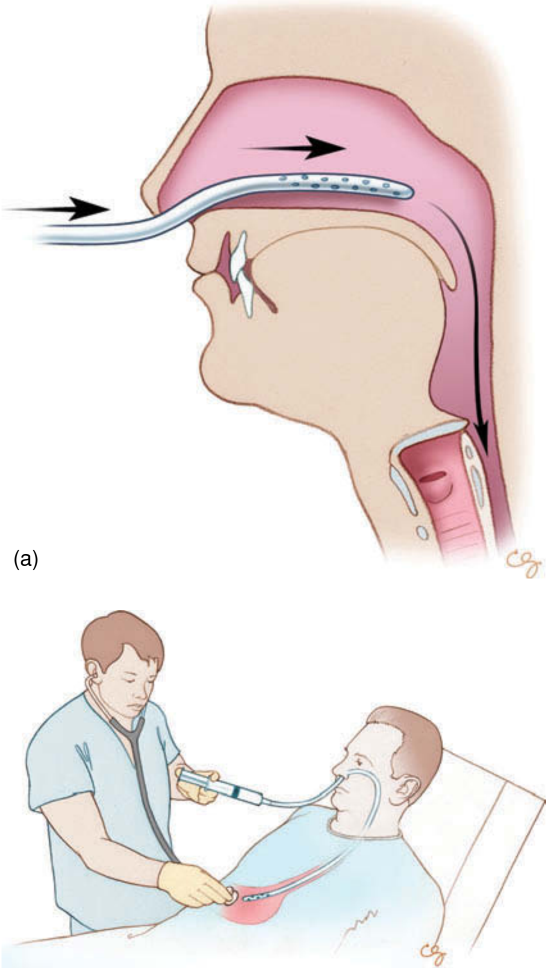

Figure B.8
Nasogastric intubation. © Chris Gralapp.

Once the tube's appropriate position has been ascertained, anchor the tube in place by wrapping adhesive tape around it and securing it to the nose. Apply a tapered 5-in-1 adapter and attach the tube to suction, if indicated.

Complications

An NG tube can be inadvertently passed though a fracture of the cribriform plate into the cranium. This is the reason that mid-facial trauma constitutes a contraindication to the procedure. The tube can also enter the trachea rather than the esophagus. This usually causes profound coughing and misting of the tube with respiration. When this occurs, withdraw the tube immediately and reattempt insertion.

Rather than advancing into the esophagus, the tube may coil in the oropharynx. This is a particularly common occurrence in the unconscious or uncooperative patient. The likelihood increases with smaller caliber tubes, and can be diminished by using a larger tube or by cooling the tube in ice water prior to insertion, thereby making it stiffer.

Injury to the nasopharynx is a relatively common complication, producing a small amount of bleeding; this is not cause for alarm. To avoid significant injury to the nasal mucosa, oropharynx or esophagus, only moderate pressure should be applied while advancing the tube. If nasal bleeding occurs, apply direct pressure to the nose until this stops.

Reference

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Bladder catheterization

Indications

Passage of a catheter into the bladder via the urethra may be necessary for a variety of reasons. Catheter placement may be required to relieve acute urinary retention due to mechanical obstruction or neurologic disease. Similarly, post-voiding residual urine volume is assessed by passage of a catheter in patients with incomplete bladder evacuation. An uncontaminated sample of urine for diagnostic analysis can be obtained via catheterization.

When diagnostic pelvic ultrasonography is performed, via the transabdominal approach, fluid may need to be instilled into the bladder via a catheter to provide an acoustic window for viewing pelvic contents. Prior to performance of diagnostic peritoneal lavage, it is recommended that (absent contraindications) the bladder be decompressed with a catheter to avoid inadvertent injury.

In some cases in which catheterization is performed for diagnostic urinalysis or urinary residual, a straight catheter can be inserted and promptly removed. In most other instances, a balloon-tipped (Foley) catheter is used, with the balloon inflated by injecting saline into the balloon port once the catheter is in the bladder and free flow of urine occurs. Foley catheters of 14-, 16- or 18-French sizes are most commonly used in adults. An indwelling urinary catheter is imperative for monitoring the urine output in seriously ill patients. Core body temperature can be assessed continuously with catheters equipped with temperature probes.

Contraindications

The most important contraindication is the presence of acute urethral injury. Signs suggesting a urethral injury include the presence of perineal hematoma, blood at the urethral meatus, or a high-riding prostate gland.

Equipment

- Commercially packaged catheter set of the following:
  - Povidone–iodine antiseptic solution
  - Cotton balls
  - Forceps
  - Lubricant jelly
  - Sterile drapes
  - Sterile gloves
  - Urinary catheter
  - 10-mL syringe
  - Sterile saline solution
  - Urine collection system (tubing and bag)
  - Surgical tape

Technique

The female patient

Place the patient in the lithotomy position, with the knees flexed and the hips flexed and abducted. Apply sterile gloves. Drape the perineum. Soak the cotton balls with antiseptic solution. Spread the patient's labia with the non-dominant hand, exposing the urethra. Grasping a cotton ball with forceps, prepare the periurethral area by applying povidone–iodine solution over the meatus in an anterior to posterior direction, once per cotton ball. Repeat several times with a new cotton ball.

Coat the tip of the catheter with lubricant. Gently introduce the catheter into the meatus and quickly advance it until about half its length has been inserted. Urine should flow through the catheter. Inflate the balloon with 10 mL of saline solution. Gently pull the catheter back until the balloon is snug against the bladder. Attach the urine collection tubing and bag. Secure the catheter to the leg with tape.

The male patient

Place the patient in the supine position. Apply sterile gloves. Drape around the penis. Soak the cotton balls with antiseptic solution. If the patient is uncircumcised, retract the foreskin. Grasp the penis with the non-dominant hand, holding it perpendicular to the perineum. Grasping a cotton ball with forceps, prepare the periurethral area by applying povidone–iodine solution over the meatus. Repeat several times, each time using a new cotton ball.

Coat the tip of the catheter with lubricant, and lubricate the urethra. Lidocaine jelly can be used to reduce the discomfort of catheterization. Introduce the catheter into the meatus and advance it until about half its length has been inserted (Figure B.9). Urine should flow through the catheter. Inflate the balloon with 10 mL of saline solution. Gently pull the catheter back until the balloon is snug against the bladder. Attach the urine collection tubing and bag. Secure the catheter to the leg with tape. It is important to return the foreskin to its proper position over the glans in an uncircumcised male after (or before) the catheter is secured.

Complications

Attempts to pass a catheter via a urethra that has been partially torn by a traumatic injury may result in complete urethral transection. For this reason, any clinical finding suggestive of urethral trauma is a contraindication to catheterization.

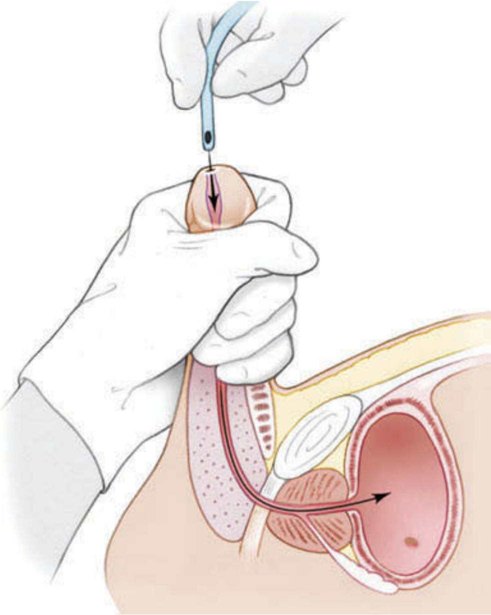

Figure B.9
Bladder catheterization. © Chris Gralapp

Microscopic and rarely gross hematuria may be produced by passage of a urinary catheter. Such bleeding is generally self-limited and requires no treatment. Urinary tract infection can result if sterile procedure is not followed.

Occasionally it is difficult to pass a catheter into the bladder. This is most commonly the case in a male patient with an enlarged prostate. Use of a J-tipped Coudé catheter or more advanced urologic techniques may be necessary in such cases.

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Lumbar puncture

Indications

Lumbar puncture (LP) is used in emergency medicine primarily as a diagnostic tool for meningo-encephalitis and subarachnoid hemorrhage. Cerebrospinal fluid (CSF) findings obtained through this procedure may also be useful in assessing a number of other neurologic diseases (e.g., multiple sclerosis, Guillain–Barré syndrome, neurosyphilis, or benign intracranial hypertension [pseudotumor cerebri]).

Indications for LP vary according to clinical setting. Although cranial computed tomography (CT) is extremely useful in the diagnosis of subarachnoid hemorrhage and various other conditions, there is no alternative diagnostic technique to LP for detecting meningitis. A strong suspicion of meningitis calls for confirmation or exclusion of the diagnosis by LP; accordingly, LP is frequently performed to exclude meningitis in infants with fever or following febrile convulsions. The indications for LP in these settings are not standardized and vary depending on the clinical presentation.

Contraindications

Lumbar puncture is contraindicated in the presence of skin or soft tissue infection overlying the puncture site because of the possibility of introducing infection into the CSF. Due to the risk of hemorrhage, the procedure is also relatively contraindicated in patients with severe bleeding diathesis, thrombocytopenia (platelet count <50,000/μL), and those who are anticoagulated. Spinal epidural or subdural hematomas are rare complications in such patients. If indications for LP are compelling, though, the procedure may be performed after efforts are made to reverse the coagulopathy.

A suspected intracranial mass (on the basis of clinical or CT findings) is a contraindication to performing an LP. Removing CSF in such cases may reduce lumbar pressure, causing a gradient that leads to rostrocaudal displacement of cerebral structures. Rapid neurologic deterioration may follow.

Equipment

Prepackaged LP kits are available that generally contain the following:

- 5-mL syringe with a 25-gauge needle
- Lidocaine 1% local anesthetic
- Spinal needle with stylet
- Four tubes for collection of CSF
- Povidone–iodine antiseptic solution
- Pressure manometer with three-way stopcock
- Sterile drapes
- Gauze pads
- Adhesive bandage

Technique

Proper positioning is essential for successful LP. The procedure is generally performed with the patient in the lateral position at the edge of the bed with the knees, hips and neck flexed, and the middle back arched outward. The shoulders and hips are positioned perpendicular to the bed. An assistant is usually needed to maintain the patient in this position. Avoid excessive flexion of the head, as this can lead to airway obstruction or impede the flow of CSF, especially in neonates.

If LP is unsuccessful in the lateral position, it may be attempted with the patient seated. This may be the best position for obese patients. Have the patient lean forward, with the arms resting on a table or Mayo stand. CSF pressure in the seated position, however, is gravity-dependent, so the measurement of opening and closing pressures is not clinically useful when the procedure is done in the sitting position.

Under sterile conditions, prepare the back with povidone–iodine solution and apply sterile drapes. Infiltrate the skin and soft tissue overlying the entry spot with 2–3 mL of 1% lidocaine. Entry may be through the L3–4 or L4–5 interspace; the L3–4 interspace can be located on a line connecting the posterior iliac crests. Use a styletted spinal needle of appropriate size, usually a 20- or 22-gauge needle 3½ inches in length in adults or 25-gauge needle 1½ inches in length in small children. Introduce the spinal needle through an anesthetic wheal in the midline of the back, midway between the spinous processes. Always ascertain the midline by palpation rather than inspection.

Advance the needle slowly with its bevel oriented horizontally and at a cephalad angle of 20–30 degrees (Figure B.10a). Direct the needle in the approximate direction of the umbilicus. If bony resistance is encountered, withdraw the needle and redirect it at a slightly different angle in the cephalo-caudad direction. A slight "pop" or "give" may be felt when the arachnoid space is entered (Figure B.10b). Once this occurs, advance the needle a few millimeters further to ensure that the entire bevel lies within the subarachnoid space. Remove the stylet and observe whether CSF flows from the needle. If it does not, rotate the needle 90 degrees to overcome a possible obstruction by a nerve root abutting against the aperture. If no fluid returns insert the stylet and, withdraw the needle almost to the skin edge before redirecting it. If the patient complains of a sharp pain radiating to the leg, the needle may have struck one of the roots of the cauda equina. If this occurs, withdraw and redirect the needle.

If blood returns through the needle and does not clear, remove the needle and repeat the procedure at another interspace. If the initial fluid is blood-tinged but subsequently clears, this likely represents a traumatic tap, with the origin of blood being the peridural venous plexus. This is not generally a dangerous complication, and no treatment is required.

When CSF appears in the hub of the needle, attach a three-way stopcock and manometer, and measure the opening pressure. Straighten the patient's legs and advise him to relax when this is being done. Straining will increase intra-abdominal pressure, which in turn will spuriously elevate intracranial pressure. The fluid level in the manometer should fluctuate slightly with respirations. Normal CSF opening pressure is 80–180 mmHg. If the opening pressure is extremely high (>350 mmHg), additional fluid should not be withdrawn, unless that was the purpose of the procedure. Remove the needle, and use the fluid already in the manometer for analysis.

In all other cases, allow fluid to drip sequentially into four tubes (1–2 mL in each tube). Specific analytic tests performed on the CSF depend on the clinical condition/s being considered. However, analysis for cell count, glucose, protein, Gram stain, bacterial culture and xanthochromia should be done in all cases. *Xanthochromia*, a reddish or yellowish discoloration of CSF due to red cell lysis, is present in most cases of subarachnoid hemorrhage (Figure 30.6b). Fluid in the first and fourth tubes should be used for cell count, the second for culture, and the third for glucose and protein levels.

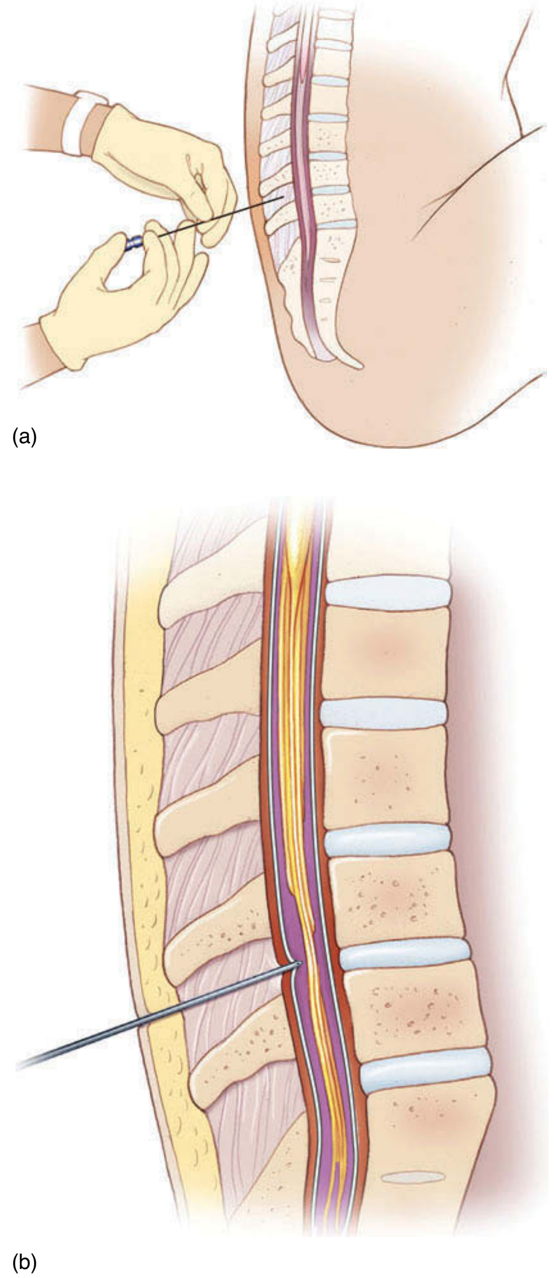


Figure B.10
Lumbar puncture. © Chris Gralapp.

Once sufficient fluid has been collected, reinsert the stylet into the needle and withdraw the needle. Cover the puncture site with a sterile dressing. Older literature suggests maintaining the patient in the recumbent position for the next 4 hours to reduce the likelihood of headache.

Complications

The most serious complication is precipitating uncal or cerebellar tonsillar herniation in patients with intracranial mass lesions. If signs of a herniation syndrome with neurologic deterioration appear following LP, immediate measures should be taken to lower intracranial pressure. LP may also precipitate neurologic deterioration in patients with spinal cord mass lesions.

The most common complication of lumbar puncture is post-procedure headache, occurring in approximately 10–15% of patients. The etiology relates to persistent leakage of CSF through the dural puncture site. This produces CSF hypotension, with resultant traction on the meninges, vessels and other pain-sensitive structures at the base of the brain. The most characteristic feature is pain that is present in the upright position and relieved by lying down. This condition can be treated by intravenous caffeine and/or an epidural blood patch.

Needles have been designed to reduce the incidence of post-lumbar puncture headache. The standard (Quincke) needle is a hollow cutting needle with an introducer. Atraumatic (Sprotte or Whitacre) needles have a duller point and an oval aperture proximal to the tip. Because of the duller point, it is relatively more difficult to puncture the skin, and an introducer is required.

Reference

Straus SE, Thorpe KE, Holroyd-Leduc J. How do I perform a lumbar puncture and analyze the results to diagnose bacterial meningitis. *JAMA* 2006;**296**:2012–22.

Slit lamp examination

Indications

The slit lamp is a valuable instrument for examining the anterior segment of the eye. It provides positional stabilization of the patient's head, with projection of a light beam onto the eye. The examiner can evaluate each eye individually by binocular inspection through the microscope eyepieces.

Slit lamp examination allows a magnified evaluation of the cornea, conjunctiva and the anterior ocular chamber. It is useful for evaluation of injury to the eye, particularly for the diagnosis of corneal abrasion, iritis, ocular foreign bodies and hyphema. Foreign body removal from the cornea and conjunctiva can be done more precisely through the use of the slit lamp.

Contraindications

There are no contraindications to use of the instrument, but the examination cannot be done if the patient is unable to sit upright.

Equipment

- Slit lamp
- Fluorescein strips

Technique

Seat the patient with the chin in the chin rest and the forehead braced against the headrest. You may improve patient comfort by adjusting the table and chin rest heights appropriately. Turn the slit lamp on with the beam initially directed over the bridge of the nose to reduce patient discomfort. Swing the light source to the lateral side of the eye to be examined, positioning it at a 45-degree angle to the eye. Use a vertically aligned light beam.

Using the white light, set the light beam to the maximum height and minimum width. Focus the beam of white light on the cornea by moving the base of the slit lamp forward and backward with the joystick until the beam is sharpest on the patient's cornea. Adjust the focus of the eyepieces as you would for a regular microscope while viewing with each eye individually. Move the base left and right to scan across the cornea and conjunctiva. The cornea can be evaluated for abrasions by instilling fluorescein onto the eye and using the cobalt blue light filter. Widen the beam to 3–4 mm for this purpose.

Focus on the center of the cornea and then push the base slightly forward to focus on the anterior surface of the lens. The depth of the anterior chamber can be assessed in this way. Pull back on the joystick to focus midway between the cornea and the lens. The height of the light beam should be 3–4 mm and as narrow as possible for this portion of the examination. Cells may be identified in the anterior chamber – inflammatory white blood cells in iritis, red blood cells in microscopic hyphema. Inflammatory cells will look like specks of dust; red blood cells will look like brown particles.

Intraocular pressure is measured using the applanation tonometer device found on most slit lamp microscopes. This technique requires a cooperative patient and the use of a topical anesthetic and fluorescein stain. It is advisable to compare pressure measurements between eyes, provided no contraindications exist (e.g., infection, ruptured globe).

Complications

None.

References

Harlan JB, Pieramichi DJ. Evaluation of patients with ocular trauma. *Ophthalmol Clin North Am* 2002;**15**:153–61.

Juang PSC, Rosen P. Ocular examination techniques for the emergency department. *J Emerg Med* 1997;**15**:793–810.

Reduction of dislocations

Shoulder

Indications

The indication for this procedure is the presence of a dislocation of the glenohumeral joint of the shoulder. This is a common dislocation, often the result of athletic injury or falls. The reason for the frequency of this injury is the lack of intrinsic bony stability of the glenohumeral joint, as well as its wide range of motion.

The diagnosis is usually obvious on clinical grounds. The arm is held in slight abduction and external rotation, and range of motion is absent or severely limited. There is a loss of the normal rounded appearance of the shoulder, with a step-off deformity and squared appearance revealing prominence of the acromion process. Although there is some controversy regarding whether radiographs should be done in atraumatic shoulder dislocations, they should be performed in all traumatic injuries resulting in dislocation.

Contraindications

None.

Equipment

- Sheets for countertraction
- Medication and equipment for IV analgesia and sedation

Technique

There are a number of techniques for reduction of shoulder dislocations. Each has its advocates, and practitioners should be familiar with several methods. Administration of parenteral analgesia, muscle relaxants or procedural sedation facilitates reduction in most cases.

The traction–countertraction technique is a frequently utilized method (Figure B.11). Position the patient supine with a folded sheet wrapped around the chest and under the axilla. Have an assistant apply countertraction with this sheet, and apply steady traction along the axis of the humerus with the shoulder abducted slightly. It may take several minutes of continued traction for the reduction to be effective.

For the external rotation approach, place the patient supine and support the elbow with one hand (Figure B.12). Adduct the shoulder and apply longitudinal traction. Slowly and gently externally rotate the shoulder. Once the shoulder is externally rotated to 90 degrees, slowly abduct the shoulder until reduction occurs.

For the scapular manipulation technique, place the patient prone, with the arm hanging off the bed (Figure B.13a). Apply downward traction on the arm. Push the inferior tip of scapula medially while stabilizing the superior portion of the scapula (Figure B.13b). Unlike other methods, this approach attempts reduction by reorienting the glenoid fossa rather than repositioning the humeral head.

Following reduction, ascertain the proper position of the shoulder and identify any fractures through post-reduction radiographs. Immobilize the patient in a sling and swath or shoulder immobilizer. Repeat a neurovascular examination and document the results in the medical record.

Complications

Injury to the axillary nerve occurs in approximately 10% of cases of shoulder dislocation. This usually represents a traction neuropraxia, which has a favorable prognosis for recovery of nerve function. To assure that the nerve has not been injured during the reduction process, it is important to test for its function prior to attempts at reduction, and to document the results of this examination in the medical record. The sensory portion of the nerve provides sensation over the lateral portion of the shoulder (the "military patch" distribution). Test sharp sensation over this area with a pin. The motor portion of the axillary nerve innervates the deltoid muscle. Have the patient attempt shoulder abduction. Feel the contraction of the deltoid muscle by placing your hand over it.

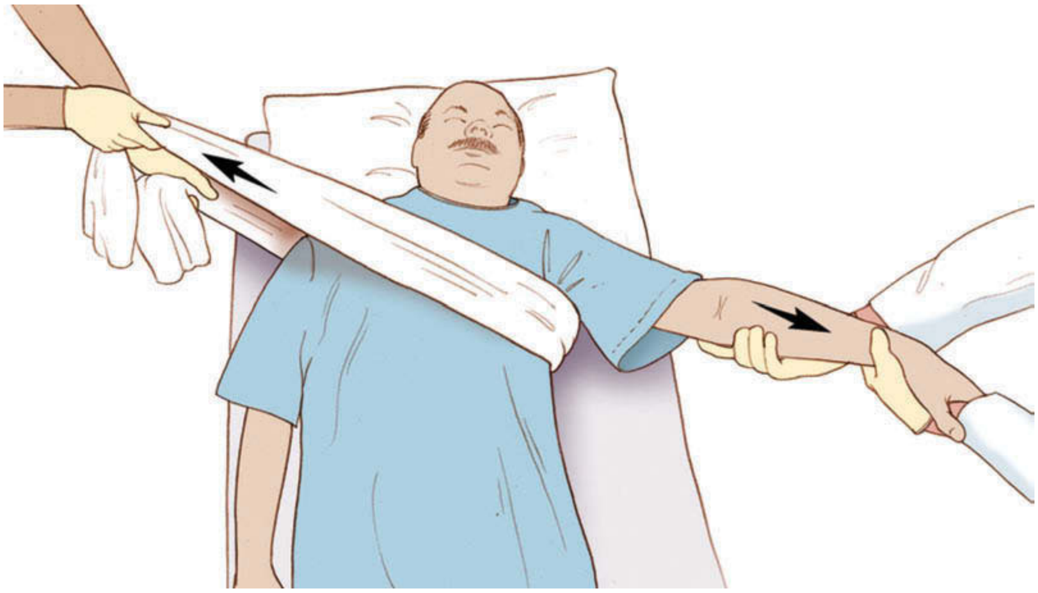


Figure B.11
Traction–countertraction technique for shoulder reduction. © Chris Gralapp.

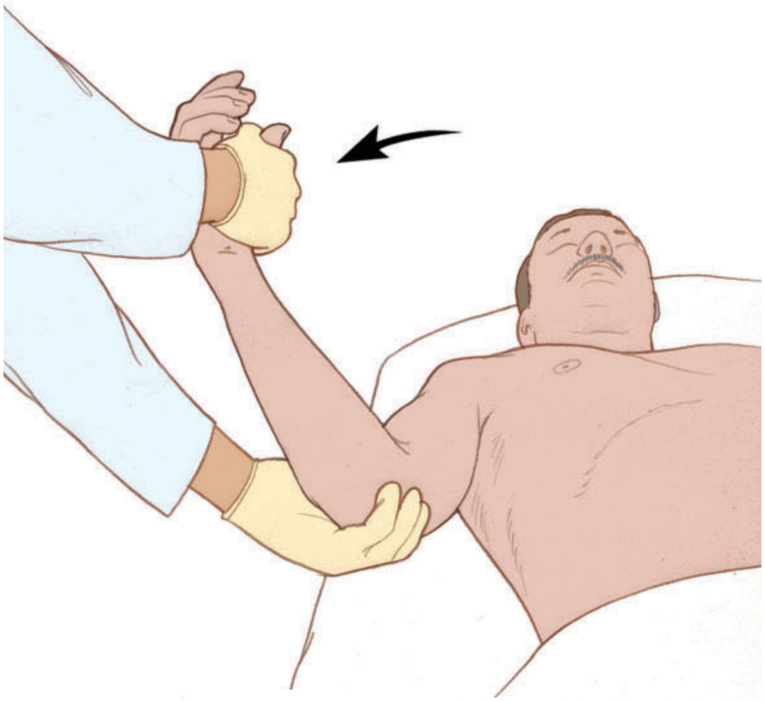


Figure B.12
External rotation method of shoulder reduction. © Chris Gralapp.

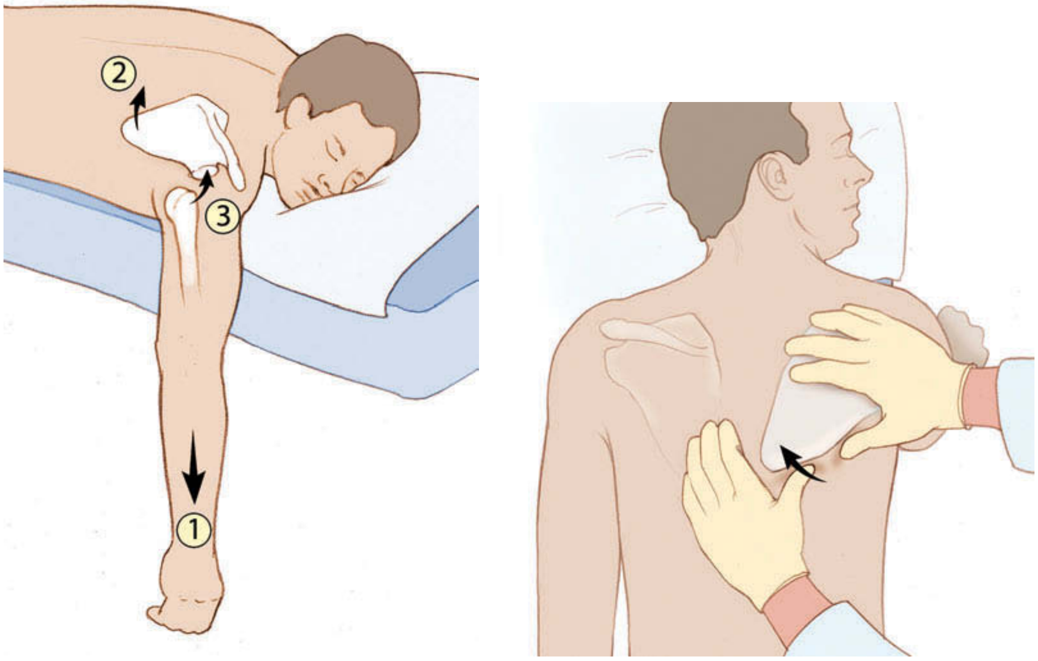


Figure B.13
Scapular manipulation technique for shoulder reduction. © Chris Gralapp.

Fracture of the humeral head may occur as a consequence of reduction.

Radial head subluxation

Indications

This procedure is indicated for an acute radial head subluxation ("nursemaid's elbow"), a very common childhood injury seen most often between the ages of 1 and 5 years, with a peak incidence between the ages of 2 and 3 years. Radial head subuxation usually results from sudden traction being applied to a child's hand or forearm, typically when a parent pulls a child up by the arm during play or to prevent a fall. This results in pulling the annular ligament over the radial head, with the interposition of this ligament (which maintains the radius in its normal relationship relative to the humerus and ulna) between the radius and humeral capitellum.

The result is usually acute pain and unwillingness of the child to move the arm. The arm is held in slight flexion and pronation. Any attempt at motion is usually resisted by the child.

Contraindications

None.

Equipment

None.

Technique

Support the elbow and forearm with one hand, and place the thumb of the other hand over the area of the radial head. Simultaneously, while applying gentle traction, flex the elbow and supinate the forearm. An audible or palpable click may be perceived with reduction. Some clinicians prefer reduction using pronation of the forearm. The child usually becomes pain-free and moves the arm normally shortly after reduction. Given the appropriate mechanism and successful reduction, neither radiographs nor immobilization is necessary.

Complications

Recurrence of the subluxation occasionally occurs.

Phalangeal

Indications

Dislocations of the interphalangeal and metacarpophalangeal joints are common injuries, often occurring during sports activities or falls. Bayonet-shaped or angulated deformities of the fingers are usually readily identified, and constitute indication for reduction. When a skin laceration accompanies the injury, irrigation of the wound and debridement of devitalized tissue may have to follow reduction. These lacerations may need to be sutured after reduction is completed, require antibiotics, and often need urgent orthopedic, plastic or hand surgery consultation.

Contraindications

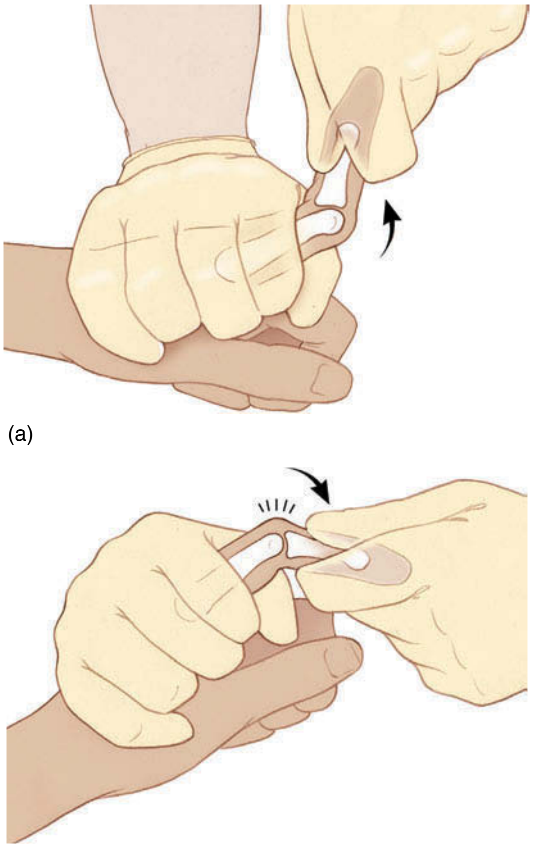
None.

Equipment

- Local anesthetic
- Syringe and 27-gauge needle

Technique

Reduction may require a digital nerve block, though it may be accomplished without anesthesia if the patient is stoic and not too much time has elapsed since the injury. Apply longitudinal traction with slight hyperextension, exaggerating the deformity (Figure B.14a). Reduction is accompanied by a palpable "click" and resolution of the deformity (Figure B.14a). Immobilize with a splint.



(a)

(b)

Figure B.14
Reduction of phalangeal dislocation. © Chris Gralapp.

Complications

Some dislocations, especially those of the metacarpophalangeal joints, may involve the interposition of soft tissue between the dislocated bones. Such dislocations resist the usual methods of reduction, and require operative management. Repeated attempts at closed reduction may produce damage to the soft tissues.

Patella

Indications

Dislocation of the patella usually results from a laterally directed force applied to the medial side of the knee. The knee is often partially flexed at the time of injury. The displacement of the patella on the lateral aspect of the knee produces a dramatic deformity. The presence of dislocation constitutes an indication for reduction. Neurovascular impairment is rarely a feature of this injury.

Contraindications

None.

Equipment

None.

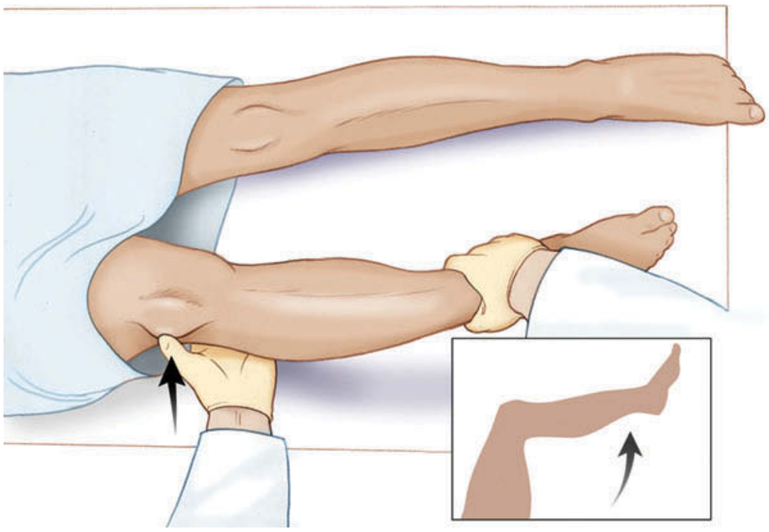


Figure B.15
Reduction of dislocated patella. © Chris Gralapp.

Technique

Although parenteral sedation may be helpful in reducing muscular spasm and pain, the reduction can often be achieved without any medication. Apply medially directed pressure over the lateral border of the patella as the knee is extended (Figure B.15). The patella can be felt to snap into place as reduction occurs. Following reduction, immobilize the knee using a knee immobilizer or a posterior plaster splint.

Complications

Although complications from the procedure are rare, a traumatic joint effusion may follow this injury. Damage to the medial supporting ligaments of the patella may occur, leading to a predisposition for recurrent dislocation.

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Aronen JG, Chronister RD. Anterior shoulder dislocations: Easing reduction by using linear traction techniques. *Phys Sport Med* 1995;**23**:65–9.
Beeeson M. Complications of shoulder dislocation. *Am J Emerg Med* 1999;**17**:288–95.
Brady WJ, Perron AD. Dislocation of the glenohumeral joint. *Crit Decis Emerg Med* 2002;**17**(1):8–16.
Hossfeld GE, Uehara DT. Acute joint injuries of the hand. *Emerg Med Clin North Am* 1993;**11**:781–96.
Schunk JE. Radial head subluxation: Epidemiology and treatment of 87 episodes. *Ann Emerg Med* 1990;**19**:1019–23.

Tube thoracostomy

Indications

Tube thoracostomy (chest tube) is performed to remove blood (hemothorax), fluid (pleural effusion), pus (empyema), or air (pneumothorax) from the pleural space. Each case must be assessed individually as not all patients with a pneumothorax or pleural effusion require tube thoracostomy.

Chest tubes come in various sizes. Larger tubes (30–40 French) are needed for evacuation of a hemothorax. Smaller tubes (12–22 French) are likely to be adequate for the treatment of pneumothorax. Pneumothorax may also be treated with smaller (9–12 French) pigtail catheters. These are inserted via the Seldinger technique (see Central venous cannulation).

The usual placement site for a thoracostomy tube is the fourth or fifth intercostal space in the mid- to anterior axillary line. For pneumothorax, a small tube is sometimes placed in the mid-clavicular line in the second intercostal space.

Contraindications

There are no absolute contraindications to chest tube insertion, provided the appropriate indications are present. Some relative contraindications exist, such as coagulopathy; the patient with a prolonged prothrombin time or thrombocytopenia should have these abnormalities corrected prior to chest tube insertion.

In massive hemothorax, pleural blood may act to tamponade a site of bleeding. Insertion of a thoracostomy tube in such circumstances may precipitate massive hemorrhage once the tamponade effect is removed. Therefore, an auto-transfuser should be available prior to the procedure.

Equipment

- Sterile gloves and drapes
- Povidone–iodine antiseptic solution
- Lidocaine 1% local anesthetic with epinephrine
- Syringe and needles
- Scalpel with No. 10 blade
- Large curved scissors

- Large and medium Kelly clamps
- Chest tubes
- Water seal drainage apparatus (Pleuravac®) with tubing and serrated connector
- Needle holder
- Silk 1–0 suture
- Vaseline-impregnated gauze
- Gauze pads (4 × 4)
- Adhesive tape
- Auto-transfuser (in the case of trauma)

Technique

Prior to initiating the procedure, fill the drainage apparatus with water to the indicated levels. Place the patient in the supine or semi-upright position. In the awake and hemodynamically stable patient, consider IV analgesics and sedatives to make the procedure less painful. Raise the patient's arm above his head on the side used. Put on sterile gloves. Prepare the skin with povidone–iodine solution and drape in a sterile fashion. Anesthetize the skin over the insertion site by injecting lidocaine with epinephrine anesthetic. Infiltrate deeper using a long 25-gauge needle to infiltrate the subcutaneous tissue, muscle and parietal pleura. As much as 20–40 mL of local anesthetic may be required.

Make a 2–3 cm incision over the rib, with the axis of the incision parallel to the rib (Figure B.16a). Bluntly dissect the subcutaneous tissues, using a Kelly clamp or large scissors to separate the tissue by opening and spreading the instrument. Perform the dissection over the top of the rib to avoid the subcostal neurovascular bundle. The dissection tunnel should be large enough to admit your index finger. Direct the dissection in a caudad direction. Penetrate the parietal pleura with a clamp (Figure B.16b). After penetrating the pleura, open the clamp to expand the opening, and then remove the clamp in the open position. Insert your index finger through the pleural opening to assure the absence of adhesions and abdominal organs, and so as not to "lose" the hole's location (Figure B.16c).

Clamp the end of the chest tube with a large clamp and guide the tube through the dissected tunnel into the pleural space (Figure B.16d). Direct it cephalad and posteriorly, and advance until all the side holes over the distal portion are within the thoracic cavity. Remove the clamp. Fluid (pleural effusion) or blood (hemothorax) should now enter the tube. In the case of pneumothorax, condensation of air will be seen on the walls of the tube coincident with respiration. Connect the tube to the water seal drainage apparatus. Tape the junction of the tube and the serrated connector.

Secure the tube to the skin with a purse-string suture, wrapping the suture around the end of the tube before cutting the ends. Cover the insertion site with Vaseline-impregnated gauze. Apply a dressing of 4 × 4 gauze pads. Tape the dressing and also tape a section of the tube in place to the skin. Obtain a post-procedure chest radiograph to assess for the proper position of the tube. The chest tube has a radiopaque stripe along its side to aid in radiographic identification.

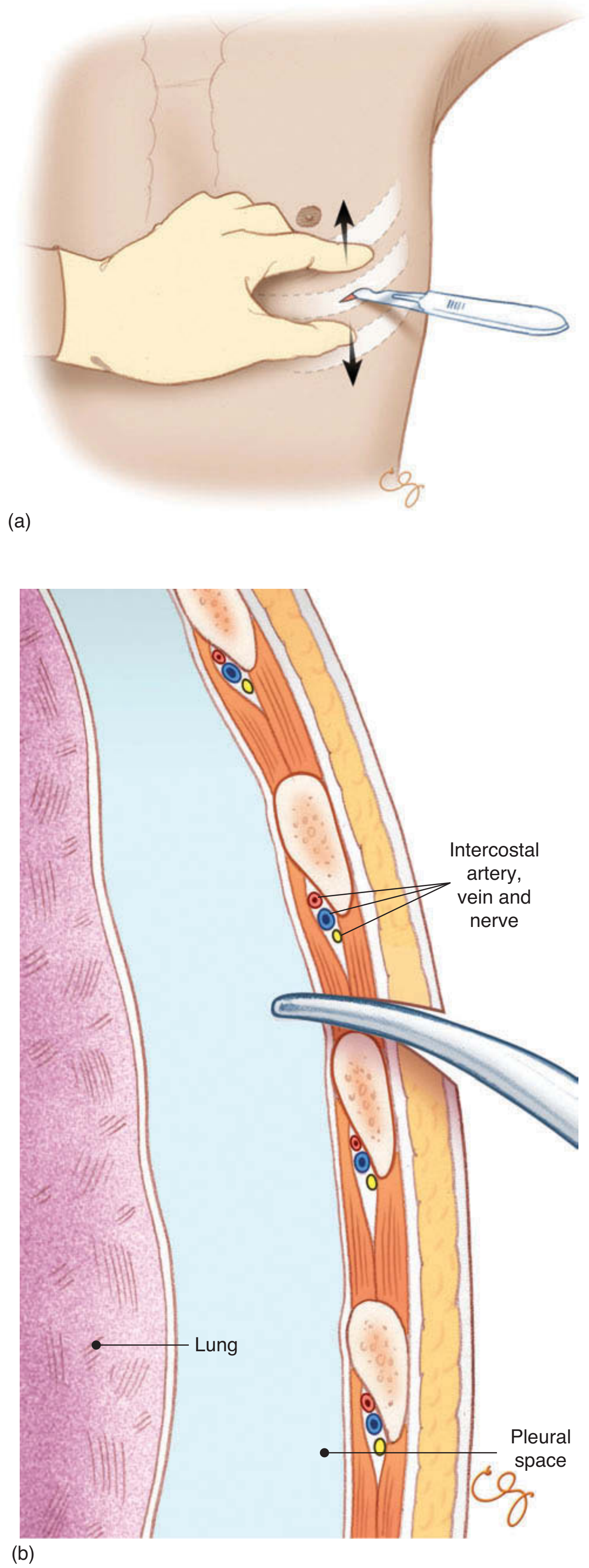
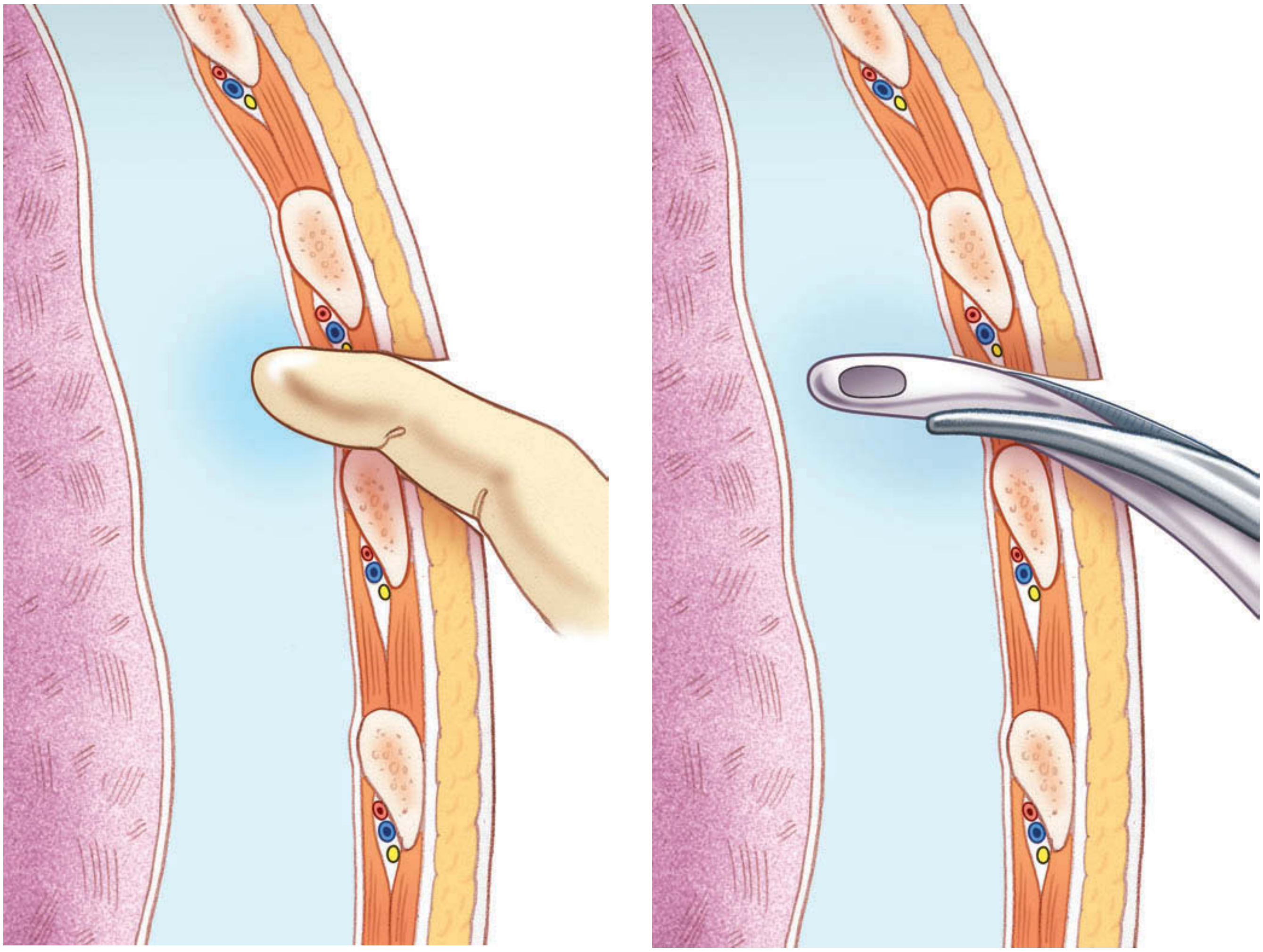


Figure B.16
The thoracostomy (a) incision, (b) clamp penetration.



(c)

(d)

Figure B.16 (*cont.*)
(c) insertion of finger and (d) insertion of chest tube into the pleural cavity using curved clamp. © Chris Gralapp.

Complications

Patients with severe chronic obstructive pulmonary disease may have large pulmonary bullae that may be mistaken for pneumothoraces. Insertion of a chest tube in this instance can worsen pulmonary function.

Failure to suture and tape the tube to the chest may result in accidental extrusion of the tube. Failure to tape the connections may cause separation of the chest tube from the water seal drainage apparatus, resulting in recurrence of the pneumothorax.

Failure to assure that all of the side holes of the thoracostomy tube are positioned within the pleural space causes aspiration of air from a pneumothorax into the soft tissue of the chest, producing subcutaneous emphysema. Passage of the tube into the subcutaneous tissue rather than the pleural space will fail to evacuate the space of fluid or air.

Bleeding may occur if an intercostal artery or vein is lacerated during the procedure. The lower border of the rib should be avoided as a site of incision to avoid these vessels. Intercostal nerves can be injured in similar fashion. Other intrathoracic vessels can be injured during insertion of the tube, and the tube should not be forced through the subcutaneous tunnel or into the pleural space. Laceration of lung adherent to the pleura, liver and other abdominal organs can occur. The entry site through the pleura should be palpated prior to insertion of the tube to avoid such complications.

References

Iberti TJ, Stern PM. Chest tube thoracostomy. *Crit Care Clin* 1992;**8**:879–95.

Quigley RL. Thoracentesis and chest tube drainage. *Crit Care Clin* 1995;**11**:111–26.

Roberts JS, Bratton SL, Brogan N. Efficacy and complications of percutaneous pigtail catheters for thoracostomy in pediatric patients. *Chest* 1998;**114**:1116–26.

Abscess incision and drainage

Indications

Indication for this procedure is the presence of a cutaneous or subcutaneous abscess (collection of inflammatory and infectious products encapsulated by granulation tissue). The clinical hallmark is a tender mass with fluctuance – the liquid nature of the contents can be palpated through the skin. In most patients, a cutaneous abscess constitutes a local infection caused by skin flora.

Contraindications

There are no absolute contraindications to performing incision and drainage of an abscess. If there is evidence of local infection (e.g., redness, swelling), but no fluctuance, the procedure may be delayed until after a trial of antibiotic therapy. On the face and other areas where a scar is undesirable, repeated aspiration may be an alternative to incision and drainage. In the septic or severely immune-compromised patient, the procedure may have to be delayed until after antibiotic therapy is initiated.

Equipment

- Sterile gloves
- Local anesthetic solution, syringe and needles
- Eutectic mixture of local anesthetics (EMLA) gel
- Sterile drapes
- Povidone–iodine antiseptic solution
- Irrigating syringe (30 mL)
- Saline irrigating solution
- Ribbon packing tape (¼ or ½-inch width)
- Scalpel with No. 11 blade
- Hemostat
- Scissors
- Gauze pads

Technique

Prepare and drape the area in sterile fashion. Performing incision and drainage is an extremely painful procedure, and it may be difficult to achieve adequate local anesthesia. Apply a layer of EMLA gel over the area of fluctuance. Cover this with a patch of Tegaderm or clear plastic wrap. After a period of 30–60 minutes, inject subcutaneous local anesthetic across the dome of the abscess (Figure B.17a). Using a scalpel with a No. 11 blade, make an incision over the area of maximal fluctuance and extend it into the abscess cavity (Figure B.17a).

Gently probe the cavity with a hemostat to free all loculated tissue (Figure B.17b). This may be the most painful portion of the procedure, as skin infiltration will not provide anesthesia in the depths of the cavity. Irrigate the abscess cavity with saline solution. Pack the wound with ribbon tape. Do not pack the cavity tightly, as this may trap purulent material. Apply a gauze dressing.

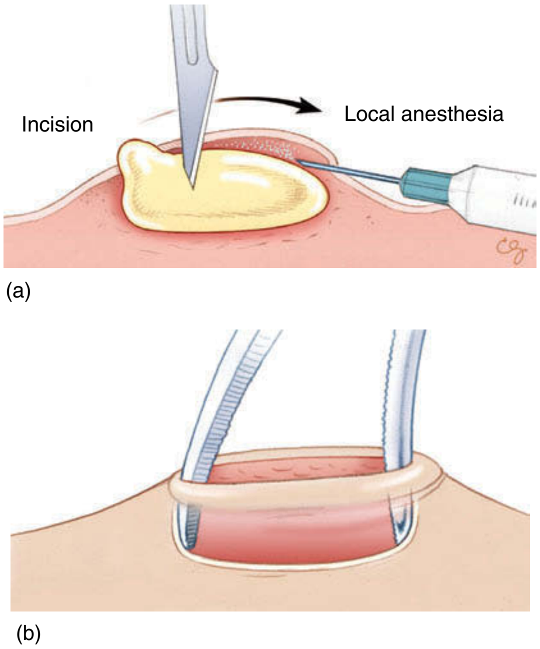


Figure B.17
Abscess infiltration, incision and drainage. © Chris Gralapp.

Complications

Whenever a skin incision is made, there is risk of injury to the surrounding tissue.

Infection can be spread into adjacent tissue or the bloodstream by abscess drainage. Inadequately probing the interior of the abscess cavity may result in the reaccumulation of infectious fluid and return of symptoms.

Reference

Bisno AL, Stevens DL. Streptococcal infections of the skin and soft tissues. *N Engl J Med* 1996;**334**:311–17.

Knee arthrocentesis

Indications

This procedure is performed chiefly for obtaining joint fluid for diagnostic analysis. In the presence of a large joint effusion or blood in the joint space (hemarthrosis), removal of fluid will also reduce the patient's discomfort.

Contraindications

The presence of cellulitis or other infection at the site of aspiration constitutes an absolute contraindication. Suspected bacteremia, bleeding diathesis or uncorrected coagulopathy represent relative contraindications. If the knee is a prosthetic joint, this procedure should only be done in conjunction with orthopedic consultation.

Equipment

- Sterile gloves and drapes
- Lidocaine 1% local anesthetic
- 18-gauge needle
- 10-mL syringe
- Povidone–iodine antiseptic solution

Technique

Have the patient lie supine on the gurney with the knee extended or slightly flexed. A rolled towel can be placed behind the knee to support it in slight flexion. Prepare and drape the area in sterile fashion. Identify the area to be entered. This should be at the midpoint of the patella, on either the lateral or medial side. Infiltrate this area with local anesthetic.

Using an 18-gauge needle and 10-mL syringe, direct the needle parallel to the gurney and advance beneath the undersurface of the patella, taking care not to injure the bone. Advance the needle and aspirate gently until synovial fluid is drawn into the syringe (Figure B.18).

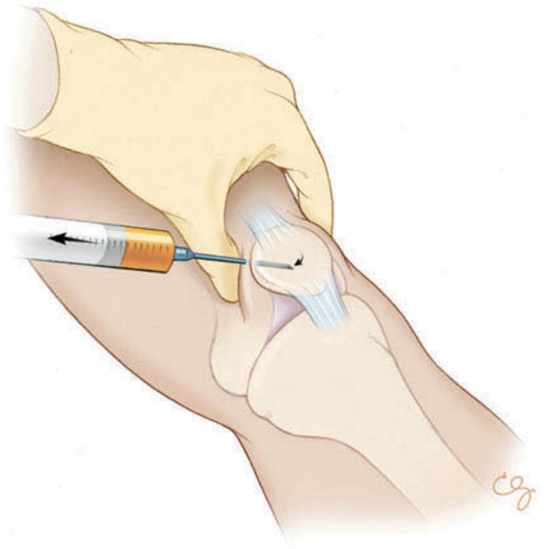


Figure B.18
Knee arthrocentesis. © Chris Gralapp.

If the 10-mL syringe becomes filled, you may leave the needle in place and remove additional fluid by attaching a larger syringe. Once all desired fluid has been withdrawn, remove the needle and apply a compression dressing.

Send the synovial fluid for the desired diagnostic testing. This usually includes white blood cell count, Gram stain, culture and crystal analysis.

Complications

Infection can be introduced into the joint space if entry is through an infected area or proper antiseptic technique is not utilized. Hemarthrosis can be produced in the presence of an underlying coagulopathy. Inadvertently striking the patella with the needle may damage the articular surface.

Reference

Thomsen TW, Shen S, Shaffer RW, et al. Arthrocentesis of the knee. *N Engl J Med* 2006;**354**:19–21.

Thoracentesis

Indications

Thoracentesis is the aspiration of fluid from the pleural space. Because such fluid can accumulate as a result of a large number of processes, the procedure is usually performed for diagnostic evaluation of pleural fluid. When a large effusion produces shortness of breath, evacuation of the fluid can be therapeutic.

Contraindications

Do not perform thoracentesis through skin that is infected. A bleeding disorder or anticoagulation constitute contraindications to the procedure. Do not attempt aspiration of an effusion known to be loculated.

Equipment

- Sterile gloves and drapes
- Lidocaine 1% local anesthetic, syringe and needle
- 16-gauge needle (with angiocatheter if indicated)
- 10-mL syringe
- Povidone–iodine antiseptic solution
- Fluid collection bottles, tubing with 3-way stopcock

Technique

Have the patient sit on the edge of the gurney, leaning forward on a Mayo stand. The level of the effusion can be determined either by physical examination (percussion dullness) or ultrasonography. Perform sterile preparation of the back two intercostals spaces below the uppermost extent of the effusion (but no lower than the ninth rib).

Palpate the superior edge of a rib in this area in the posterior axillary line. Infiltrate this area with 3–5 mL of local anesthetic. For diagnostic thoracentesis, use a 16- or 18-gauge needle attached to a syringe. For therapeutic aspiration, use an angiocatheter over a needle. Insert the needle over the superior margin of the rib and advance perpendicular to the thorax. Aspirate for fluid (Figure B.19).

When performing a therapeutic thoracentesis, once fluid is aspirated, advance the angiocatheter over the

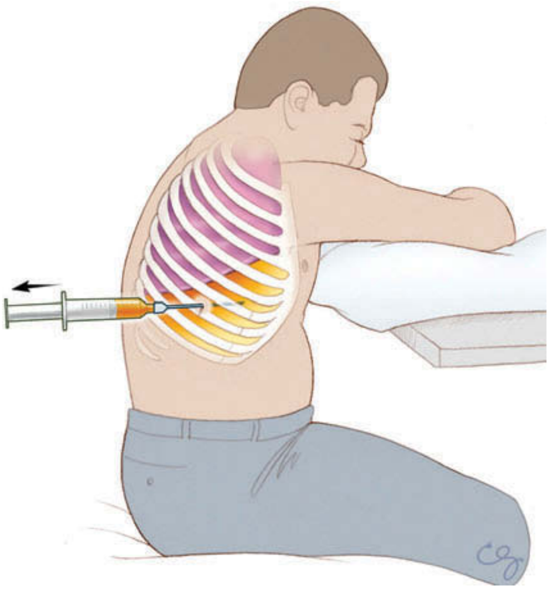


Figure B.19
Thoracentesis. © Chris Gralapp.

needle and withdraw the needle. Cover the angiocatheter hub with your thumb. Have the patient briefly avoid inspiration, and attach a three-way stopcock and collection tubing to the angiocatheter. Attach the tubing to the collection bottle. Turn the stopcock so that it is open to the bottle, and allow the desired amount of fluid to be collected.

Remove the catheter or needle and apply an adhesive dressing to the puncture site. Obtain a post-procedure chest radiograph.

Complications

Infection can be introduced into the pleural space or subcutaneous tissue if entry is through an infected area or proper antiseptic technique is not used. A pneumothorax can be produced by allowing air to enter the pleural space through the needle or angiocatheter. Their apertures must be covered at all times and the collecting system maintained closed to the atmosphere.

Injury to the intercostal vessels, diaphragm, or pulmonary parenchyma can be produced with the needle. Inasmuch as the intercostal neurovascular structures travel along the inferior border of the ribs, injury to these structures can be avoided by adhering to the procedural landmarks and staying above the rib.

Re-expansion pulmonary edema may occur following thoracentesis. This is not a complication of faulty procedure, but should be suspected (along with pneumothorax) if the patient develops shortness of breath following fluid evacuation.

References

Minolga GP. Re-expansion pulmonary edema. *J Emerg Med* 2009;**36**:122–7.

Qureshi N, Momin ZA, Brandstetter RD. Thoracentesis in clinical practice. *Heart Lung* 1994;**23**:376–83.

Paracentesis

Indications

Paracentesis is the aspiration of ascites fluid from the abdominal cavity. It is usually performed in patients with newonset ascites of unknown origin. It is also used as a diagnostic procedure in patients with pre-existing ascites who develop findings (e.g., fever, abdominal pain) suggestive of infection.

The procedure can be performed therapeutically in the patient with tense, large-volume ascites.

Contraindications

The puncture site should not be at an area of abdominal wall cellulitis. Uncorrected bleeding diathesis constitutes a contraindication. The procedure must be done with extreme caution in the presence of known intra-abdominal adhesions or intestinal obstruction.

Equipment

- Sterile gloves and drapes
- Lidocaine 1% local anesthetic with syringe and needle
- 16- or 18-gauge needle (with angiocatheter if indicated)
- 10-mL syringe
- Povidone–iodine antiseptic solution
- For therapeutic paracentesis, 1-liter fluid collection bottles and tubing

Technique

Have the patient empty the bladder or insert a urinary catheter. Position the patient on the gurney with the head elevated to 45 degrees. The exact location of the ascites fluid can be determined by ultrasound. The preferred aspiration site is the abdominal midline 2 cm inferior to the umbilicus. Alternatively, the puncture can be made in either lower abdominal quadrant (Figure B.20).

Prepare the area to be aspirated with povidone–iodine antiseptic solution and apply sterile drapes. Anesthetize the area with lidocaine. Use a 16- or 18-gauge needle on a 10-mL syringe to aspirate the fluid. Some recommend the use of the "Z-track" technique to prevent the subsequent development of ascites leaks. Once the needle penetrates the skin, pull the abdominal skin downward with the non–needle-bearing hand. Advance the paracentesis needle and do not release the skin until the peritoneum has been penetrated and fluid aspirated into the syringe. The skin will then return to its original position and help seal the entry tract.

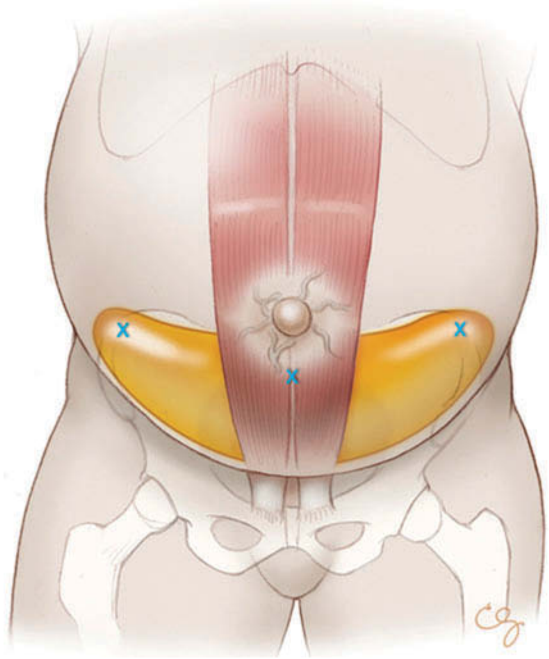


Figure B.20
External landmarks for paracentesis. © Chris Gralapp.

Once fluid has been aspirated into the 10-mL syringe, a larger syringe can be attached to obtain addition fluid. The sample should be sent for diagnostic analysis, as indicated. In consideration of peritonitis, a white blood cell count, Gram stain and fluid culture are essential.

When fluid aspiration is complete, remove the needle and apply a dressing to the puncture site.

Complications

Introduction of infection is a potential complication, and chances can be reduced by sterile technique. Persistent ascites leak at the puncture site occurs periodically, as does local wound hematoma. Perforation of intraperitoneal viscera or vascular structures is a hazard, but is rare. Hypotension has been described following removal of large-volume ascites fluid.

Reference

Runyon BA. Paracentesis of ascetic fluid: A safe procedure. *Arch Intern Med* 1986;**146**:2259–61.

Appendix C Laceration repair

Wendy Coates, MD **and Michelle Lin,** MD

Scope of the problem

Over 12 million patients with wounds present to emergency departments (EDs) in the United States annually. Complications such as wound infection (which occurs in approximately 3–7% of all traumatic wounds), wound dehiscence and poor cosmesis are directly related to the adequacy of wound preparation. This appendix addresses injury assessment, wound preparation, and various approaches to laceration repair.

Anatomic essentials

As the body's largest and most exposed organ, the skin is subject to a variety of external forces encountered in daily activities. The skin's anatomic structure and layers must be considered when planning a repair (Figure C.1).

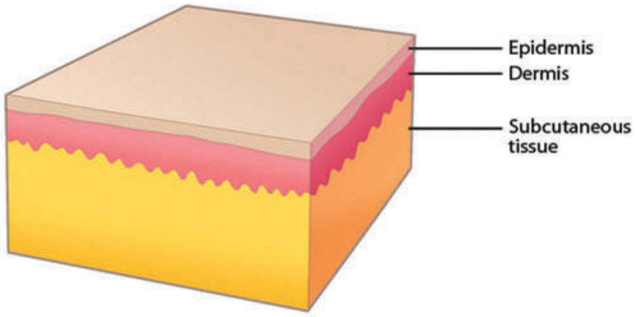


Figure C.1
Cross-section of the skin. © Chris Gralapp.

Lacerations penetrating only the epidermis are minor and may not warrant major repair. The underlying dermis is one to several millimeters thick, depending on its location. Repair of this layer provides structural integrity for a healing wound. In some cases, especially wounds under tension, a separate deep dermal closure may be required to provide continued support after the epidermal sutures have been removed. The dermis rests on subcutaneous tissue, which contains adipose and other loose connective tissue. The repair of subcutaneous tissue does not contribute to final wound strength. However, re-approximation of subcutaneous tissue can eliminate potential spaces, thereby decreasing the risk of infection in selected wounds.

Wound healing and the final cosmetic outcome following laceration repair depend on many factors, including static and dynamic tension. Static tension is determined by intrinsic skin factors such as collagen concentration. Anatomic determinants such as underlying bone, tendon, muscle, and location over a joint space impact the dynamic tension of a repaired laceration. Wounds oriented along the body's natural lines of tension are under less stress, and lead to more favorable cosmetic outcomes. Lacerations that are oriented perpendicular to these lines are under higher tension and leave more noticeable scars; therefore, the method selected for closure may require more tensile support.

History

When did it happen?

Time from injury to cleansing and closure affects the likelihood of wound infection and guides the provider's decision to close a wound primarily (Table C.1). Lacerations on the head and face generally can be closed up to 24 hours after injury, whereas injuries on the hands, feet and trunk are at higher risk for infection after a 12 hour delay. Wounds evaluated beyond this time frame may be considered for delayed primary closure (Table C.2).

Table C.1 Wound closure in relation to time

Primary closure	Physical re-approximation of wound edges soon after injury
Healing by secondary intention (granulation)	Allowing a wound to granulate in from the edges with no wound edge re-approximation
Delayed primary closure (healing by tertiary intention)	Physical re-approximation of wound edges after debridement, packing and antibiotic prophylaxis for 3–4 days

What was the mechanism of injury?

Crush injuries, puncture wounds, burns and contaminated wounds (human or animal saliva, feces, soil, organic material) are at higher risk for wound infection and warrant special consideration. Some of these wounds should not be immediately repaired in the ED and/or may require prophylactic antibiotics to decrease the chance of infection. The mechanism of injury (e.g., wound sustained on broken glass) may raise concern for a retained foreign body and warrant imaging and meticulous exploration before wound closure.

Table C.2 Delayed primary closure technique

Wound characteristics	Day of evaluation (day 1)	Re-evaluation (day 3)
• Sustained >12 hrs prior to evaluation (>24 hrs on face) • "Dirty" wound (e.g., animal or human bite, infected wound)	• Assess wound • Anesthetize • Detailed examination • Irrigate wound • Non-adherent gauze between wound edges • Sterile dressing • 5–7 days of antibiotics (e.g., cephalexin)	• Re-evaluate wound for infection, maceration, necrotic tissue • Repair according to standards, or let heal by secondary intention (referral may be indicated) • Finish antibiotic course

Table C.3 Guide to tetanus prophylaxis in routine wound management among adults aged 19–64 years

Characteristic	Clean, minor wound		All other wounds[a]	
History of adsorbed tetanus toxoid (doses)	Tdap or Td[b]	TIG	Tdap or Td[b]	TIG
Unknown or <3	Yes	No	Yes	Yes
≥3	No[c]	No	No[d]	No

[a] Such as, but not limited to, wounds contaminated with dirt, feces, soil, and saliva; puncture wounds; avulsions; and wounds resulting from missiles, crushing, burns, and frostbite.

[b] Tdap is preferred to Td for adults who have never received Tdap. Td is preferred to TT for adults who received Tdap previously or when Tdap is not available. If TT and TIG are both used, Tetanus Toxoid Adsorbed rather than tetanus toxoid for booster use only (fluid vaccine) should be used.

[c] Yes, if ≥10 years since the last tetanus toxoid-containing vaccine dose.

[d] Yes, if ≥5 years since the last tetanus toxoid-containing vaccine dose.

From Kretsinger K, Broder KR, Cortese MM, et al. Preventing tetanus, diphtheria, and pertussis among adults: Use of tetanus toxoid, reduced diphtheria toxoid and acellular pertussis vaccine. *MMWR Recomm Rep* 2006;55(RR-17):1–37.

Td: tetanus-diphtheria; Tdap: tetanus-diphtheria-acellular-pertussis; TIG: tetanus immune globulin; TT: tetanus toxoid.

A comprehensive history may uncover related conditions that require more urgent attention than the laceration. For example, syncope leading to a minor forehead laceration obligates a careful work-up, compared with a mechanical fall leading to a laceration in the same area. Wounds to the face and multiple bruises in different stages of healing should arouse suspicion for intimate partner violence or child abuse.

Do you have allergies to medications?

It is important to know medication allergies before administering tetanus prophylaxis, analgesics, wound anesthesia, antibiotics, or using latex products.

What is your tetanus immunization status?

An assessment of tetanus immunization status and subsequent immunization with tetanus immune globulin (TIG) or tetanus toxoid is important. Current recommendations for immunization include the administration of one Tdap (tetanus toxoid, reduced diphtheria toxoid and acellular pertussis vaccine) for adolescents and adults. Table C.3 provides guidelines for administering tetanus immunization according to the Centers for Disease Control and Prevention (CDC).

Past medical

Information regarding preexisting patient illnesses and comorbidities may impact disposition. This information may help guide decisions of whether to use prophylactic antibiotics, and may predict which patients are prone to impaired wound healing (Table C.4).

Physical examination

The physical examination should take place in a well-lit area with the patient as comfortable as possible. Assess the wound in relation to the history provided by the patient to identify other possible injuries (e.g., retained foreign bodies). When focusing on the wound itself, there are several important principles to help guide your examination.

Description of the wound

Describe the type, anatomic location, length and depth of the wound. The health of the wound margins and degree of tension should be noted (Table C.5).

Distal neurologic integrity

Examine for motor *and* sensory deficits prior to anesthetizing the wound. The remainder of the physical examination can be performed after the wound has been anesthetized. Nerves can be partially lacerated. Sensory information is carried peripherally in nerve bundles; loss of sensation may be the only manifestation of a partial nerve injury. Because nerves and vessels are in close proximity, a vascular injury should raise concern for an accompanying nerve injury.

Distal vascular integrity

Assess and document adequacy of perfusion using a combination of pulses, capillary refill and skin color.

Table C.4 History that may impact care of a patient's wound

Immunocompromised	
Diabetes	Extremity injuries often lead to infection because of poor circulation and immune system dysfunction. There can be relative tissue hypoxia. Have a low threshold for antibiotics and early wound re-evaluation. Avoid unnecessary deep sutures.
HIV/AIDS	Lower threshold for using appropriate antibiotics in wounds that are at high risk for infection.
Steroid/immunosuppressant use	These medications cause prolonged healing time and weaken the immune system. These patients may need a prolonged period of healing prior to suture removal, and are at high risk for infection.
Vascular	
Bleeding disorder	Obtain impeccable hemostasis before closing these wounds. Assess for anemia. A hematoma can serve as culture medium for wound infection.
Peripheral vascular disease	Poor peripheral circulation increases wound infection risk. Carefully assess neurovascular status before repairing extremity injuries.
Other	
Cardiac disease	Make sure that the wound resulted from a mechanical and not cardiac etiology.
Domestic/child/elder abuse	The key is to suspect these situations. Have a high index of suspicion and a low threshold for reporting when the history does not match the injury.

Table C.5 Types of wounds and characteristics

Type of wound	Characteristics	Treatment considerations
Abrasions	Superficial	Clean and dress.
Avulsions	Tissue is missing	Clean, complex repair, or referral.
Burns	1st, 2nd, or 3rd degree	Local wound care and possible resuscitation or transfer to a burn specialty center.
Crush injuries	Caused by blunt forces, may have significant tissue edema. Wound infection occurs at a lower bacterial load.	Address underlying trauma life support considerations, then use standard wound care techniques.
Lacerations	Sharply demarcated borders. Usually have good healing.	Follow standard wound care techniques.
Puncture wounds	May be small but indeterminate depth.	Evaluate for presence of foreign body and underlying injury. Surface cleaning and dressing.

Vascular bruits may suggest pseudoaneurysms, vascular lacerations, or fistulas. Ankle brachial indices (ABIs) can be measured when there is concern for an arterial injury. Signs of poor perfusion should be explained and addressed before laceration repair. Determine and document neurovascular status following repair to ensure iatrogenic injuries did not occur during the repair.

Tendon integrity

Lacerated tendons are at risk for rupture. The tendon must be visualized during the examination throughout its entire range of motion through the wound. Tendon lacerations in the position of injury may not be visible in the position of examination. Many specialists advise tendon repair if there is greater than 50% disruption. Some extensor tendon lacerations of the hand and foot can be repaired by emergency physicians skilled in this procedure. Flexor tendons should be repaired under sterile conditions by a trained specialist in the operating room. Alternatively, some specialists prefer splinting of partial tendon lacerations followed by a program of rehabilitation.

Joint space involvement

Laceration extension into a joint space portends morbidity for the patient and may require washout in the operating room. Maintain a high suspicion for joint capsule disruption with wounds located over joints, mechanisms suggesting deep penetration, debris visualized on X-ray over a joint space, or the presence of a radiopaque foreign body in the joint space itself. These wounds may need to be assessed by a specialist and treated with prophylactic antibiotics.

Bone involvement

Obtain plain film radiography in any laceration with suspected underlying fracture or exposed bone. Cortical disruption of bone with proximity to a laceration is classified and treated as an open fracture. These wounds require copious irrigation and systemic antibiotics to minimize the risk of osteomyelitis.

Foreign bodies and wound contamination

Wound contamination obligates a thorough cleaning, an assessment of whether or not to close the wound primarily (Table C.1), and careful exploration for other foreign bodies in the wound. Imaging modalities used to detect foreign bodies include plain X-rays, xerography, ultrasound (US), computed tomography (CT) and magnetic resonance imaging (MRI). MRI should not be performed if a metallic foreign body is suspected or other contraindications are present.

Devitalized tissue

Crush, blast, or high-speed missile injuries impart large forces to tissues and can cause necrosis of dermal and subcutaneous structures. Necrotic tissue can serve as a nidus for infection and impede normal tissue healing. Devitalized tissue should be debrided conservatively to preserve as much viable tissue as possible to minimize wound tension. This affords a cosmetic surgeon the most flexibility for subsequent wound revision, if necessary.

Referral and consultation guidelines

When the history and physical examination is complete, the provider should determine whether he or she possesses the skill necessary to proceed with the repair. Adequate wound preparation should take place prior to the referral, and a clean, dry dressing should be applied. Wound characteristics that should be considered for consultation and referral are noted in Table C.6.

Table C.6 Wounds appropriate for consultation/referral

- Provider is unable to perform optimal repair
  - Skill level does not match complexity of wound
  - Practice setting does not allow adequate time or have appropriate equipment for repair
- Underlying injury
  - Tendon
  - Nerve
  - Vascular
- Joint involvement or underlying fracture
- Eyelid: tarsal plate or lacrimal duct involvement
- Patient requests specialist
- Operative repair necessary
  - Skin grafting
  - Flap creation or rotation

Diagnostic testing

Patients with lacerations rarely require an extensive diagnostic work-up. Radiologic imaging may be indicated for suspicion of a foreign body, joint space involvement, osteomyelitis, fracture, or underlying injury (e.g., CT of the brain). In patients presenting with a laceration as a minor part of their overall condition (e.g., a multiple trauma victim or patient with syncope), the work-up should focus on the primary event in addition to repairing the laceration.

Wound preparation

Setup

Preparation of both the patient and provider is essential in any case requiring wound management. Patient preparation includes:

1. Establishing sufficient lighting for good wound visualization;
2. Placing the patient in a reclined or semi-reclined position to anticipate a possible vasovagal event from pain or anxiety;
3. Positioning the patient so that the wound is most accessible to the provider, such as raising the patient's gurney to the provider's waist level.

The provider must follow universal precautions, including a gown, face or eye protection, and gloves. This is especially important during wound irrigation, when high-pressure irrigation fluid may splash bloodborne products unpredictably.

Anesthesia

After a complete neurovascular examination distal to the wound, the next step is to anesthetize the wound. This allows the provider to copiously irrigate and meticulously explore the wound, and then examine tendon function before closure, as patient discomfort is no longer a limiting factor. According to a large American College of Emergency Physicians' study of medicolegal claims from the ED, missing retained foreign bodies in wounds is the fifth leading cause of lawsuits.

Lidocaine and the longer-acting bupivacaine are the two most commonly used anesthetic agents. Both share onset of pain relief in 2–5 minutes. Lidocaine lasts for approximately 1–2 hours; bupivacaine lasts 4–8 hours. For the pediatric population, small volumes of an anesthetic may be toxic. It is therefore important to calculate the toxic anesthetic dose prior to administration (Table C.7). Lidocaine and bupivacaine toxicity can cause seizures, dysrhythmias, and cardiac arrest. Concurrent use of epinephrine improves hemostasis and reduces systemic anesthetic absorption by local vasoconstriction. Traditionally, epinephrine should not be used for end-circulation anatomic areas, including the fingers, toes, ears, nose and penis. A study by Whilhelmi et al. demonstrated that digits injected with lidocaine plus epinephrine had no added complications compared with lidocaine alone, suggesting that this dictum may be over-conservative. For those "allergic" to these

Table C.7 Local anesthetic toxicity

Adult example		
	Maximum dose	*Maximum volume for a 70 kg adult patient*
Bupivacaine	3 mg/kg	Using 0.25% bupivacaine: (3 mg/kg)(70 kg) / (2.5 mg/mL) = 84 mL
Lidocaine	5 mg/kg	Using 1% lidocaine: (5.0 mg/kg)(70 kg) / (10 mg/mL) = 35 mL
Lidocaine + epinephrine	7 mg/kg	Using 1% lidocaine + epinephrine: (7 mg/kg)(70 kg) / (10 mg/mL) = 49 mL
Pediatric example		
	Maximum dose	*Maximum volume for a 2 year-old child (12 kg) with a leg laceration*
Lidocaine	5 mg/kg	Using 1% lidocaine: (5 mg/kg)(12 kg) / (10 mg/mL) = 6 mL

Note: In the pediatric example, the provider should not use more than 6 mL (or 60 mg) of 1% lidocaine.

anesthetics, cardiac lidocaine (which is preservative-free) may be used. If a true lidocaine allergy exists, 1% diphenhydramine can serve as an effective alternative agent, although it causes relatively more pain on administration.

There are four different approaches to achieve wound anesthesia: topical, local infiltration, regional block and procedural sedation.

Topical

Topical anesthesia can be used as the sole means of anesthetizing a wound, or it may be used in conjunction with local infiltration. Due to the high vascularity of the face and scalp, topical anesthetics are very effective in this area. Commercially prepared agents are (1) TAC (a mixture of tetracaine, adrenaline/epinephrine and cocaine); (2) LET (a mixture of lidocaine, epinephrine and tetracaine); and (3) eutectic mixture of local anesthetics (EMLA) cream. EMLA is intended for use only on intact skin and may be effective when applied to the site where a regional anesthetic injection is to take place. TAC has been associated with several case reports of seizures and death from inadvertent mucosal absorption of cocaine; as a result, it is unavailable in most EDs.

A cotton ball soaked with approximately 3–5 mL of the anesthetic agent or a gel formulation (e.g., LET) is applied to the open wound for at least 10 minutes. To maximize absorption, apply firm pressure with a strong adhesive tape. Alternatively, for the frightened pediatric patient, a family member wearing gloves can apply the cotton ball to the patient's laceration. The presence of blanched wound edges marks successful absorption of anesthetic, as absorption of epinephrine from TAC or LET causes local wound vasoconstriction. Caution should be taken with application around the eyes to prevent inadvertent corneal exposure.

Local infiltration

Local infiltration, the most common approach to anesthesia in wound care, involves injecting an anesthetic into both wound edges at the dermal-subcutaneous layer. Starting at one apex, deposit a small amount of anesthetic in the subcutaneous tissue within the wound. Avoid injecting through intact skin because this is more painful than injecting in the wound itself. Be sure to check for inadvertent vascular cannulation by aspirating for blood before instilling anesthetic. Advance the needle to its full length along one wound edge. Deposit anesthesia while retracting the needle out of the skin. Reinsert the needle at the leading edge of anesthesia and continue this process circumferentially around the wound (Figure C.2).

Five different techniques can reduce the pain of anesthetic infiltration.

1. Premedicate the wound with a topical anesthetic (described above) or ice. This partially anesthetizes the wound edges before injecting with the needle.
2. Local anesthetics such as lidocaine and bupivacaine are weak acids; mixing the medication with bicarbonate produces a more neutral pH and less painful anesthetic. For 1% lidocaine, mix 1 mL of 8.4% bicarbonate with 9 mL of the anesthetic. For bupivacaine, mix 0.1 mL of 8.4% bicarbonate with 9.9 mL of the anesthetic. Excess bicarbonate may cause solute precipitation.

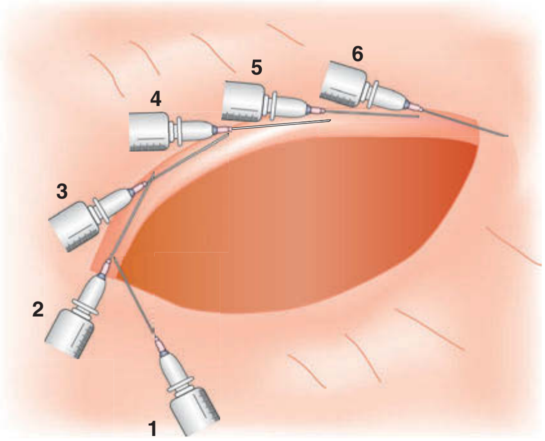


Figure C.2
Local anesthetic infiltration technique. Local infiltration first starts with a subcutaneous wheal of anesthetic at one apex from within the wound (syringe 1 on bottom left). Subsequent injections (syringes 2–6) along the wound edge start at the leading edge of anesthesia. The process should be continued for the other side of the wound for complete anesthesia.

3. Warm the anesthetic syringe in your hand for several minutes to room temperature to reduce the pain of infiltration.
4. Inject the local anesthetic with the smallest diameter needle. A 27- or 30-gauge needle is preferred.
5. Slow the rate of medication injection, as pain results when the soft tissue stretches.

Regional block

An elegant technique in wound anesthesia is a regional nerve block, in which anesthesia is administered proximal to the wound site to block sensory perception in the affected area. The primary advantage of this approach is the preservation of wound edges. Comparatively, local anesthetic infiltration often distorts landmarks. This is especially crucial when wound edges are under tension or poorly approximated. Commonly used regional blocks include the digital block; supraorbital, infraorbital and mental nerve blocks; median, ulnar and radial nerve blocks at the wrist or elbow; and lower extremity nerve blocks of the saphenous, deep peroneal, superficial peroneal, posterior tibial and sural nerves. The key to successful regional anesthesia is familiarity with the anatomy and proper technique.

The digital block anesthetizes an entire digit distal to the metacarpal–phalangeal or metatarsal–phalangeal joint. Each finger has a medial and lateral digital nerve, located along the ulnar and radial volar (palmar) aspect of the digit, respectively. Each toe has a similar medial and lateral digital nerve located along the plantar aspect of the digit. In cross-section, these nerves lie at approximately the 4 and 8 o'clock positions of a digit, with the surface of the fingernail or toenail at 12 o'clock. As a consequence, two injections are required for adequate anesthesia. First, after positioning the patient's hand or foot on a flat surface with the dorsal aspect facing up, insert the needle at the level of the web space. Insert the needle at a 45-degree angle aiming volarly along the proximal phalanx to reach the first digital nerve. After aspirating to check for inadvertent vascular cannulation, slowly inject 1–2 mL of the anesthetic. Remember to avoid using epinephrine. Repeat this process for the opposite digital nerve. For the thumb and great toe, deposit a subcutaneous line of anesthesia to block the superficial branches supplying the dorsal part of the digit (Figure C.3).

Procedural sedation

Although the topic of procedural sedation is beyond the scope of this section (see Appedix D), it provides another option for wound closure in patients with extensive wounds or anxiety. Common agents include propofol, methohexital, etomidate, ketamine, or a combination of fentanyl and midazolam. While the patient is sedated, a local or regional anesthetic may be administered for pain relief (especially after the patient awakens). Vigilant monitoring of vital signs and cardiorespiratory status is critical throughout the procedure and recovery period.

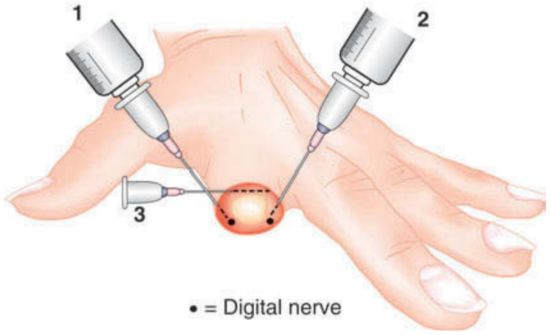


Figure C.3
Regional anesthesia: digital nerve block. A digital nerve block can be achieved with injections at sites 1 and 2. For the thumb and great toe, a third subcutaneous line of anesthesia (site 3) is necessary to block superficial dorsal sensory nerves.

Despite improved cosmesis from patient immobilization and decreased psychologic trauma, procedural sedation is not without risks. One disadvantage is a longer ED stay for the patient, because the patient must have had nothing by mouth for 4–6 hours prior to sedation, and usually requires time for recovery following sedation for the procedure. Depending on the procedural sedation agent used, risks include agitation, vomiting, respiratory depression and apnea.

Wound cleaning

Thorough cleansing of the wound prior to wound closure reduces the incidence of infection. Although a variety of irrigation solutions have been studied, the current standard is sterile normal saline with high-pressure irrigation. The type of solution used for irrigation may not be important; several studies have shown equal efficacy using tap water versus sterile normal saline. However, high-pressure irrigation is the cornerstone of wound cleaning. By applying at least a 7-psi force to the wound, the irrigation fluid dislodges foreign bodies, contaminants and bacteria. Although many commercially available kits can provide high-pressure irrigation, a simple device can be made by using a 30-mL syringe and an 18-gauge angiocatheter. Irrigation with this setup delivers >7 psi of pressure (Figure C.4). Low-pressure irrigation, such as squeezing fluid out of a puncture hole made in a saline bottle, is inadequate. Although no studies have looked at the ideal irrigation volume, a common practice is to irrigate with about 100 mL for every 1 cm of wound length. Slightly more irrigant may be used for distal extremity or contaminated wounds, which have a higher incidence of infection, and slightly less for facial and scalp wounds, which have a much lower incidence. Hollander et al. suggests that facial and scalp wounds require minimal, if any, irrigation prior to wound closure because the high vascularity of these areas significantly reduces the incidence of wound infection; an added benefit was improved cosmesis as high-pressure irrigation can cause unnecessary soft tissue swelling.

There are two common misconceptions in wound cleaning. The first is that swabbing povidone–iodine

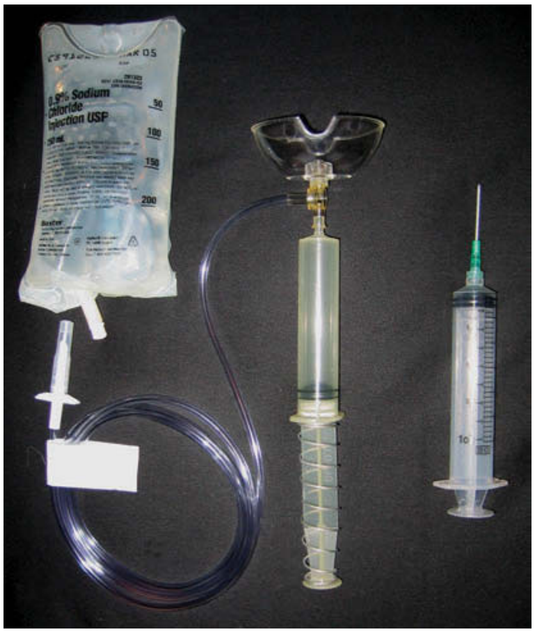


Figure C.4
Wound irrigation equipment. A commercially available unit (middle) or a syringe with an 18-gauge angiocatheter (right) can deliver high-pressure irrigation to a wound.

solution or chlorhexidine into the wound is required for further sterilization. Studies have shown that although these agents impede bacterial growth, they are also cytotoxic and impair wound defenses. When using these agents, be sure to swab only the intact skin adjacent to the wound but not the wound itself. A second myth is to scrub the wound vigorously before wound closure. This practice injures underlying viable soft tissue and impairs optimal wound healing. Unless there are multiple small foreign bodies (e.g., gravel) embedded in the skin, scrubbing is not recommended.

Challenges in local wound preparation

After anesthesia and irrigation, the wound should be examined under sterile conditions to reduce the risk of wound infection. Careful wound exploration should check for the presence of foreign bodies and involvement of more complex structures, such as tendon, muscle, joint capsule and bone.

Three problems commonly arise during wound preparation. The first is bleeding, especially after wound irrigation. Early clots may become dislodged, and ongoing venous or arterial bleeding can prevent adequate visualization of the wound. Hemostasis can usually be achieved by applying direct external pressure for several minutes. Persistent bleeding despite direct pressure can be addressed by applying a tourniquet on an extremity proximal to the wound for no more than 60 minutes. The cuff pressure should be above the patient's systolic blood pressure. Patients often will not tolerate this tight tourniquet for more than 20–30 minutes. Application of an ice pack may aid in vasoconstriction and produce hemostasis. Additionally, wounds that ooze slowly can be washed over with an anesthetic containing epinephrine to achieve transient vasoconstrictive hemostasis before wound closure. Following thorough exploration, wound repair and closure itself will generally stop further bleeding.

Another common problem with wound preparation is the proximity of hair to the wound. If hair strands become trapped in the wound during closure, a foreign body reaction may ensue. Do not shave hair, because this increases the rate of wound infection by providing a portal for bacterial entry. Eyebrow hair should never be cut or shaved because it grows slowly and irregularly after being cut, causing cosmetic asymmetry. Furthermore, eyebrows assist with wound edge approximation by serving as a landmark for skin edges. Instead, for lacerations near hair, apply a thin coat of a sterile petroleum-based jelly in the hair, such as neosporin or bacitracin, and mat the hair away from the wound edges.

A third problem frequently encountered in wound preparation is the presence of devitalized tissue along a wound edge. Non-viable tissue along wound edges impairs wound healing and can provide a nidus for infection, as the usual mechanism for intrinsic wound repair cannot take place without adequate blood supply to the wound edges. Excisional debridement of devitalized tissue allows more precise reapproximation of wound edges. Be careful of over-debridement, which may create excessive wound tension upon closure. Debridement near tendons and peripheral nerves may cause iatrogenic injuries.

Wound closure

There are multiple methods available to repair lacerations. The most commonly used method is wound edge reapproximation by suturing.

Suturing

Basic suturing supplies consist of needle drivers, tissue forceps (or skin hooks), scissors, sterile drapes, sterile gloves, suture materials (Table C.8), and sterile gauze.

Numerous methods of suturing can be used to repair a laceration. Some approaches have specific advantages that may benefit certain laceration types. To begin the suturing process, hold the needle driver in the palm of the dominant hand and grasp the needle. Extend the index finger along the arms of the closed needle driver, such that it points toward the needle. The palm may be used for opening and closing the needle driver's locking mechanism (Figure C.5). Holding the needle driver instrument in this fashion allows optimal needle rotation through the wound. An alternate method is to place the tips of the thumb and ring digits into the rings of the handle. The needle should always enter and exit at 90 degrees to the skin surface to minimize tissue damage. This maximizes wound edge eversion, which is necessary for vertical tissue alignment along dermal layers to optimize wound cosmesis.

Table C.8 Types of sutures and their characteristics

Type	Subtype	Strength	Reactivity	Infection risk	Comment
Absorbable	Chromic gut	+ +	+ + +	+	Often used for intraoral repairs
	Monofilament	+ + + +	+	+	Used for most external closures
	Braided co-polymer	+ + + +	+	+ + +	Useful for deep sutures requiring strength and absorbability
Non-absorbable	Silk	+	+ + + +	+ +	Very reactive substance with few applications in ED
	Monofilament	+ + +	+	+	Use for most external closures
Suture size	**Anatomic location**				
6–0	Face				
5–0	Hands				
4–0	Most extremity, scalp, and trunk wounds				
3–0	Large wounds in non-cosmetic areas (not used in routine wound repair)				
2–0 and larger	Emergent, life-saving closures (e.g., post-thoracotomy, scalping injury)				
Staples	Scalp, non-cosmetic areas				
Needle type	**Characteristics and applications**				
Conventional cutting	Sharp point; useful for skin				
Reverse cutting	Extra sharp point; minimal tissue trauma, useful for skin, tendons				
Taper (round edged)	Pierces tissue; useful in peritoneum, viscera, myocardium				
3/8-circle shape	Most common for routine repairs				
Half-circle shape	Confined spaces (oral, nasal cavity, fascia)				

Simple interrupted suture

The simple interrupted suture is the most frequently used technique in wound approximation (Figure C.6). With the needle at a 90-degree angle to the skin surface, penetrate the dermis. Drive the needle through to the opposing side of the laceration, using extreme care to meticulously approximate each level of tissue. This can be accomplished in one or two actions (bites) to ensure constant visualization of underlying tissue and control of the needle. To secure the suture, perform an instrument

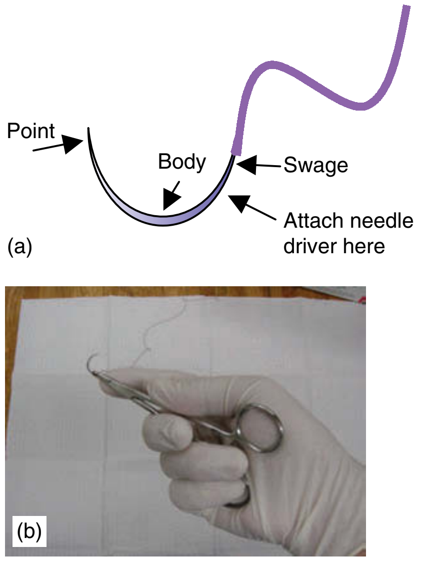


Figure C.5
(a) The needle is grasped at the proximal one-third of the body.
(b) The needle driver rests in the palm of the hand.

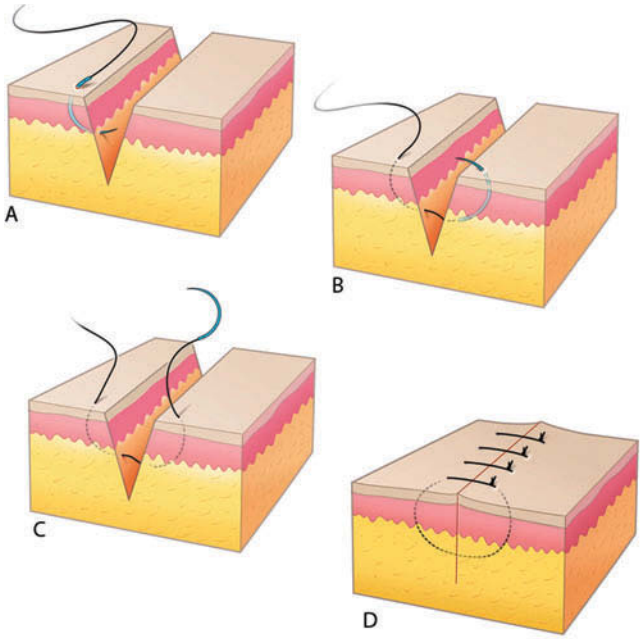


Figure C.6
Sequence illustrating the placement of a simple interrupted suture. © Chris Gralapp.

tie with a surgeon's knot (a flat double throw) followed by single throws tied sequentially in opposite directions (Figure C.7).

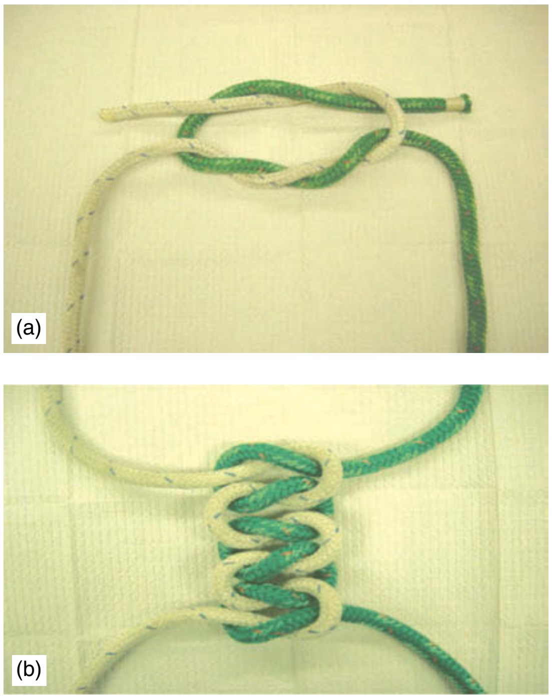


Figure C.7
(a) Initial knot used to secure a suture. Notice that the initial knot has two throws on the bottom and a single throw on top. This facilitates keeping tension in the suture in order to keep the wound margins together. (b) Picture illustrating sequential knots with throws in opposite directions.

Five knots are needed to secure nylon sutures, whereas three knots are generally adequate for braided, soft sutures. Wound edges should be gently approximated (not strangulated). When each knot is finished, gently retract it to one side of the wound. This prevents the knot from serving as a nidus for contamination, impinging on the healing wound tissue, and making an unsightly divot in the final scar (Figure C.8). Suture placement should be symmetric. Cosmetically important areas may require a smaller suture interval for better appearance. There should be a consistent relationship between needle entry point and suture interval.

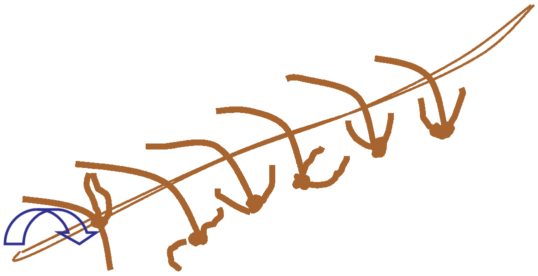


Figure C.8
Placing all suture knots off the wound and on one side improves cosmesis and facilitates suture removal.

Horizontal mattress suture

The horizontal mattress suture is used to close a wound under mild to moderate tension. It is an especially useful alternative to two-layer closure in a patient who is at high risk for developing a wound infection. Its proper placement disperses tensile forces over a larger area, providing better perfusion of healing wound edges (Figure C.9). Note that this suture is not indicated in cosmetically important areas, such as the face. This suture works especially well on the palms and soles of the feet.

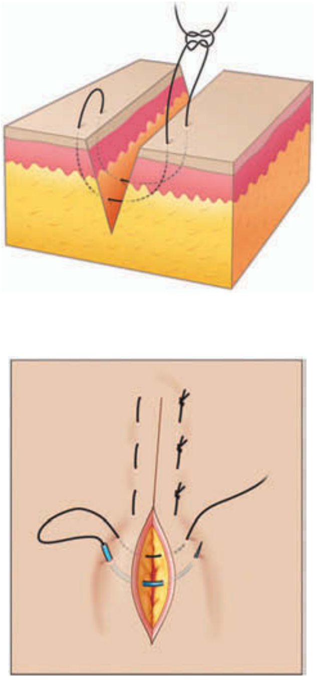


Figure C.9
Horizontal mattress sutures alleviate some of the "strangulation" effect of placing simple interrupted sutures along the margin of a wound under moderate tension. © Chris Gralapp.

Deep (intradermal) suture

For a laceration of substantial depth and/or tension, the deeper tissues should be approximated. This helps relieve tension along the upper wound margins and improves healing and cosmesis. Placing absorbable sutures in the deeper structures requires "burying the knot" by entering the wound margin deeply on one side and reversing needle entry on the opposite side. Tying the knot as shown in Figure C.10 places the knot at the bottom of the laceration and prevents it from disrupting the surface appearance by "spitting" through the wound site. A superficial layer of closure can then be applied to re-approximate the epidermis. This can be done with simple interrupted sutures, wound approximation strips, or tissue adhesives. Placing a deep layer of absorbable sutures may increase the wound infection rate, especially in patients with dirty wounds (human or animal bites) or underlying medical conditions, such as diabetes mellitus or poor circulation.

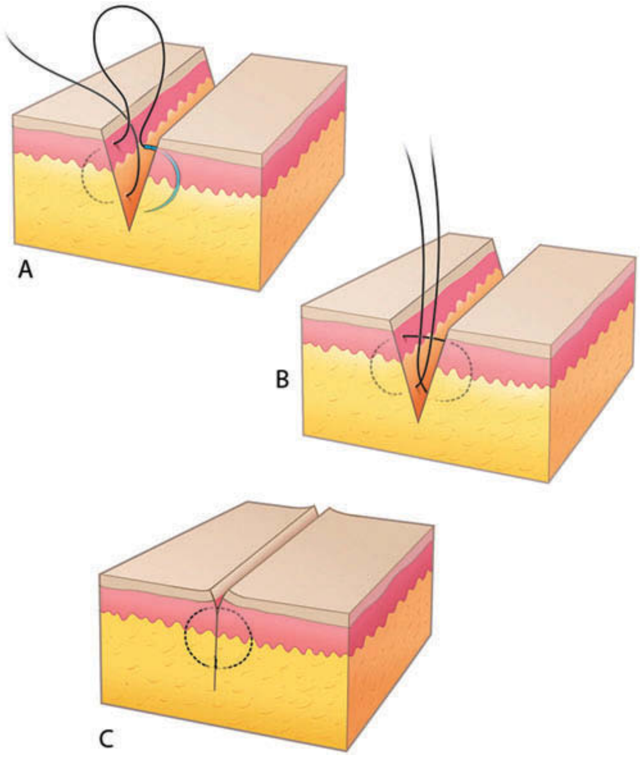


Figure C.10
Placing intradermal sutures increases the risk of infection in a wound. However, deep sutures can remove "potential" spaces where fluid collection may distort tissue anatomy and/or predispose to infection. In addition, deep sutures facilitate closure of wounds that are widely open at the surface. This sequence demonstrates burying the knot. © Chris Gralapp.

Corner suture

"V"-shaped lacerations consist of a small apical flap of tissue that can be friable and poorly perfused. Placing sutures directly through this fragile tissue flap may cause necrosis and a poor cosmetic outcome. The half-buried mattress suture can help preserve distal perfusion and spreads tension along the distal flap. Care must be taken to assure that the suture is at the same tissue depth on each side of the laceration (Figure C.11).

Staples

Some wounds have less cosmetic importance and require rapid closure. As a definitive or temporizing measure, stapling a laceration is an effective means for closing such wounds. Wounds appropriate for staples are linear, without ragged edges, and under minimal tension (edges need to align well without the use of tissue forceps when working alone). Although staples are most often used for scalp lacerations in the ED, it is imperative to remember that most men will have some balding, and a scar, which was initially covered by hair, may become a cosmetic problem with a receding hairline. To place staples, the stapling device is placed at the skin surface, and the staple is brought forward with a smooth motion using the trigger mechanism (Figure C.12). It is important not to indent the skin surface as this may cause improper vertical alignment of wound edges. Timely removal of staples will improve cosmetic outcome because staples are larger in diameter than most sutures. Commercial staple removers should be used to properly (and painlessly) remove staples.

Tissue adhesives

Tissue adhesives have recently emerged as an effective approach for laceration repair. Lacerations appropriate for tissue adhesives are clean, linear, and under little or no tension. Wounds are assessed and prepared in the same way as any other wound prior to closure. Impeccable hemostasis and dry overlying skin are required for successful closure. Wound edges are held together with a commercially available device or finger tips as the liquid adhesive is stroked across the tissue defect, taking care to avoid getting adhesive within the wound or on the skin approximation device. Extreme caution should be taken if tissue adhesives are used around the eyes, to prevent corneal or lid adhesion. Tissue adhesives are ineffective and should not be applied on mucous membranes.

After tissue adhesive application, there is no need for a wound dressing. The wound should be kept dry; ointment (including antibacterial) should not be used because it may dissolve the adhesive and cause the wound to prematurely dehisce. The use of these ointments can be helpful in removing unintended applications of tissue adhesive. In particular, ophthalmic ointments can aid in removing tissue adhesive from the eyes. Routine evaluation and treatment for corneal abrasions should follow if this occurs.

Tissue adhesives are well tolerated by pediatric patients. They provide a painless alternative to suturing.

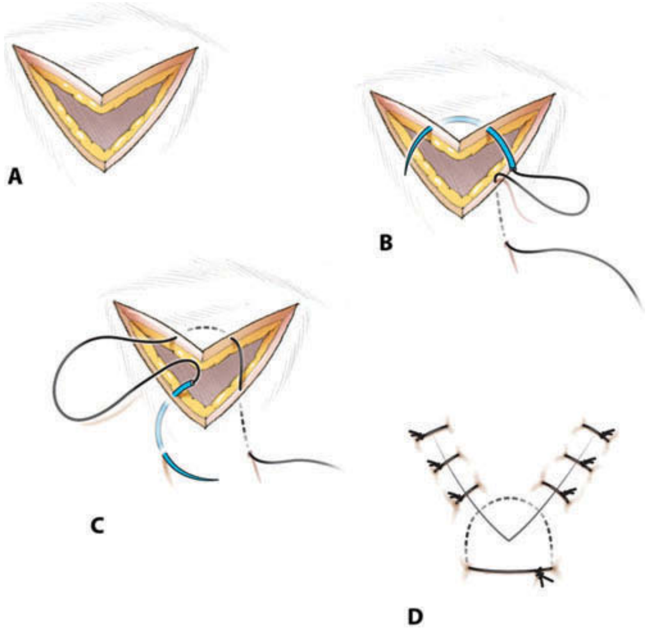


Figure C.11
Corner sutures with a half-buried mattress stitch may avoid necrosis of the distal tip of the flap. © Chris Gralapp.

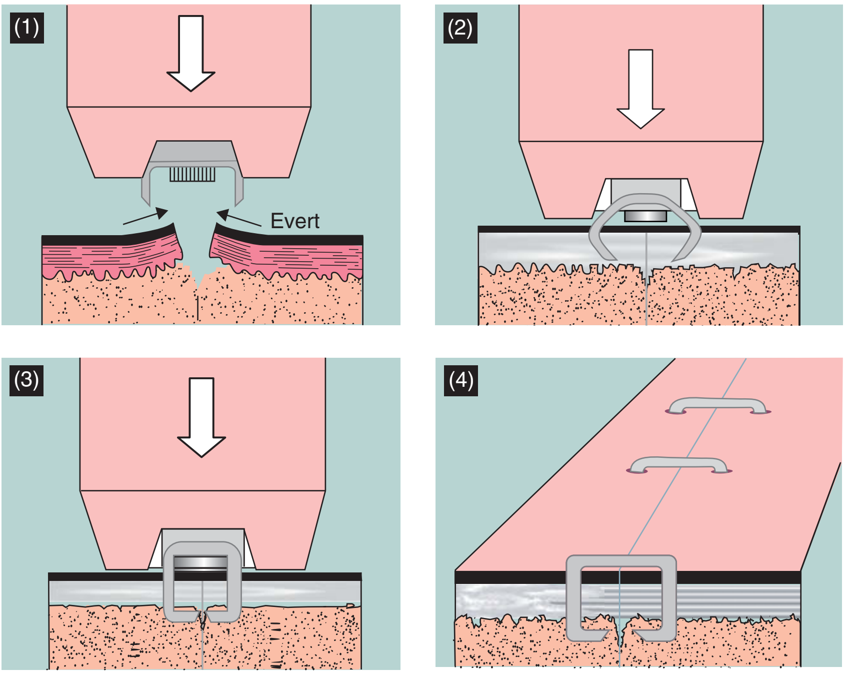


Figure C.12
A sequence illustrating wound stapling.

Some patients experience a warm sensation as the bonding process occurs. Choosing a topical anesthetic with vasoconstrictive properties is appropriate for reduction of pain when using this modality.

Wound taping (butterfly closure)

For this approach, the wound is assessed and prepared in the usual manner. Wounds appropriate for this technique are linear or curvilinear, clean and under minimal tension. Pediatric patients tend to pick, soak or pull at wound adhesive tape, which may cause a wound to prematurely dehisce. Taping can be used in conjunction with a layer of deep sutures in order to keep the overlying skin closely approximated. Taping may also be used prior to the application of tissue adhesives.

Prior to placing tape, wound edges should be cleaned and tincture of benzoin adhesive applied to both sides of the wound margin (taking care not to place inside the wound). After several seconds of air drying, adhesive strips are placed at appropriate intervals to approximate the skin edges. One part of a laceration may require more strips than another part (Figure C.13). Wound adhesive strips generally fall off within a week of application. Advise patients to avoid getting the wounds and adhesive tapes wet to prevent the tape from falling off prematurely.

Wound care and patient disposition

Continued care after wound closure plays an integral role in optimal wound healing. In order to prevent contamination and limit scab formation, apply a dressing consisting of a topical antibiotic ointment underneath a dry sterile gauze. For tissue adhesives, petroleum-based products should not be applied because they can degrade the adhesive. When applying a circumferential wound dressing,

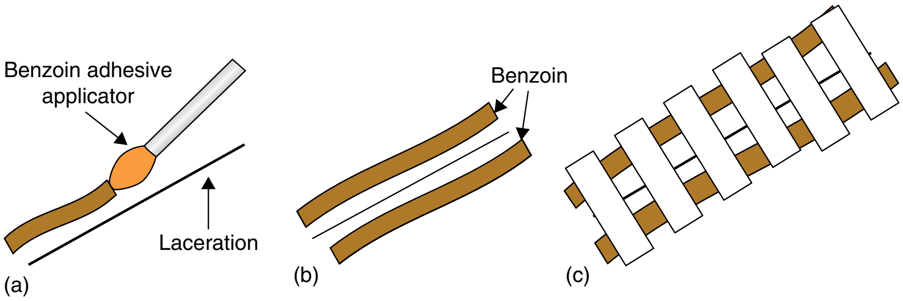


Figure C.13
(a) The application of Benzoin next to the laceration. (b) Benzoin has been applied as an adjunct for the tape strips. (c) Tape strips placed in a symmetric fashion to approximate the wound edges.

wrap the site loosely to avoid a tourniquet-like effect from inevitable soft tissue swelling. For wounds overlying joint surfaces, which may dehisce when the joint is flexed or extended, a splint should be applied to prevent range of motion of the joint.

Antibiotics

Clinicians should not prescribe prophylactic systemic antibiotics for simple lacerations and wounds. The primary means of reducing wound infection is sterile irrigation, scrupulous wound exploration, and meticulous wound closure, not antibiotics. However, antibiotics may be indicated in special high-risk circumstances (Table C.9).

Table C.9 Indications for systemic antibiotics for traumatic wounds

Injury >12 hours old on the extremities
Injury >24 hours old on the face and scalp
Cartilage involvement
Comorbid disease (diabetes mellitus, extremes of age, steroid use, morbid obesity)
Complex intraoral wound
Mammalian bite
Puncture wound
Tendon, joint or bony involvement

Suture removal

The duration of suture placement and wound tension are proportional to increased scarring. However, sutures must remain long enough to allow the healing process to begin. Cosmetically important areas, such as the face, should have sutures in place for a shorter amount of time to reduce scarring. Table C.10 lists guidelines on the timing of suture removal based on anatomic location.

Table C.10 Suture removal guidelines (approximate)

Anatomic location	Days (average)
Face	3–5
Trunk	7–10
Arm (not over the joint)	7–10
Leg	10–14
Joint	14
Scalp	10–14

Discharge instructions

Discharge paperwork for the patient should include explicit instructions on wound care management, as well as specific comments about the possibility of wound infection (Table C.11). For the wound at higher risk of infection (e.g., a contaminated laceration of a finger, a human bite of an extremity, or a traumatic wound with devitalized tissue), a scheduled wound check within 48 hours is prudent to look for early signs of infection.

Table C.11 Sample patient discharge instructions

Keep your wound elevated above the level of your heart to reduce tissue swelling.
For sutures: Keep the wound dressing on and dry for 24 hours to prevent wound contamination. After that, change the dressing using dry gauze or a bandage. Gently clean the wound edges with mild soap and water. Keep the wound covered and dry as much as possible.
For tissue adhesives: Keep the tissue adhesive completely dry until it spontaneously peels off in 7–10 days. Do not apply any topical ointment.
Follow-up with your primary care physician or a wound clinic in ________ days for suture removal.
Return to the ED if you experience a fever (temperature > 100.4°F), or your wound becomes red around the edges, breaks open, or releases pus. Your wound may be infected and require antibiotics.
After your sutures are removed (or your tissue adhesive falls off), the wound is not completely healed. Therefore, treat it gently. Apply sunscreen to sun-exposed wounds to prevent increased pigment uptake and darker scarring as a result of ultraviolet light. Continue this practice for the next 6–12 months.

Pearls, pitfalls and myths

- Although wound irrigation may be time-consuming, it plays a significant role in minimizing infection.
- Meticulous wound exploration should occur before wound closure.
- Regional blocks are vastly underutilized in providing anesthesia prior to wound closure. When compared with local infiltration of anesthesia, these offer the significant advantages of preserving landmarks and not increasing wound tension.
- Do not to shave hair adjacent to a wound. Instead, splay hair tufts away from the wound edges using a sterile petroleum-based product, or gently trim hair at the level of the skin.
- Povidone–iodine and chlorhexidine should only be applied to intact skin. If applied into the wound itself, these may cause tissue damage and delay wound healing.
- Foreign bodies should be considered prior to wound closure.
- Devitalized tissue should be debrided conservatively.
- To optimize wound cosmesis, wound edges should be approximated meticulously with gentle eversion of the edges.
- Careful attention to proper technique makes a good cosmetic outcome more likely. The dermis provides the strength necessary for healing.
- Tissue adhesives are excellent alternatives for repairing clean wounds that are neither bleeding nor under tension.
- Routine use of antibiotics in uncomplicated wounds is not necessary with adequate wound preparation.
- Careful discharge instructions should warn about the possibility of wound infection, and describe methods to decrease (not eliminate) scarring. These should also

provide clear instructions regarding wound care and all necessary return visits.
- Tetanus status should be updated according to current guidelines in all patients sustaining wounds or lacerations.

Acknowledgment

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Appendix D Procedural sedation and analgesia

Eustacia (Jo) Su, MD

Scope of the problem

Procedural sedation and analgesia (PSA) represents one of the great advances in emergency medicine's maturation as a specialty. It has become a routine part of our practice, encompassing fundamental clinical skills such as airway assessment and management, and critical resuscitation. PSA is very safe when performed by a properly trained practitioner in the correct setting, with appropriate nursing support, monitoring and resuscitation equipment.

Older terminology attempted to describe the state of sedation and analgesia in static terms, such as *conscious sedation*. This definition required that patients given agents providing both sedation and analgesia remained conscious, and were still able to reflexively protect their airways. The terminology has evolved to reflect the continuum upon which sedation and analgesia occur, ranging from *minimal sedation* (anxiolysis) to *moderate sedation* (analgesia and light sedation), through *deep sedation* and *general anesthesia* (Table D.1).

All of these levels may be applied to the emergency department (ED) setting with the exception of general anesthesia; this implies a complete loss of consciousness and protective airway reflexes.

Many ED procedures and studies commonly require PSA (Table D.2).

General treatment principles

First, consider alternatives to the administration of drugs. The toddler who needs a head computed tomography (CT) may stay still enough if his caregiver dons lead protection and comforts him during the the brief time required for the procedure. Oral sucrose analgesia may provide adequate analgesia for brief procedures in young infants and neonates. Bier blocks, regional anesthesia, nerve blocks and intra-articular injection of an anesthetic agent may obviate the need for deep sedation.

The major considerations in selecting drugs for PSA include depth of sedation, anticipated painfulness of the procedure, need for amnesia and/or muscle relaxation, and duration of the procedure. The first step is to determine the desired depth of sedation. A toddler who has sustained a minor head injury and is undergoing a head CT requires much less sedation (if any) and essentially no analgesia as compared with a child who is having a burn debrided or a long bone fracture reduced.

Agents should be selected that provide adequate analgesia. Most sedatives provide little, if any, analgesia. Although many analgesics provide some sedation, the dosing necessary to achieve an adequate level of sedation may be associated with significant respiratory depression.

Procedure duration also influences drug choice and route of delivery. Debate exists regarding whether patients who are deeply sedated (without analgesia) and amnestic to a brief procedure suffer the same physiologic consequences as patients who remember the pain.

Individuals respond differently to drugs, even when the dose is calculated according to their weight. Careful titration (considering the pharmacokinetics of the agent(s) used) produces the optimal response. This is best done using intravenous (IV) administration, since repeated oral doses are unpredictable and usually slow to onset, and repeated intramuscular (IM) injections are not appropriate.

Table D.1 Continuum of depth of sedation: Definitions of general anesthesia and levels of sedation (American Society of Anesthesiologists)

	Minimal sedation (anxiolysis)	Moderate sedation (conscious sedation)	Deep sedation	General anesthesia[a]
Responsiveness	Normal response to verbal stimulation	Purposeful response[b] to verbal or tactile stimulation	Purposeful response[b] following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

[a] *Monitored anesthesia care* does not describe the continuum of depth of sedation; it describes "a specific anesthesia service in which an anesthesiologist has been requested to participate in the care of a patient undergoing a diagnostic or therapeutic procedure."

[b] Purposeful response does not include reflex withdrawal from a painful stimulus.

Table D.2 Indications for procedural sedation and analgesia

- Incision and drainage of a large abscess
- Wound debridement
- Reduction of a fracture, dislocation, or prolapsed viscera (hernia)
- Tube thoracostomy
- Repair of a complicated laceration, especially in children
- Vascular procedure (e.g., central line placement, difficult IV access)
- Diagnostic imaging (CT or MRI)
- Lumbar puncture (in selected cases)
- Cardioversion
- Removal of an embedded foreign body
- Painful or anxiety-inducing procedures (e.g., pelvic examination in a young rape victim)
- Procedures in selected patient populations (e.g., psychiatric, mentally challenged, special health needs)

CT: computed tomography; IV: intravenous; MRI: magnetic resonance imaging.

Patient assessment and selection

If a patient needs a procedure that requires sedation and/or analgesia, first determine whether the patient is able to tolerate PSA (Table D.3). Candidates for procedural sedation in the ED *must* fall into either American Society of Anesthesiologists (ASA) class I or II. Patients with higher classifications are better served in a more controlled environment, such as the operating room (OR) or intensive care unit (ICU).

Next, select a target level of sedation and the duration required. Determine the drug and dosage range that will most likely produce the desired sedation and pain control for the duration of the procedure.

Table D.3 American Society of Anesthesiologists' (ASA) classifications for risk stratification

Class	Patient status
I	Normally healthy patient. The pathologic process for which the procedure is to be performed is localized and not a systemic disturbance.
II	Mild systemic disease under control (e.g., asthma).
III	Severe systemic disease from any cause.
IV	Severe systemic disease that is a constant life-threat, not always correctable by the operative procedure.
V	Moribund patient who is not expected to survive without the operation.

The next risk factor to consider is time since the patient's last oral intake. Loss of protective airway reflexes increases the risk of aspiration of stomach contents. Gastric emptying time is variable, even among patients of similar ages. The American Academy of Pediatrics (AAP), American College of Emergency Physicians (ACEP) and the American Society of Anesthesiologists (ASA) all have separate guidelines for nil per os (NPO) times, ranging from 4–6 hours. Some distinctions are made between NPO times depending on whether the food consumed was solid, full liquid or clear liquid, with solid food consumption requiring longer NPO times prior to sedation. It is important to remember that preservation of life or limb takes precedence over NPO status. NPO status for young children and toddlers is slightly more complex (Table D.4).

Table D.4 Recommended NPO status for children and adults

Children <6 months old
- 2-hr fast for clear liquids
- 4-hr fast for milk, solids

Children 6 months–3 years
- 3-hr fast for clear liquids
- 6-hr fast for milk, solids

Children >3 years old
- 3-hr fast for clear liquids
- 6–8-hr fast for milk, solids

Adults
- 2-hr fast for clear liquids
- 6-hr fast for milk, solids

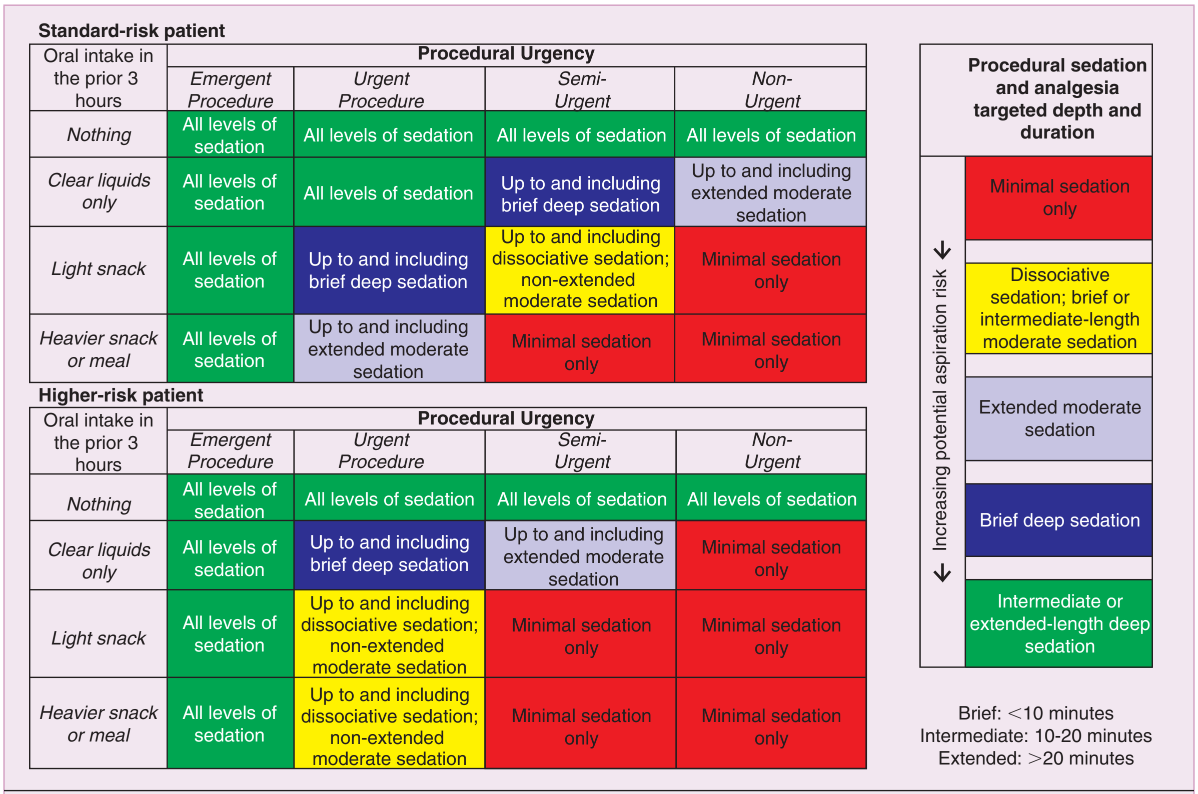
Green et al. have introduced a "consensus-based clinical practice advisory" for the non-fasted patient that is more clinically relevant to emergency medicine practice (Table D.5). This clinical tool risk stratifies patients based on four key clinical factors:

1. Patient risk
2. Timing and nature of recent oral intake
3. Urgency of the procedure
4. Prudent limit of targeted depth and length of PSA

A pre-sedation history and physical examination, beyond a general medical screening examination, should focus on issues pertaining directly to the sedation procedure. The history should elicit factors that might increase the direct risks of medications and symptoms, such as upper respiratory tract illness or abnormality, including asthma exacerbation or viral syndrome. Patients must also be asked about allergies, prior experiences with sedation or general anesthesia, and time of last food or liquid intake.

The physical examination should focus on baseline vital signs, the oral cavity, and the cardiorespiratory system. In particular, any potential impediments to endotracheal

Table D.5 Prudent limits and targeted length and depth of ED procedural sedation and analgesia according to pre-sedation assessment of aspiration risk



Standard-risk patient

Oral intake in the prior 3 hours	Procedural Urgency			
	Emergent Procedure	*Urgent Procedure*	*Semi-Urgent*	*Non-Urgent*
Nothing	All levels of sedation	All levels of sedation	All levels of sedation	All levels of sedation
Clear liquids only	All levels of sedation	All levels of sedation	Up to and including brief deep sedation	Up to and including extended moderate sedation
Light snack	All levels of sedation	Up to and including brief deep sedation	Up to and including dissociative sedation; non-extended moderate sedation	Minimal sedation only
Heavier snack or meal	All levels of sedation	Up to and including extended moderate sedation	Minimal sedation only	Minimal sedation only

Higher-risk patient

Oral intake in the prior 3 hours	Procedural Urgency			
	Emergent Procedure	*Urgent Procedure*	*Semi-Urgent*	*Non-Urgent*
Nothing	All levels of sedation	All levels of sedation	All levels of sedation	All levels of sedation
Clear liquids only	All levels of sedation	Up to and including brief deep sedation	Up to and including extended moderate sedation	Minimal sedation only
Light snack	All levels of sedation	Up to and including dissociative sedation; non-extended moderate sedation	Minimal sedation only	Minimal sedation only
Heavier snack or meal	All levels of sedation	Up to and including dissociative sedation; non-extended moderate sedation	Minimal sedation only	Minimal sedation only

Procedural sedation and analgesia targeted depth and duration

Increasing potential aspiration risk

- Minimal sedation only
- Dissociative sedation; brief or intermediate-length moderate sedation
- Extended moderate sedation
- Brief deep sedation
- Intermediate or extended-length deep sedation

Brief: <10 minutes
Intermediate: 10-20 minutes
Extended: >20 minutes

Reprinted with permission from Green SM, Roback MG, Miner JR et al. Fasting and emergency department procedural sedation and analgesia: A consensus-based clinical practice advisory. *Ann Emerg Med* 2007;49:454–61.

intubation and ventilation should be noted (Chapter 2). Underlying reactive airway disease or upper respiratory tract infection should be ruled out with a careful examination of the pulmonary system. If the patient has an upper respiratory tract infection, the risk of developing laryngospasm is increased by PSA agents. The heart should be evaluated for the presence of ischemia, murmurs, or dysrhythmias.

Drug selection

Consider the following variables when choosing PSA agents for a particular patient and procedure: patient comfort, how still the patient has to remain, degree of procedural invasiveness, degree of muscle relaxation, amount of pain and duration of procedure. The ideal drug for PSA would have predictable dosing, instantaneous onset, easily adjustable duration of action, short recovery time, and both amnestic and analgesic properties. It would also have minimal hemodynamic effects, no respiratory depression, and would preserve protective airway reflexes (Table D.6).

The combination of a benzodiazepine (midazolam or diazepam) and a narcotic (morphine or fentanyl) has been a mainstay of procedural sedation. The combination of these agents has been applied widely across many procedures performed by emergency physicians. Important side effects include respiratory depression and hypotension. One recommendation has been to give the narcotic agent (which poses the greater risk of respiratory depression) first, and then titrate the benzodiazepine. Table D.7 provides a list of reversal agents, should they be needed.

Ketamine, etomidate and propofol have become the PSA agents of choice in the ED because they have rapid onset and a short recovery time. Compared with the benzodiazepine–narcotic combination, these agents provide deeper sedation or dissociation with less respiratory depression and shorter recovery times. Their rapid onset also allows for much better titration.

Propofol and etomidate do not possess analgesic properties, so an analgesic should be co-administered for painful procedures. Be vigilant, however, as this combination increases the risk of respiratory depression. A recent study showed that patients recovering from propofol-only sedation did not recall or report any pain immediately afterward, questioning the true physiologic impact of brief, unrecalled pain. The combination of propofol and ketamine does not seem to offer a significant advantage over either agent alone.

Table D.6 Procedural sedation and analgesia medications

Medication	Recommended dosage	Route	Onset	Duration	Additional instructions/mode of action	Precautions/contraindications
Etomidate	0.1–0.3 mg/kg	IV over period of 30 sec	60 sec	10 min	Considered a general anesthetic. Cardiac, respiratory and blood pressure monitoring required. Question of adrenal suppression (probably clinically insignificant). Not approved for children <12 years of age, although, much evidence supports its safe use in children <12.	May cause significant myoclonic jerking. Also pain at injection site. Avoid if seizure disorder, nausea, vomiting.
Fentanyl	1–3 mcg/kg 1–2 mcg/kg	IV IM	1–2 min 7–15 min	Peak 10-min 30–45 min	Potent rapidly acting analgesic. Hemodynamically stable but may cause respiratory depression and apnea. Dose can be titrated. Maximum dose 5 mcg/kg. Synergistic action with concomitant benzodiazepine administration.	Push and flush slowly; monitor closely for respiratory depression, bradycardia, apnea. *Rigid chest syndrome* may be associated with large doses administered rapidly; appears to be an idiosyncratic reaction. May not be reversible with naloxone; paralytics may be required to ventilate.
Ketamine	1–2 mg/kg (repeat 1 mg/kg as needed every 20 min) 2–5 mg/kg	IV IM	1–3 min 2–3 min	10–20 min 30–60 min	Dissociative agent provides analgesia, anxiolysis and amnesia. Dissociation is a binary process (patients either are or are not dissociated) and not a dose-dependent phenomenon (patients are not more dissociated with increasing doses). Use with benzodiazepine potentially reduces emergence reactions. May be combined with glycopyrrolate 0.01 mg/kg or atropine 0.01 mg/kg to decrease secretions. Combine with anti-sialagogue in single syringe for single IM injection. Positive cardiac inotrope.	Hallucinatory emergence reactions common in adults but uncommon in children. Potential for laryngospasm (1 in 250) so avoid in patients with active pulmonary or upper respiratory tract infection. Also avoid for intraoral procedures; consider alternative drugs in patients with increased intraocular or intracranial pressure.
Methohexital (Brevital)	1 mg/kg 25 mg/kg	IV PR	1 min 2–5 min	10 min 45 min	Ultra-short barbiturate. Provides significant muscle relaxation. May cause hemodynamic instability, apnea.	Discontinue immediately if extravasation occurs to minimize tissue necrosis. If given intra-arterially, thrombosis and gangrene possible. Contraindicated in severe hepatic dysfunction or porphyria.
Midazolam (Versed)	0.05–0.1 mg/kg *Suggested pediatrics combination: 0.1 mg/kg IV when used with 1–2 mcg/kg IV fentanyl*	IV 0.1 mg/kg IM	1–3 min 2–5 min	1 hour 1–2 hours	Potent amnestic and muscle relaxant. No analgesic property. May repeat IV dose 0.1 mg/kg by giving increments of 0.05 mg/kg until adequately sedated. Must be given slowly over 30 seconds. With IM route use 5 mg/mL concentration to reduce volume.	Can cause respiratory depression if given rapidly or in addition to barbiturate therapy. Paradoxical hyperactivity possible. Will obtain faster onset with higher doses. PO route useful for anxiolysis. PR and intranasal (atomized) routes advocated by some.

(*continued*)

Table D.6 Procedural sedation and analgesia medications (*cont.*)

Medication	Recommended dosage	Route	Onset	Duration	Additional instructions/mode of action	Precautions/contraindications
Morphine sulfate	0.1 mg/kg	IV/IM	3 min peak effect	15–30 min	Opioid narcotic. Successful use dependent on appropriate dosing. Titration >0.1mg/kg/dose may be required. Extremely painful injuries (e.g., femur fractures) require judicious titration to achieve appropriate levels of pain relief. Dosing in infants 0.05 mg/kg due to decreased hepatic clearance.	Respiratory depression, apnea, histamine release, hypotension, prolonged sedation, bradycardia.
Nitrous oxide	Colorless gas mixed 50% with oxygen	Demand valve mask	3–5 min	3–5 min on withdrawal	Provides mild analgesia, anxiolysis and detached attitude toward pain and surroundings. Patient must be old enough to cooperate with face mask (approx. 5 yrs). Must washout with 100% oxygen for 5 min post-procedure.	Nausea, vomiting, disorientation, agitation and expansion of air-filled cavities.
Pentobarbital (Nembutal)	0.5–2 mg/kg 2–6 mg/kg	IV IM	30–60 sec 10–15 min	15+ min	Barbiturate with no analgesic properties. Titrate dosage based on the child's response; not to exceed a maximum dose of 6 mg/kg *or* 150 mg total dose.	Monitor carefully for respiratory depression. Contraindicated in patients suffering from acute intermittent porphyria, liver failure.
Propofol (Diprivan)	0.5–1 mg/kg Maintain infusion rate 50–200 mcg/kg/min	IV	5–7 sec	8–11 min per bolus dose or upon withdrawal of infusion.	Potent non-benzodiazepine non-barbiturate sedative hypnotic with no analgesic properties. Ideal for short painful procedures (consider analgesic adjunct).	Screen for egg or soy allergy. Induces very deep levels of sedation and may cause respiratory depression, apnea and rarely hypotension. Pain frequently noted on injection; may be attenuated by adding lidocaine 1 mg/kg (maximum 20 mg) to initial bolus syringe. Older patients are more prone to respiratory depression; consider reducing dosage for >55 years of age.

IV: intravenous; IM: intramuscular; PO: per os; PR: per rectum.

Table D.7 Reversal agents

Medication	Recommended dosage	Route	Onset	Duration	Additional instructions/ mode of action	Precautions/ contraindications
Flumazenil (Mazicon)	*Adult:* 0.2 mg May repeat in 60 sec × 3 Maximum dose 1 mg *Pediatric*: 0.01 mg/kg	IV IV	1–2 min	20 min	Used for the reversal of benzodiazepine-induced sedation. Administer slowly to avoid adverse consequences of abrupt awakening, such as dysphoria and agitation. Repeat doses of flumazenil may be given (typically at 20-min intervals) as needed to reverse sedation. Maximum dose of 1 mg at any one time; not to exceed 3 mg in 1 hour. Dose of 1 mg sustains antagonism for approximately 48 min.	Seizures may occur with reversal of sedative effects. Do not administer if myoclonic jerking noted. Duration of action shorter than midazolam and other benzodiazepines. Re-sedation may occur following initial reversal; monitoring must be performed for an appropriate period after initial reversal. Contraindicated in status epilepticus, increased intracranial pressure, tricyclic antidepressant overdose and chronic benzodiazepine use (>2 weeks).
Naloxone (Narcan)	*Adult*: 5–10 mcg/kg *Titrate to desired effect.* (Standard historical dose for OD is 0.4–2 mg) *Pediatric:* 5–10 mcg/kg *Titrate to desired effect.* Common starting dose 0.2 mg	IV/ET/IM IV/ET/IM	1–2 min	Dependent on the dose and route of administration; 30–60 min typical.	A narcotic antagonist preventing or reversing the effects of opioids, including respiratory depression, sedation and hypotension. *Titration preferred to rapid bolus.* Maximum dose of 10 mg. Dose conservatively to avoid acute withdrawal and agitation.	The patient responding satisfactorily to naloxone should be kept under continuous surveillance; repeated doses may be required since the duration of action of most narcotics exceeds that of naloxone. May use IV drip. Excessive use beyond the recommended dosage may actually potentiate respiratory depression in an already depressed patient.

ET: endotracheal; IM: intramuscular; IV: intravenous; OD: overdose.

Procedural preparation, monitoring and risk awareness

Most EDs have written policies on sedation which outline staffing requirements, monitoring guidelines, approved medications, post-procedure observation, and discharge criteria. These should be reviewed carefully before attempting PSA. Obtain informed consent, and discuss risks and benefits of PSA with every patient before proceeding. Ensure that a responsible adult is available to receive instructions, transport and observe the patient following the procedure.

Almost all of the agents used for PSA diminish respiratory drive and produce varying degrees of loss of airway protective reflexes. Administration of these agents may lead to respiratory or cardiopulmonary arrest, depending on the medication, dosing, rate of administration and patient sensitivities. Prepare ahead of time by assembling all appropriate equipment (Table D.8).

Move the crash cart to the bedside. Place the patient on cardiac, blood pressure, pulse oximetry and capnography monitors. Ensure that oxygen is immediately available, both by nasal cannula and bag-mask ventilation device. Most practitioners prefer to administer oxygen to the patient before starting and throughout PSA, to minimize the likelihood and duration of hypoxia. Pre-oxygenation may delay the recognition of hypoventilation when pulse oximetry is used as the mainstay of monitoring. It is even more important to closely watch the patient's respirations if oxygen is being administered.

Table D.8 Advance preparation for procedural sedation and analgesia

Pre-procedure checklist

- High-flow oxygen[a]
- Suction with large-bore catheter and appropriately sized tip for patient[a]
- Vascular access equipment[a]
- Airway equipment (e.g., bag-mask ventilation device, intubation equipment)[a]
- Monitoring
  - Capnography (recommended)
  - Pulse oximetry[a]
  - Cardiac and blood pressure monitors[a]
- Crash cart (with advanced life support equipment and medications)[a]
- Reversal agents (specific for sedative and analgesic agents used)[a]
- Adequate staff for monitoring and documentation[a]
- Informed consent[a]

[a] Suggested minimum equipment.

Assemble intubation equipment so it is close at hand and ready for use. Select the appropriate endotracheal tube sizes, stylet and laryngoscope blades, making sure that the handle and blades are functional. It is not necessary to open all the packaging, but be ready to do so quickly if the need arises. Turn on the suction and attach the appropriate suction catheter to the tubing. Ensure that the suction apparatus is working properly prior to giving sedative or analgesic medications.

If an IV is not already in place, assemble the necessary equipment so that one can be started quickly. If you are using drugs with known antidotes, make sure these medications are at the bedside, ready to be drawn up. Post the calculated doses and volumes at the bedside, making sure that the team can easily see this information.

After completing all the preparations outlined above, document the pre-sedation history and physical examination, the PARQ (procedure, alternatives, risks and questions) discussion, and patient consent. Assemble the team and ensure that every member understands the plan for the sedation, the procedure, and the anticipated recovery course. Some institutions insist on a documented "team time-out" (or pause) prior to the procedure.

Assign one person to closely monitor the patient from procedure onset until complete patient recovery. This dedicated provider should observe and repeatedly assess the patient's level of consciousness, skin color, respiratory rate and depth, heart rate, blood pressure, cardiac rhythm, oxygen saturation and end-tidal capnography (if available). Although end-tidal capnography may detect inadequate ventilation before oxygen desaturation occurs, it does not replace human vigilance. At a minimum, all vital signs should be checked pre-procedure, every few minutes and as necessary following administration of the PSA medications, on completion of the procedure, during early recovery and following complete recovery.

Recovery and discharge

The risk of apnea or hypoxia is greatest right after completion of a painful procedure but before the PSA drugs have worn off. Never leave a drowsy patient unattended. It is critical that a skilled health care provider continues to closely monitor the patient until he or she has adequately recovered from PSA (i.e., breathing normally, protecting their airway, and returned to baseline cognitive and motor function). If an antidote (i.e., reversal agent) was used, continue to closely monitor the patient for at least an hour beyond the time the antidote was expected to wear off. This will ensure prompt identification of recurrent respiratory depression or sedation from the PSA agents.

Vomiting may occur, especially after ketamine or certain opioids. Ondansetron may reduce the incidence of post-procedural emesis, while midazolam has been shown to reduce the incidence of emesis following ketamine sedation.

Discuss discharge criteria with the patient and family or friends who will drive the patient home. Do not allow the patient to drive. Instruct the patient that subtle cognitive deficits and drowsiness may persist for hours after PSA, and he or she should neither drive nor operate heavy machinery for at least 24 hours after PSA. Patients and family or friends should fully understand which signs and symptoms mandate immediate return to the ED.

Patients are considered safe for discharge when they are able to fulfill specific discharge criteria (Table D.9).

All patients must have a full understanding of late side effects that may occur as a result of their sedation. Vomiting may occur after discharge following ketamine or narcotic analgesics. In rare cases, post-sedation hallucinations may occur with ketamine for several weeks.

Table D.9 Criteria for safe discharge of patients after procedural sedation and analgesia

- No evidence of respiratory distress, hypoxia, or hypoventilation
- No or minimal nausea, vomiting, or dizziness
- Able to take fluids and medications by mouth
- Responsible person to transport patient and monitor him/her at home; must be able to understand discharge instructions and criteria prompting emergent return
- Vital signs stable for at least 30 min
- Baseline mental status achieved (alert, oriented, able to retain information, or age-appropriate behavior)
- Return to pre-procedural sedation and analgesia or baseline motor function (age-appropriate behavior)

Special patients

Infants and toddlers have the most variable response to medications. Medications should be dosed according to a current and accurately measured weight. Before starting PSA, the appropriately sized airway equipment should be within easy reach at the bedside.

Patients on psychotropic medications and those with post-traumatic stress disorder may react unpredictably to some analgesics and sedatives, particularly ketamine.

The elderly often have diminished cardiorespiratory reserve and are often taking several medications that might interact with PSA agents. Therefore, elderly patients require careful, slow titration of medications and hypervigilance in monitoring their oxygenation and ventilation status.

IV drug users usually have a high tolerance to opiate analgesia, but not necessarily to respiratory depression induced by the opiates. These patients require additional precaution.

A hypovolemic patient should be optimally fluid resuscitated prior to giving sedative–analgesics, in order to minimize the likelihood of hemodynamic compromise or cardiovascular collapse.

Pearls, pitfalls and myths

- Prior planning and careful preparation for the worst possible scenarios are the hallmarks of safe PSA. Begin with careful patient selection, proper monitoring procedures, availability of advanced airway management equipment, and close post-procedure and recovery observation. The most dangerous time is when the painful procedure is over but the patient has not yet fully recovered.
- Pulse oximetry and capnography are essential for adequate monitoring but do not replace human vigilance. Assign an appropriate individual to constantly monitor the patient's chest movement, color and mental status.
- Supplemental oxygen in a sedated, hypoventilating patient may improve oxygenation but has no effect on ventilatory status. Manual stimulation and airway repositioning should be the first measures used to address decreasing oxygen saturation or increasing carbon dioxide levels.

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Appendix E Guide to ED ultrasound

Section 1 Introduction and glossary of terms

Sarah R. Williams, MD

Ultrasound (US) is rapidly becoming the stethoscope of the twenty-first century. Many medical specialties use this technology to enhance patient care, both in the diagnosis and treatment of disease.

This appendix introduces several critical applications of emergency ultrasound: focused assessment with sonography in trauma (FAST) and extended FAST (E-FAST), emergency echocardiography, evaluation for abdominal aortic aneurysm, first-trimester pelvic ultrasound (including evaluation for suspected ectopic pregnancy), biliary evaluation, and rapid ultrasound in shock (RUSH).

Equipment

It is important to select the best probe for each ultrasound application. Higher frequency probes provide excellent images of superficial structures; however, they cannot penetrate deeply into the tissue. Lower frequency probes are used to image deeper structures.

Low-frequency probes (usually in the 2–5 MHz range) are optimal for echocardiography and scanning abdominal/pelvic organs (Figure E1.1). Both small footprint and large curved probes are available in this frequency range.

- The small footprint transducer is optimal for imaging between the ribs. Because it minimizes *rib shadow* (artifact caused by the ribs blocking the ultrasound beam), this transducer is ideal for FAST and echocardiography, where intercostal views are necessary.
- The larger footprint curved probe is optimal for aortic and pelvic evaluation.

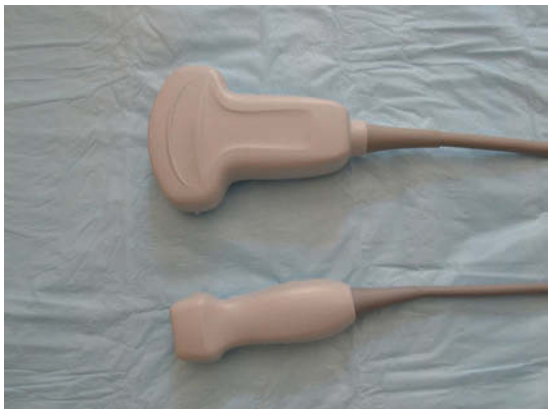

Figure E1.1
Low-frequency transducers.

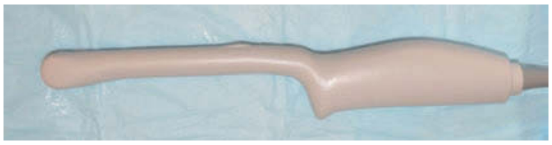

Figure E1.2
Endocavitary transducer.

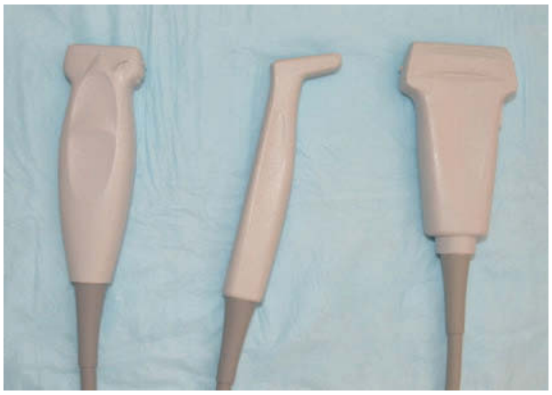

Figure E1.3
High-frequency linear transducers.

Medium frequency endocavitary probes (usually in the 5–8 MHz range) are used for endovaginal scanning (Figure E1.2). The probe's proximity to the uterus affords excellent resolution and facilitates assessment of early intrauterine pregnancy.

High-frequency linear probes (commonly in the 5–10 [or higher] MHz range) are optimal for scanning superficial structures, such as the pleural line just beneath the ribs (for the E-FAST exam) or the internal jugular vein (Figure E1.3).

Glossary of terms

Coronal: the ultrasound plane that divides the body into “front” and “back” (Figure E1.4). Example: the image obtained when the ultrasound probe is held in the mid-axillary line aiming into the center of the body (i.e., the FAST exam).

Depth: an ultrasound system control that allows for increasing or decreasing the depth of body tissue seen on the screen. Example: if scanning a large patient, the depth setting to evaluate the aorta would need to be set higher than for a thinner patient.

Far field: the aspect of the ultrasound image that is towards the bottom of the screen, correlating to the tissue "far" from the transducer.

Gain: an ultrasound system control that increases or decreases the overall signal output of the image on the screen. If gain is set too low, the image is too dark; if set too high, the image would be too bright.

Indicator marker: a bump or mark on the ultrasound probe that allows for orientation. Example: the FAST exam convention is to have this marker towards the patient's right side or head.

Longitudinal: the vertical ultrasound plane that divides the body into "left" and "right" (Figure E1.4). Example: the image obtained for evaluation of the long axis of the uterus, when the ultrasound probe is held just above the pubic symphysis, pointing back, with the indicator towards the head. Also termed *Sagittal.*

Near field: the aspect of the ultrasound image that is towards the top of the screen, correlating to the tissue "near" to the transducer.

Sagittal: see *Longitudinal*

Screen marker: the mark on the ultrasound screen that allows for orientation. Example: the FAST exam convention is to have this marker on the left side of the image on the screen, correlating with the patient's right side.

Shadow: when dense material impedes the ultrasound beam, a shadow results from blockage of sound. Example: rib shadows that may impede visualization of the liver or spleen for the FAST exam.

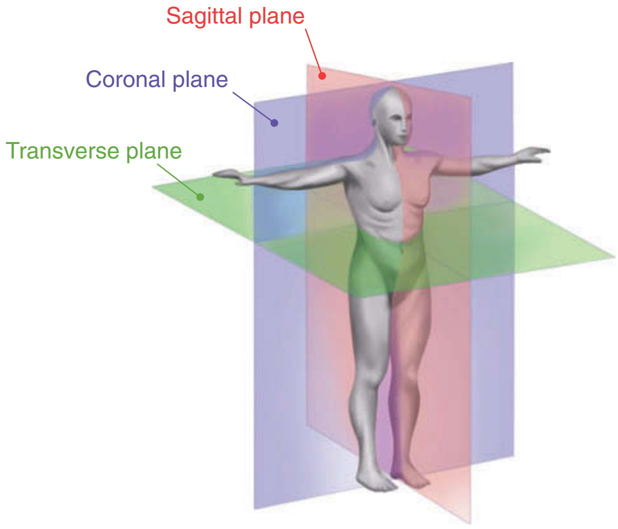

Figure E1.4
Schematic of body planes, representing transverse, coronal and sagittal (also referred to as longitudinal) planes.

Transverse: the horizontal ultrasound plane that divides the body into "upper" and "lower" (Figure E1.4). Example: the image obtained for evaluation of the short axis of the aorta (circular appearance), when the ultrasound probe is held in the mid abdomen, pointing backwards, with the indicator towards the patient's right.

Section 2 FAST (focused assessment with sonography in trauma)

Teresa S. Wu, MD, Diku Mandavia, MD and Sarah R. Williams, MD

Background

- FAST is a highly focused, limited, goal-directed exam with the express purpose of answering a specific question: “Is there free fluid in the abdomen?”
- Four primary views: right upper quadrant (RUQ), left upper quadrant (LUQ), cardiac and suprapubic (Figure E2.1).
- More cost-effective, less time-consuming, and less invasive than computed tomography (CT) or diagnostic peritoneal lavage (DPL).
- Reliably detects 200–500 mL of intra-abdominal free fluid with sensitivity of 73–88% and specificity of 98–100%.
- Blood within the peritoneum often indicates the need for laparotomy, or at least close observation in the ICU.
- Identifies actual organ injury less than 50% of the time; does not evaluate retroperitoneal hemorrhage; less acute with concurrent pelvic fracture.
- Can be performed immediately, simultaneously, and in parallel with clinical management.
- Can be performed on the unstable patient and repeated if the clinical scenario changes.
- No associated radiation exposure.

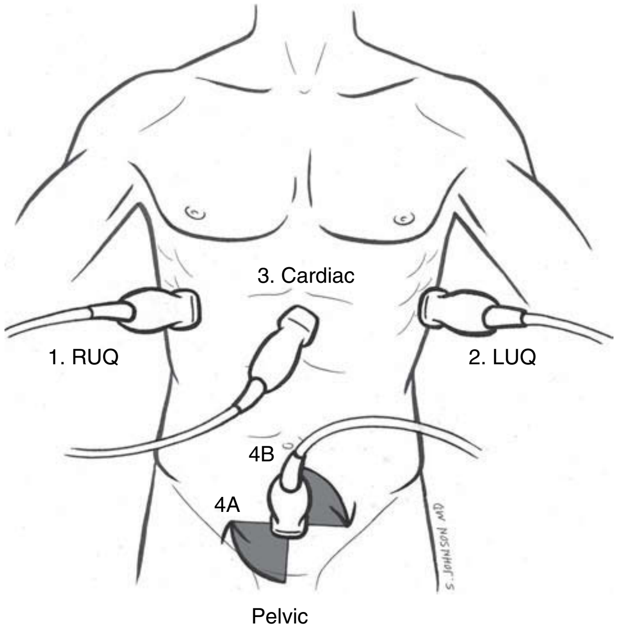

Figure E2.1
Overview of FAST exam probe placement. Four views are standard: Morison’s pouch (RUQ), perisplenic (LUQ), subcostal (cardiac) and suprapubic (pelvic). Printed with permission from Phillips Perera, MD.

Indications

- Rapid detection of hemoperitoneum from intra-abdominal injury, pericardial effusions and pleural effusions/hemothorax
- Unexplained shock
- Should be incorporated into ATLS algorithm for evaluation of patients with:
  - Blunt/penetrating abdominal trauma
  - Penetrating thoracic injury
  - Blunt thoracic injury
  - Fractures suggesting a significant mechanism of injury
  - Trauma with other mitigating factors:
    - Drugs or alcohol impairment
    - Paralysis (paraplegia and quadriplegia)
    - Unresponsiveness secondary to head trauma, hypoxia, or hypovolemia
    - Any unexplained blood loss or signs of hemorrhagic shock

Probe choice/system presets

- **Probe choice:** In most patients, the 2.5–5 MHz curvilinear or phased array transducer should be used for all elements of FAST. The small footprint phased array transducer is preferred due its small size and easier fit between the ribs (Figure E2.2).
- **Machine presets:** Use the ultrasound system abdominal imaging presets. The convention is to place the screen marker on the left side of the image (corresponding to the patient’s right side or head) (Figure E2.3). If these conventions are not followed, the images will be reversed from standard.

Anatomy and ultrasound technique

- When a patient is lying supine, fluid in the abdomen usually flows to “Morison’s pouch” (right hepatorenal fossa), the most dependent supramesocolic location (Figure E2.4).

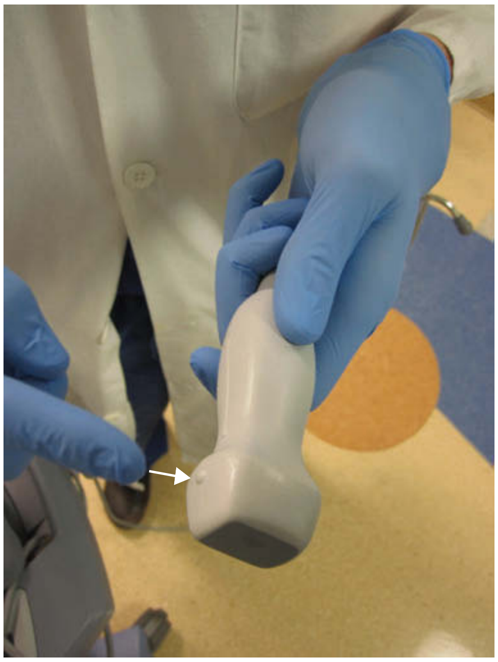

Figure E2.2
Example of a small-footprint low-frequency probe; the raised bump (white arrow) indicates the probe marker.

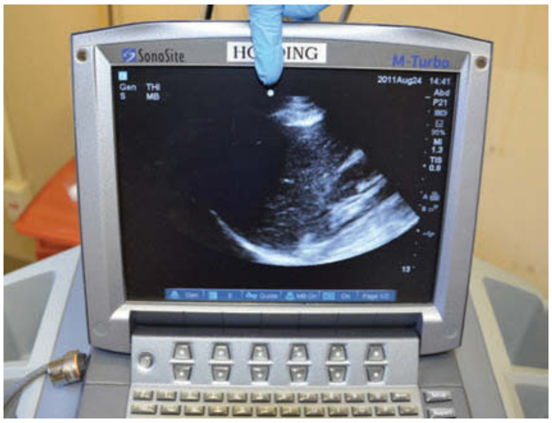

Figure E2.3
Example of an ultrasound machine screen, showing green screen dot on left side of image (mid-top of screen).

- Fluid in the pelvis usually localizes in the retrovesicular space in men, and in the pouch of Douglas (cul-de-sac) in women (Figure E2.4).

RUQ anatomy

- Also known as "Morison's Pouch" or the "right hepatorenal" view.
- Visualizes the interface between the liver and Gerota's fascia of the right kidney.
- Most sensitive view for visualizing free fluid in the abdomen.
- Interface resides at the vertebral level between T12 and L3.

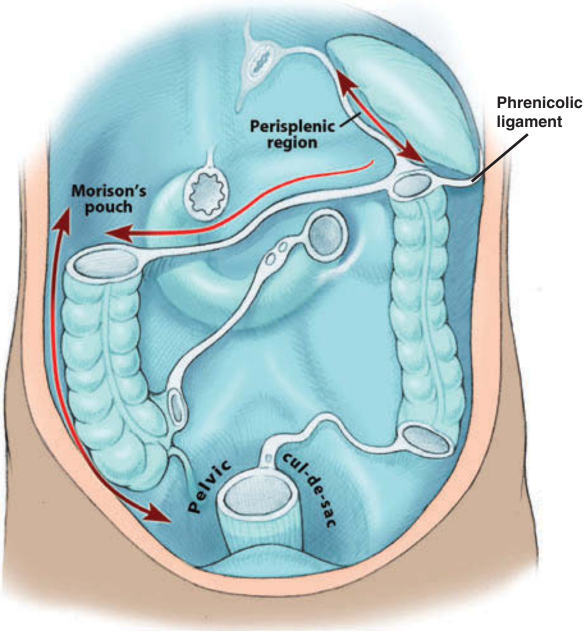

Figure E2.4
Intraabdominal mesenteric reflections and fluid-flow patterns.
© Chris Gralapp.

- Long access of the interface runs almost parallel to the body.
- The renal fascia (Gerota's fascia) encloses the perirenal fat and the fibrous renal capsule of the kidney.
- Gerota's fascia attaches superiorly to the fascia of the inferior diaphragm, and posteriorly to the fascia over the psoas major muscle.
- The descending part of the duodenum passes across the hilum of the anterior-inferior portion of the right kidney.
- The suprarenal, duodenal and colic areas of the kidney are not covered by peritoneum and create a potential space.
- The hepatorenal recess is the potential space between the anterior-lateral pole of Gerota's fascia. It is continuous superiorly with the inferior fascia of the diaphragm, so the level of the hepatorenal interface may vary up to 3 cm with respirations.
- The adrenal gland covering the anterior-medial aspects of the superior pole of the right kidney may be visualized with an RUQ scan. Do not mistake this structure for free fluid.

RUQ/Morison's pouch imaging technique

- Place the probe at the right seventh or eighth intercostal space, near the mid-axillary line (Figure E2.5).
- Orient the probe indicator marker cephalad and slightly posterior, resulting in a slightly oblique orientation. This will penetrate directly through the intercostal space, and minimize rib shadows.
- Recall that the rib cage curves inferiorly as you move from posterior to anterior.
- Use the liver parenchyma as your sonographic window.

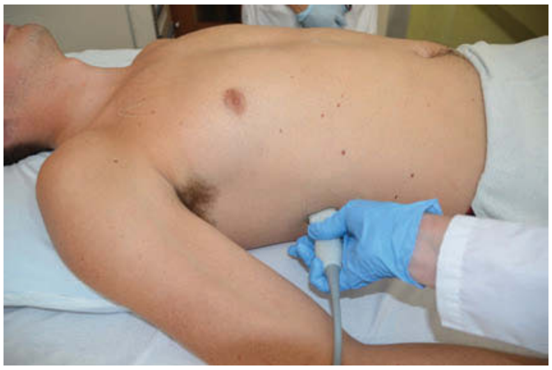

Figure E2.5
FAST exam RUQ probe placement.

- The bright white line of Gerota’s fascia lies flush against the liver parenchyma with a negative/normal exam (Figure E2.6).
- A positive exam for free fluid in Morison’s pouch will reveal an anechoic (black) stripe between the echoic (white) stripe of Gerota’s fascia and the liver parenchyma (Figure E2.7).
- Attempt to visualize the echoic (white) diaphragm cephalad to the liver to evaluate for fluid in the lung base. Sometimes you will need to move up an interspace to do this, but otherwise the probe orientation is the same.
- Asking the patient to inspire slightly will bring the diaphragm into view.

LUQ anatomy

- Also known as the perisplenic or the “splenorenal” view.
- Visualizes the interface between the spleen and Gerota’s fascia of the left kidney.
- Interface resides at the vertebral level between T11 and L2.
- Long access of the interface runs almost parallel to the body.
- The renal fascia (Gerota’s fascia) encloses the perirenal fat and the fibrous renal capsule of the kidney. It attaches superiorly to the fascia of the inferior diaphragm, and posteriorly to the fascia over the psoas major muscle.
- Anterior to the left kidney lie portions of the stomach, spleen, pancreas, jejunum and descending colon. Care should be taken not to mistake a fluid-filled stomach for free fluid.
- The splenorenal (aka lienorenal) ligament attaches the superior pole of the left kidney anterolaterally to the spleen.
- The spleen is completely surrounded by peritoneum except at the hilum, where the lienorenal ligament attaches.
- Fluid may accumulate in the splenorenal recess representing the junction between the spleen and left kidney adjacent to the splenorenal ligament; however, it usually collects first between the spleen and the diaphragm.
- Free fluid may also be visualized coursing along the superior-lateral pole of the spleen under the peritoneum below the left diaphragm.
- Note that Gerota’s fascia is continuous superiorly with the inferior fascia of the diaphragm, so the level of the splenorenal interface may vary up to 3 cm with respirations.
- The adrenal gland covering the anterior-medial aspects of the superior pole of the left kidney may be visualized with an LUQ scan. Do not mistake this structure for free fluid.

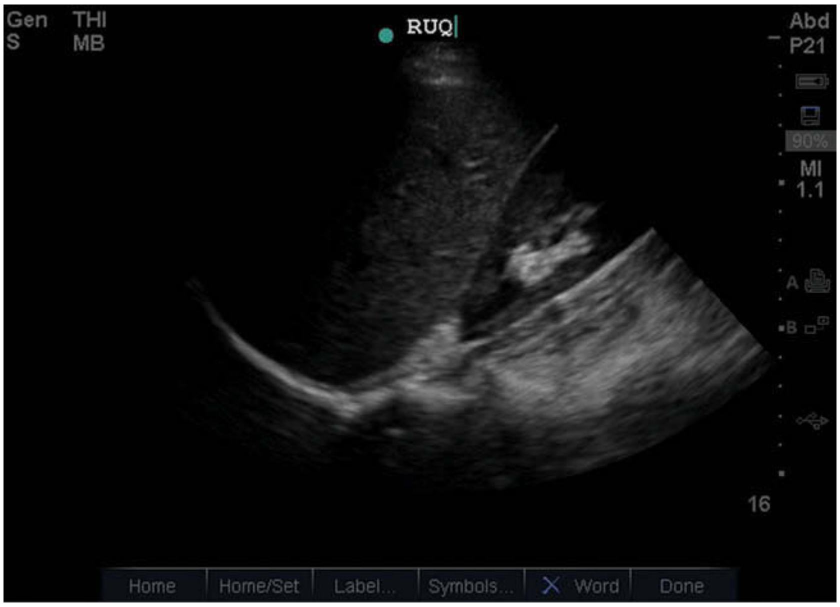

Figure E2.6
Negative FAST RUQ image.

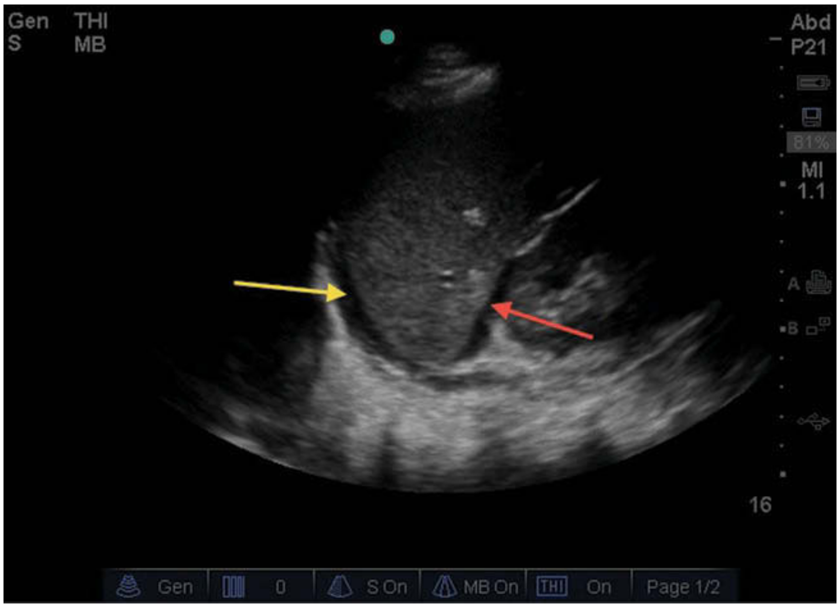

Figure E2.7
Positive FAST RUQ image, with free fluid in Morison's pouch (red arrow) and between the liver and diaphragm (yellow arrow).

LUQ/Perisplenic imaging technique

- Place the probe in the left seventh, eighth or ninth intercostal space, in the mid to posterior axillary line (Figure E2.8). The probe will typically be held more cephalad than for the RUQ view.
- The splenorenal view is a much more difficult window than the Morison's pouch view. Occasionally the patient will need to be rolled slightly onto his or her right side to allow optimum visualization of the target organs. A good rule of thumb is to place the probe further posterior and cephalad compared with the right side. Sometimes the operator's hand has to squeeze in between the backboard and the patient for an optimum view.
- Orient the indicator marker cephalad and slightly posterior, resulting in a slightly oblique orientation. This will penetrate directly through the intercostal space, and minimize rib shadows.

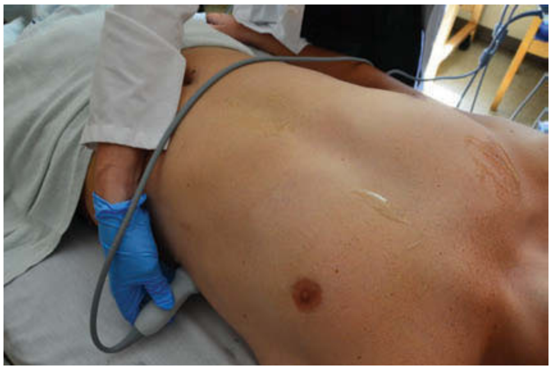

Figure E2.8
FAST exam-LUQ probe placement.

- Recall that the rib cage curves inferiorly as you move from posterior to anterior.
- The bright white line of Gerota's fascia will lie flush against the spleen with a negative/normal exam (Figure E2.9).
- A positive exam for free fluid in the perisplenic recess will reveal an anechoic (black) stripe between the echoic (white) stripe of Gerota's fascia and the spleen or between the thick white dome of the diaphragm and the spleen (Figure E2.10).
- You may need to utilize intercostal spaces cephalad or caudad in order to achieve a clear view of the entire perisplenic recess.
- Attempt to visualize the echoic (white) diaphragm cephalad to the spleen to evaluate for fluid in the lung base or free fluid between the diaphragm and the spleen. You may need to move the probe up an interspace to do this, but the probe orientation will be the same.
- Asking the patient to inspire slightly will bring the diaphragm and the suprasplenic area into view.

Pelvis anatomy

- An empty bladder will lie almost entirely in the pelvis minor, inferior to the pelvic floor and posterior to the pubic symphysis.
- With a full bladder, the sonographic window may rise as high as the umbilicus. Therefore, it is much easier to ultrasound when the bladder is full.
- In the female, the bladder peritoneum is reflected from the superior-posterior surface of the bladder onto the anterior wall of the uterus, at the junction of the uterine body and cervix.

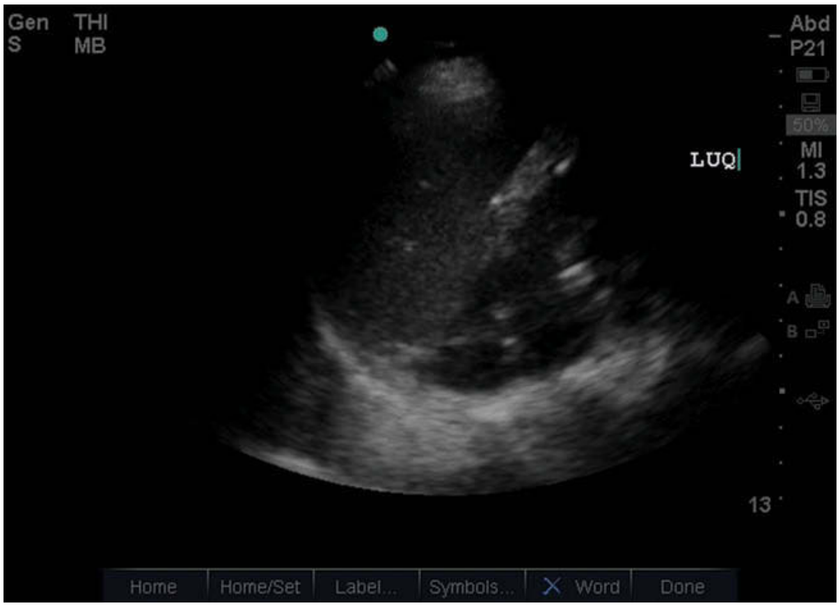

Figure E2.9
Negative FAST image of the LUQ.

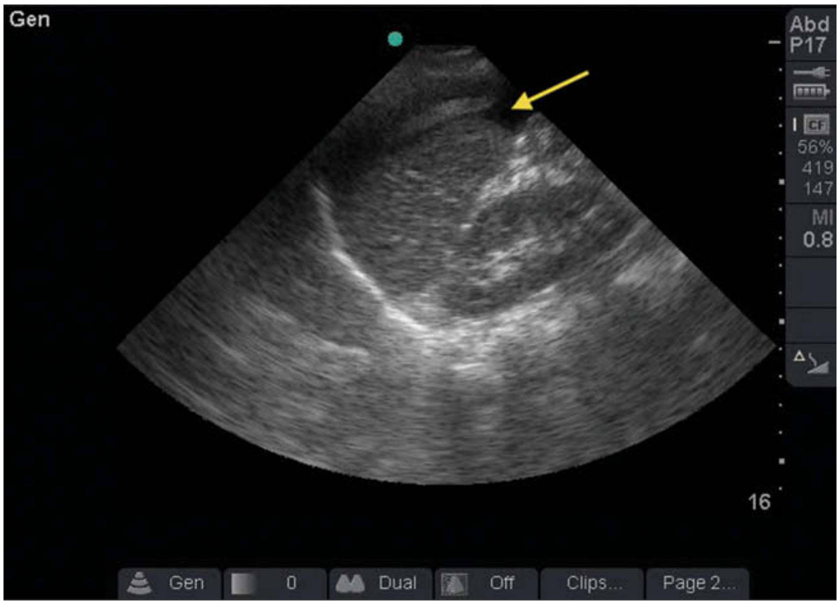

Figure E2.10
Positive FAST image of LUQ, with free fluid around the spleen (yellow arrow).

- This reflection of peritoneum creates a recess called the vesicouterine pouch, or pouch of Douglas. This region is also called the *cul-de-sac*.
- In the male, the bladder peritoneum is reflected posteriorly towards the rectum, forming a recess called the rectovesicular pouch.
- The vesicouterine pouch in females and the rectovesicular pouch in males communicate directly with the paracolic and infracolic gutters superiorly, such that free intraperitoneal fluid from any abdominal level may gravitate and accumulate in the recesses.

Pelvis/suprapubic view imaging technique

- This view evaluates the rectovesicular pouch in males, and the Pouch of Douglas (aka *cul-de-sac*) in females.
- Place the probe in a transverse orientation across the patient's suprapubic region, and aim it down into the pelvis, with the indicator marker pointing towards the patient's right side. A good rule of thumb is to make contact with the pubic symphysis with the probe, then angle down towards the feet for optimum visualization (Figure E2.11).

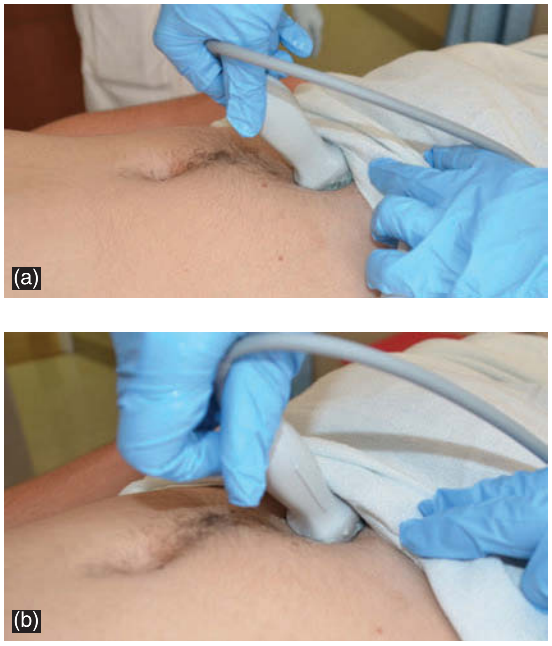

Figure E2.11
(a) FAST exam-suprapubic probe placement, in longitudinal orientation and (b) transverse orientation.

- Rotate your probe 90 degrees cephalad (with the probe marker pointing towards the patient's head) and obtain a longitudinal view of the bladder and surrounding structures. The longitudinal view will help delineate the uterus in women and the prostate in men.
- Using the bladder as your sonographic window, attempt to visualize the space far field (posterior) to the bladder.
- A normal view in females will show the bladder, uterus behind it, then bowel gas (Figure E2.12).
- A normal view in males will show the fluid-filled bladder and bowel gas behind it (Figure E2.13).
- Free fluid in the pelvis will appear as an anechoic (black) stripe behind the uterus (Figure E2.14) or the bladder (Figure E2.15).
- Try to perform the suprapubic exam prior to placement of a Foley catheter so you don't lose your sonographic window. You may also fill the bladder in a retrograde fashion using normal saline if the Foley has already been placed and repeat scans are required.
- At times, the rectum can appear as a fluid-filled anechoic structure sitting behind the bladder wall. If there is question regarding whether the anechoic fluid collection is free fluid or simply an enlarged rectum, determine whether or not the fluid collection layers out with patient movement. Bowel contents will also often show peristalsis. Free fluid should be seen extending laterally in the pelvis when the patient is tilted onto his or her side.
- Subtle or small fluid collections can be missed. Therefore, visualize the entire space surrounding the bladder by fanning through the entire organ superiorly, inferiorly, and from one side to the other.
- Ultrasound is limited in its ability to diagnose bleeding from pelvic fractures. Other imaging techniques, including CT, should be considered in such cases.

Cardiac anatomy

- The pericardium is a double-walled fibroserous sac made up of two parts: a tough fibrous pericardium externally, and the double-layered sac of transparent membrane called the serous pericardium internally. The potential space between the parietal and visceral layers of the serous pericardium is called the pericardial cavity.

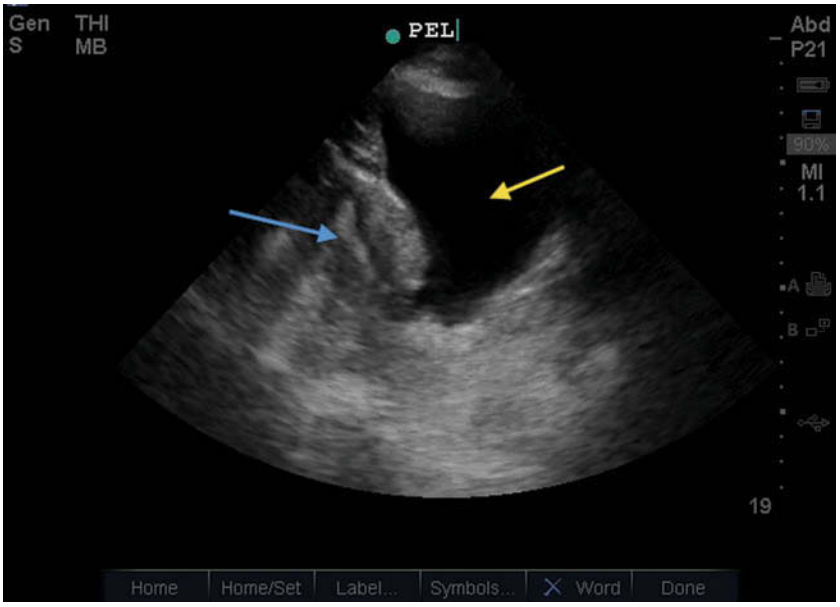

Figure E2.12
Negative FAST- female pelvis (blue arrow-endometrial stripe; yellow arrow-bladder).

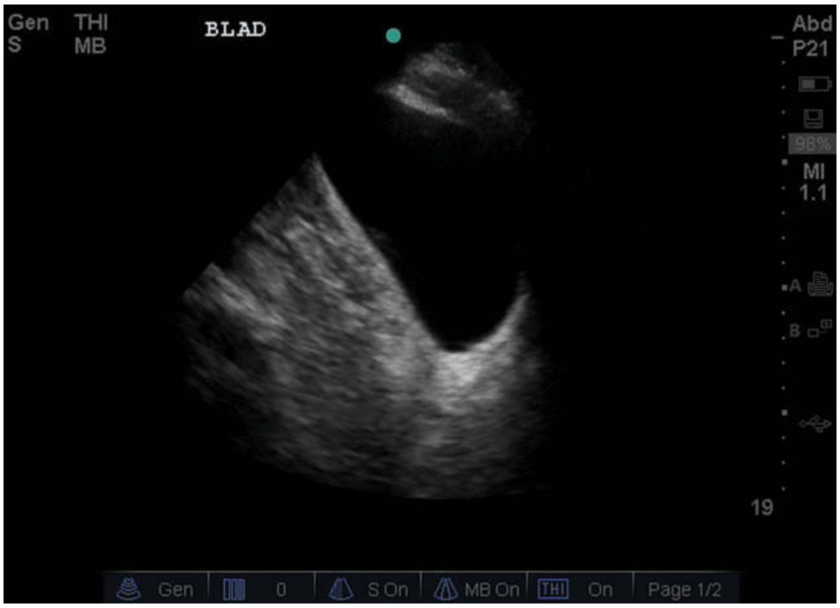

Figure E2.13
Negative FAST (male pelvis).

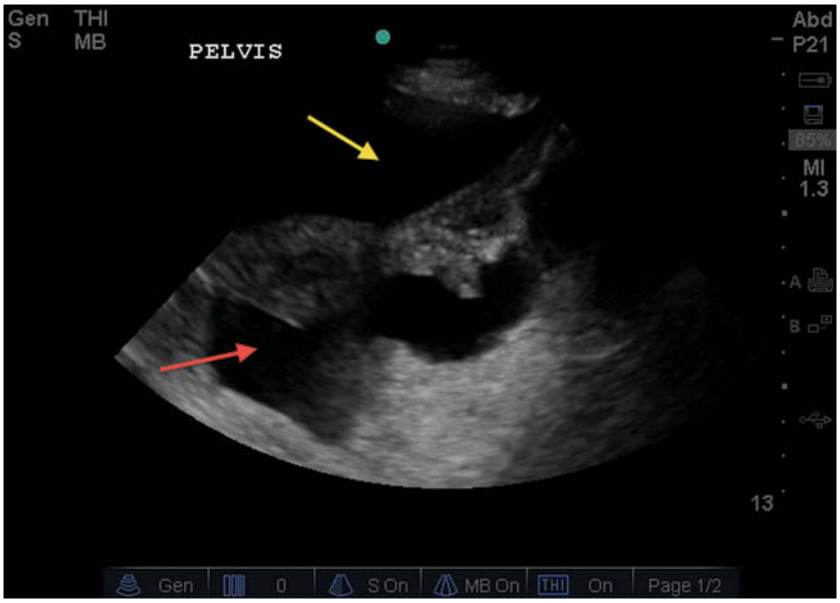

Figure E2.14
Large free fluid behind uterus (red arrow); bladder (yellow arrow).

- The appearance of the pericardial sac is influenced by respiration.
- Both blunt and penetrating trauma can cause bleeding into the pericardial cavity between the double-layered serous pericardium. If a fluid collection in this area accumulates quickly, shock and death can quickly occur due to the inability of the heart to pump against this pressure ("cardiac tamponade").
- The inferior diaphragmatic surface of the heart is comprised mainly of the right ventricle and a small portion of the left ventricle. When being evaluated with ultrasound from the subcostal view, the right ventricle is therefore the closest chamber to the probe and appears immediately behind the liver (Figure E2.16).
- Small effusions may layer posteriorly with the patient lying supine, whereas large effusions (>300 mL) will be visible in the anterior and posterior portions of the pericardial cavity (Figure E2.17).
- The left lobe of the liver is used as the sonographic window, as this crosses just under the sternum.

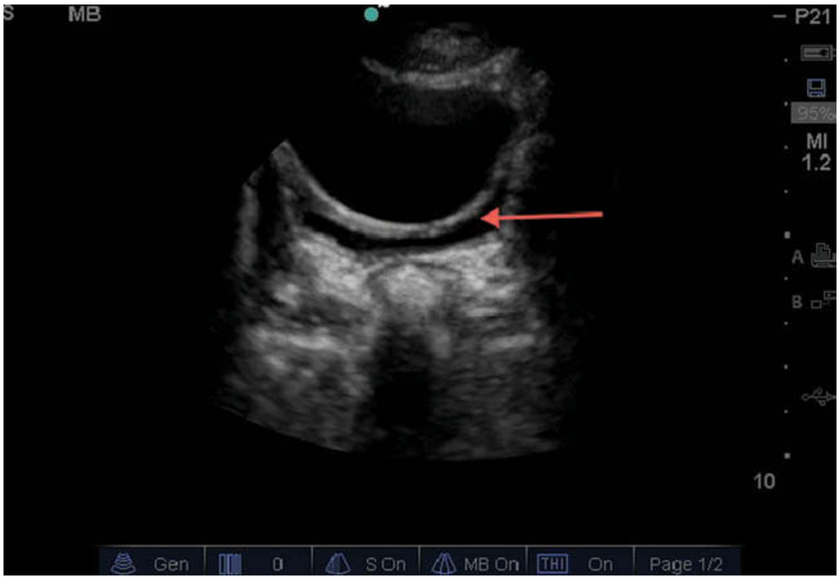
MB
P21
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Figure E2.15
Free fluid (red arrow) behind bladder.

FAST cardiac imaging

The standard FAST utilizes the subcostal (or "subxiphoid") view to evaluate the heart. If this view is difficult due to abdominal pain or poor visualization, the parasternal long axis view ("PLAX" view) can be used instead.

Subcostal (subxiphoid) imaging technique

- Place the probe in the epigastric region, just inferior to the xiphoid process. A good rule of thumb to ensure you are in good position is to run the probe up until it contacts the xiphoid process of the sternum, then flatten the probe and aim it towards the patient's left shoulder (Figure E2.18).

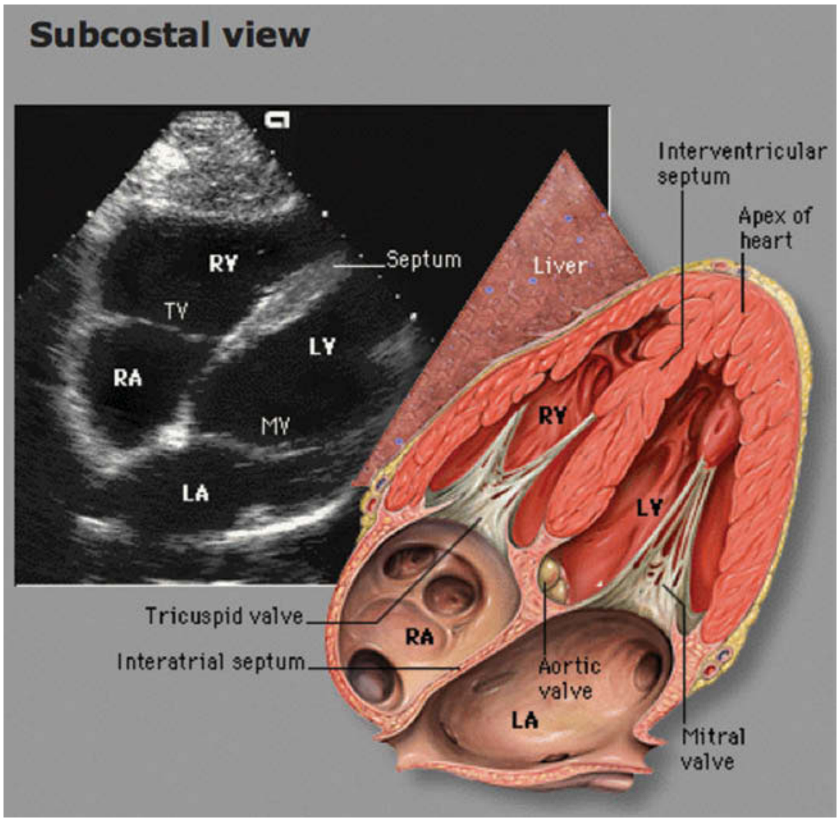

Subcostal view
RV
Septum
TV
LV
RA
MV
LA
Liver
Interventricular septum
Apex of heart
RV
LV
Tricuspid valve
Interatrial septum
RA
Aortic valve
LA
Mitral valve

Figure E2.16
Subcostal view of heart (echo image and corresponding anatomy). Courtesy: Patrick J. Lynch and C. Carl Jaffe, Yale University, 2006.

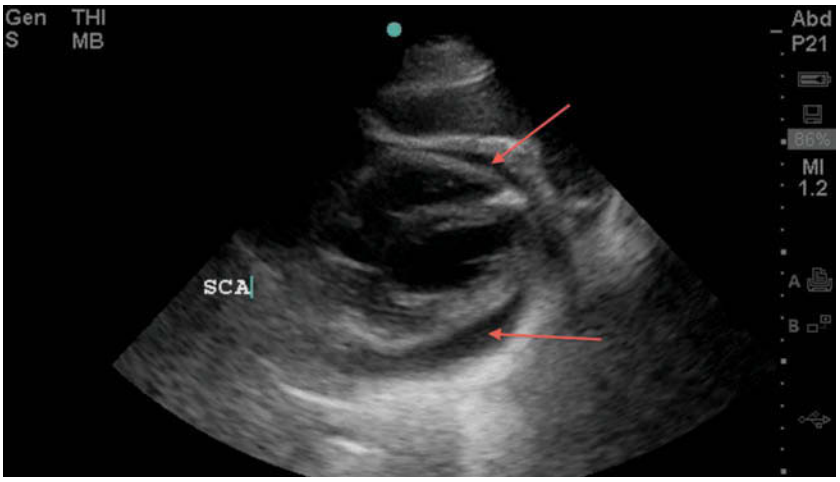


Figure E2.17
Subcostal cardiac view showing circumferential pericardial fluid around the heart (red arrows).

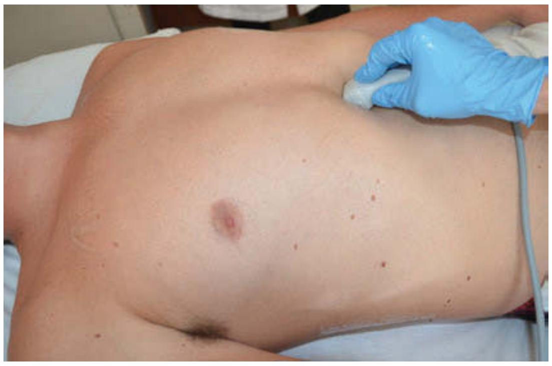


Figure E2.18
Probe placement for FAST exam-subcostal cardiac view.

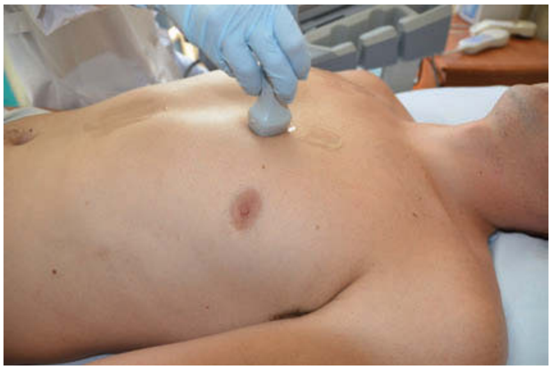


Figure E2.19
Probe placement for FAST exam intercostal (parasternal) view. Note probe marker oriented to left hip.

- By convention, orient the probe marker towards the patient's right side.
- Obtain a four-chamber view of the patient's heart by placing the probe flush against the patient's skin. Apply direct pressure on the body of the probe so that the face of the probe remains in contact with the patient's skin and the ultrasound beams are directed under the patient's rib cage.
- It is important not to let the probe lose contact with the skin as you angle up, as this will disrupt the image.
- In this view, all four chambers of the heart will be visualized. The right ventricle will be the most anterior anechoic chamber, appearing immediately behind the liver (Figure E2.16).
- Watch the pericardium through several cardiac cycles and evaluate for small pericardial effusions in the posterior pericardial sac.
- Occasionally, it may be difficult to see the heart, so asking the patient to inspire deeply will bring the entire pericardial sac towards your probe. Another trick to improve visualization is to increase the depth on the ultrasound system.
- If you do not obtain an adequate subxiphoid view (i.e., the patient is obese, has a barrel chest, or has too much abdominal pain), utilize the intercostal (parasternal) view.

Intercostal (parasternal) imaging technique

- Place the probe in the 2nd–4th intercostal space, just to the left of the sternum, with the probe marker pointed towards the patient's left hip (Figure E2.19). Decrease the depth setting compared with the subxiphoid view.
- Aim the beam posteriorly, perpendicular to the chest wall.
- A positive pericardial exam will show an anechoic (black) stripe of fluid between the echoic (white) fibrous pericardium and the chambers of the heart (Figure E2.20).
- If an effusion is identified in the subxiphoid view, confirm its presence in the parasternal view. Pleural effusions can be mistaken for pericardial effusions in the subxiphoid area (Figure E2.21).

Pearls, pitfalls and myths

- Morison's pouch is the most dependent position when the patient is lying supine, so begin the FAST exam with an RUQ view unless you are concerned about the possibility of cardiac tamponade. Be sure to look through the entire liver and kidney, both superior and inferior, medial and lateral, as

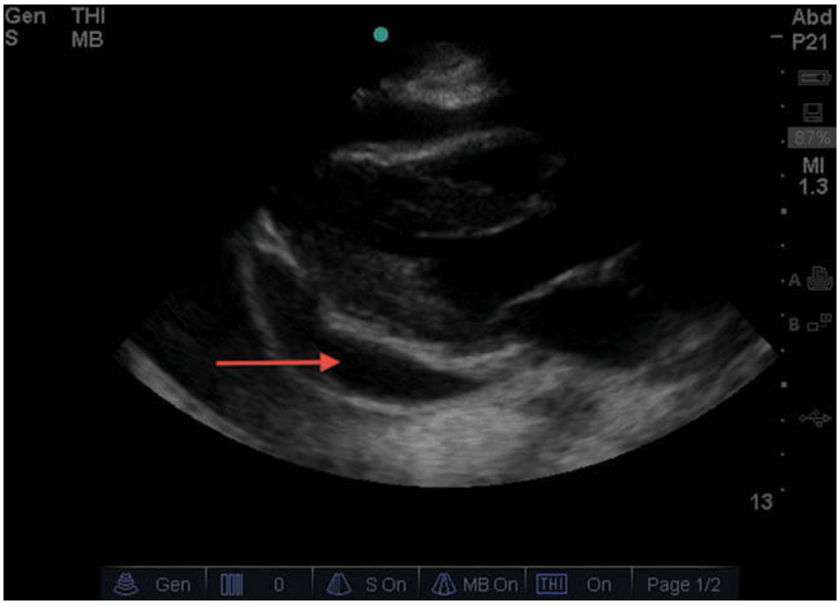


Figure E2.20
Intercostal (parasternal) view revealing pericardial effusion (red arrow).

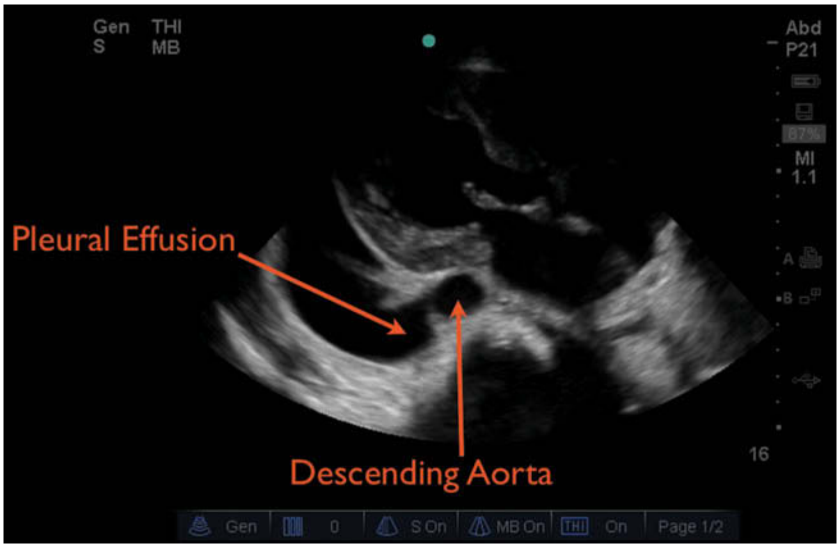


Figure E2.21
Pleural effusion is seen pointing behind the descending aorta in the parasternal long axis view.

small amounts of free fluid may appear in atypical locations.

- Placing the patient in slight Trendelenburg position may encourage free fluid to accumulate in the hepatorenal and splenorenal recesses, thereby enhancing your study.
- Ultrasound cannot rule out hemorrhage from retroperitoneal injury or pelvic fracture.
- Ascites can give a false-positive result; if unsure of the etiology of fluid, consider performing a diagnostic paracentesis. Other etiologies of free fluid include bladder rupture and fluid from hollow viscous injury.
- Visualize both the hepatorenal and splenorenal views through a few inspiratory cycles to further investigate for small areas of free fluid pooling, hemothorax or possibly diaphragmatic rupture. Although rare, it is possible to catch a diaphragmatic rupture during an RUQ or LUQ scan.
- Always attempt to scan through the entire target organ in order to visualize fluid accumulating in atypical anatomic locations.
- Approach organs from alternative angles if bowel gas is occluding the views.
- Patients who are obese may have significant amounts of perinephric fat, which may appear sonographically hypoechoic, and may lead to a false-positive reading. Remember that free fluid will move with patient re-positioning.
- Consider performing serial exams in patients in whom you have a high clinical suspicion for intra-abdominal injury or in patients in whom bowel gas obstructed earlier views.
- If the clinical scenario worsens, repeat the exam!

Section 3 Chest ultrasound for pneumothorax

Sarah R. Williams, MD **and Laleh Gharahbaghian,** MD

Background and indications

- The **E**xtended **FAST** (E-FAST) includes chest ultrasound (US) evaluation for pneumothorax (PTX).
- Chest US assessment for PTX combined with FAST assessment for hemothorax identifies the need for emergent chest tube thoracostomy.
- In ambulatory patients, air in the pleural cavity tends to collect apically; in supine patients, air collects anteriorly, right behind the chest wall.
- Chest US has a higher sensitivity than supine chest X-ray (CXR) for detection of PTX (when CT is used as the gold standard).
- Indications:
  - Assessment for PTX in patients with blunt or penetrating chest trauma
  - Assessment for spontaneous pneumothorax in patients with chest pain and/or shortness of breath

Probe choice/system presets

- **Probe choice:** a high frequency 7.5–10 MHz linear probe is best, as the pleura are fairly superficial (just below the ribs). If this probe is not available, the lower frequency probe used for the standard FAST can be used, but the image resolution is worse.
- **Machine presets:** Use the standard ultrasound presets when the linear probe is activated. The convention is to place the screen marker on the left side of the image (corresponding to the patient's head in the longitudinal plane). If these conventions are not followed, the images will be reversed from standard. Both 2D and M-Mode settings are utilized for this evaluation.

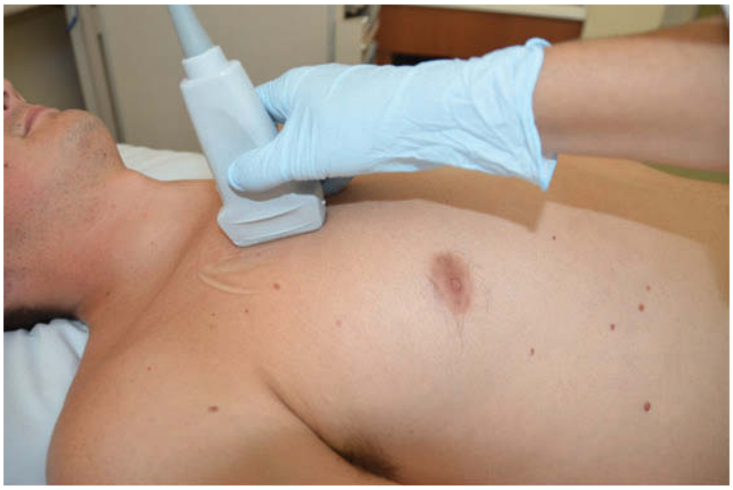


Figure E3.1
Physician placing linear probe vertically in the second intercostal space in the mid-clavicular line on the right side of chest.

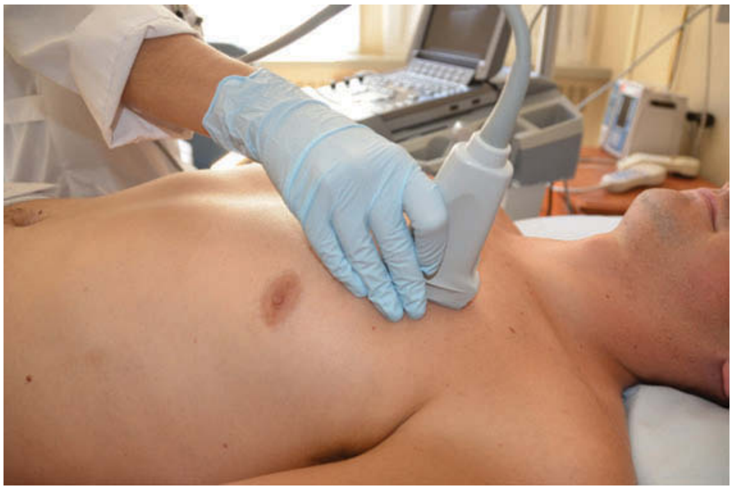


Figure E3.2
E-FAST probe placement, left chest.

Anatomy and ultrasound technique

- Initially, the probe is held vertically, with the probe indicator pointing towards the patient's head. Place the probe in the second intercostal space in the mid-clavicular line on each side of the chest (Figures 3.1 and 3.2).
- Identify the rib shadows and pleurae. The pleura, sonographically seen as a white line, covers the lung and runs immediately beneath the ribs, appearing as a bridge between the ribs. The appearance has been described as a "bat wing": the pleural line is the body of the "bat" and the ribs are the wings (Figure E3.3).
- In real time, evaluate for *lung sliding*, a gliding motion of the parietal-on-visceral pleura (sonographically identified by sliding of the pleural line). The appearance is similar to ants marching along a line. This phenomenon is seen only in normal lungs, and is absent in patients with PTX due to air tracking into the pleural space. Lung sliding is dynamic motion and cannot be appreciated in a still photo.
- Look for *comet tail* artifacts, which arise from the pleural line and are only present in normal lungs (Figure E3.4).
- If lung sliding and comet tail artifacts are visualized, a PTX is absent in the region of the lung being evaluated. With high suspicion for PTX, multiple regions should be assessed.
- The *lung point*, the transition point between normal and abnormal pleura, is found where comet tailing

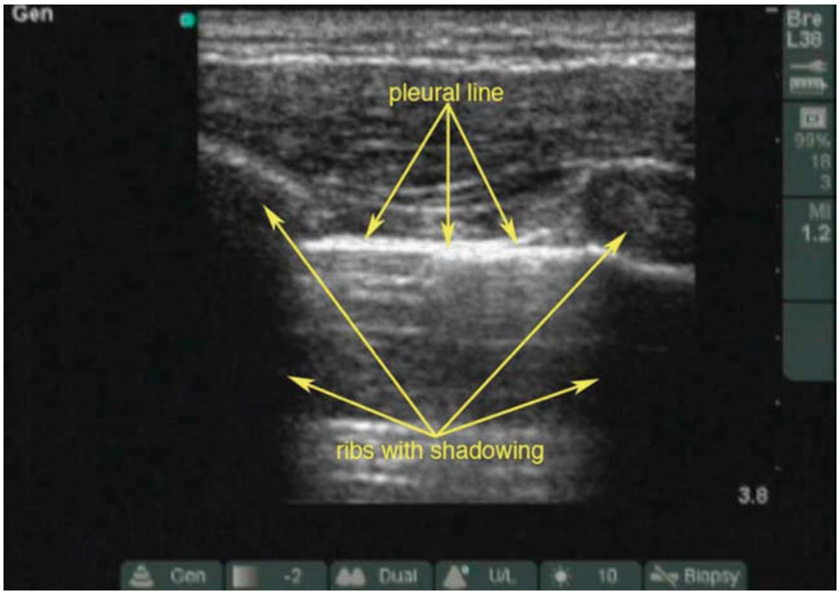


Figure E3.3
Ultrasound image illustrating normal lung-ribs with associated shadowing and the bright hyperechoic pleural line.

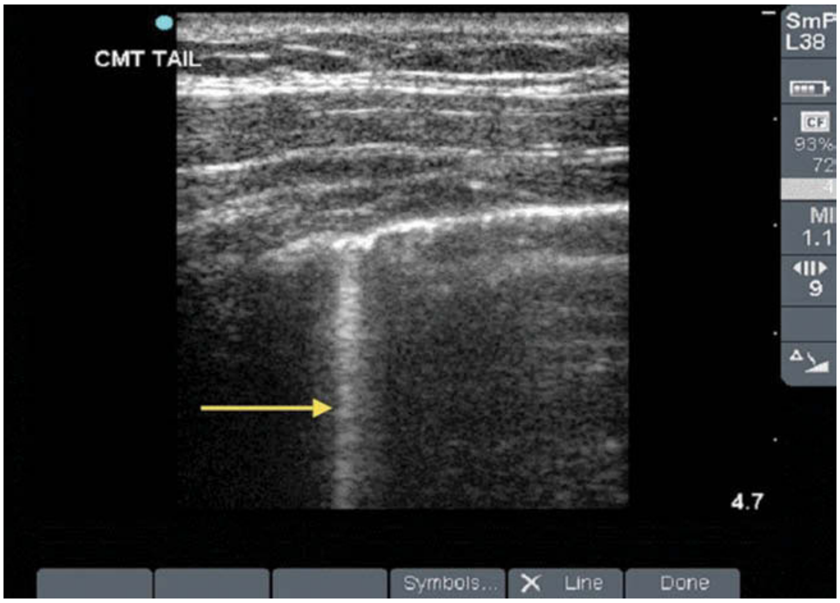


Figure E3.4
Vertical comet tail artifact (yellow arrow) arising from the pleural line, indicating no pneumothorax in this region.

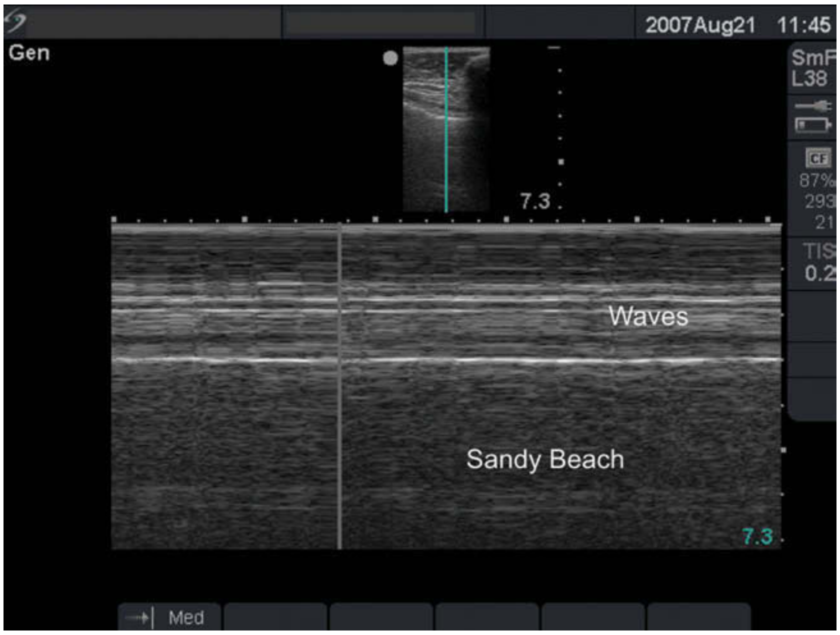


Figure E3.5
“Sandy beach sign” indicating abscence of pneumothorax in this region.

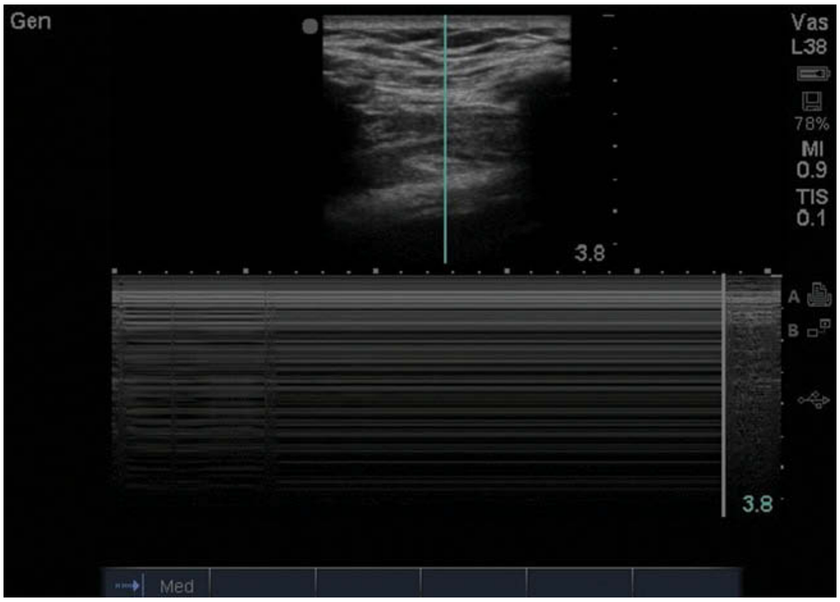


Figure E3.6
“Stratosphere sign” indicating presence of pneumothorax in this region. The sandy beach is replaced by horizontal lines.

and lung sliding end abruptly, and is very specific for PTX.

- If suspicion for PTX persists, use ultrasound *M-mode* setting. Place the M-mode line over the pleural line (between the ribs) and evaluate for the presence of either:
  - *Sandy beach sign:* indicates absence of PTX. Lines of subcutaneous fat and muscle (“waves”) are distinctly separated from sandy/grainy lung motion (“sandy beach”). The pleural line is the bright white line where the waves meet the beach (Figure E3.5).
  - *Stratosphere sign*: indicates presence of PTX. Waves are present but sand is absent (Figure E3.6).

Pearls, pitfalls and myths

- Pleural adhesions can give false positives by restricting sliding along the pleural interface.
- Changing the position of the patient immediately prior to the US scan can miss a small pneumothorax as the air may not have coalesced.
- Do not mistake chest wall movement or underlying cardiac motion for lung sliding.
- If suspicion is high, look in multiple areas of the chest, not just the second intercostal space.
- The exam should be repeated if suspicion is high or conditions change.

Section 4 Emergency echocardiography and IVC evaluation

Sarah R. Williams, MD **and Laleh Gharahbaghian,** MD

Background

Bedside echocardiography can rapidly assess for significant cardiac life-threatening conditions and quickly give vital information in shock states. When combined with critical adjuncts such as ECG, chest X-ray, and physical exam findings, bedside echo can quickly focus the differential diagnosis and speed delivery of appropriately targeted therapy.

Indications

- Rapid evaluation of patients with chest pain and shortness of breath, with or without clinical signs of hypoxia, hypotension and tachycardia.
- Identify presence of pericardial effusion and/or cardiac tamponade.
- Improve diagnostic accuracy in patients with undifferentiated shock or hypotension.
- Establish presence or absence of cardiac activity in cardiac arrest.
- Evaluate for right ventricular (RV) strain and dilatation associated with hemodynamically significant pulmonary embolism (PE).
- Assess left ventricular (LV) function and screen for significant wall motion abnormalities.
- Assist with procedures (e.g., transvenous pacemaker placement and pericardiocentesis).
- Estimation of central venous pressure (CVP).

Probe choice/system presets

- **Ultrasound system:** A basic grayscale system is sufficient for evaluation of pericardial effusions and wall motion, but color flow will be necessary for more advanced applications such as assessment of valvular function.
- **Probe choice:** In most patients, the 2.5–5 MHz curvilinear or phased-array transducer should be used for all elements of cardiac echo. The small-footprint phased-array transducer is preferred due to its small size and easier fit between the ribs.
- **Machine presets:** There are two conventions for cardiac echo, which sometimes causes confusion. Emergency physicians have classically utilized the abdominal (FAST scanning) presets for echo. However, cardiologists use CARDIAC presets and orientation. This convention is to place the screen marker on the RIGHT side of the image (corresponding to the patient's left side), and the probe marker goes towards the patient's LEFT side or head. Either of these conventions will result in correct images. The image texture will change slightly, but the image orientation will be the same. For the purposes of this section, FAST scanning conventions for probe orientation are described.
- **Depth settings:** Adjust system depth settings in each view to visualize the entire heart and surrounding pericardium.
- **Patient positioning:** Place patient in the supine position for all views except for the apical four-chamber view (when the patient should instead be placed on his/her left side if possible).

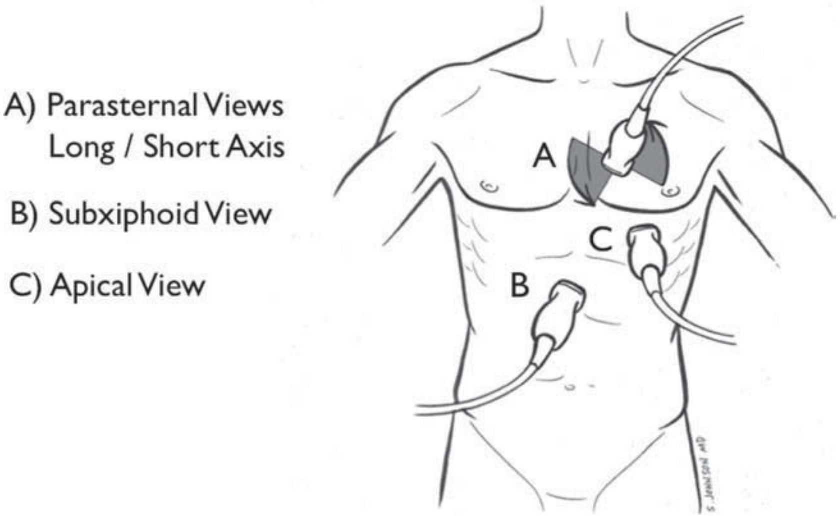


Figure E4.1
Probe placement for echo views. Printed with permission from Phillips Perera, MD.

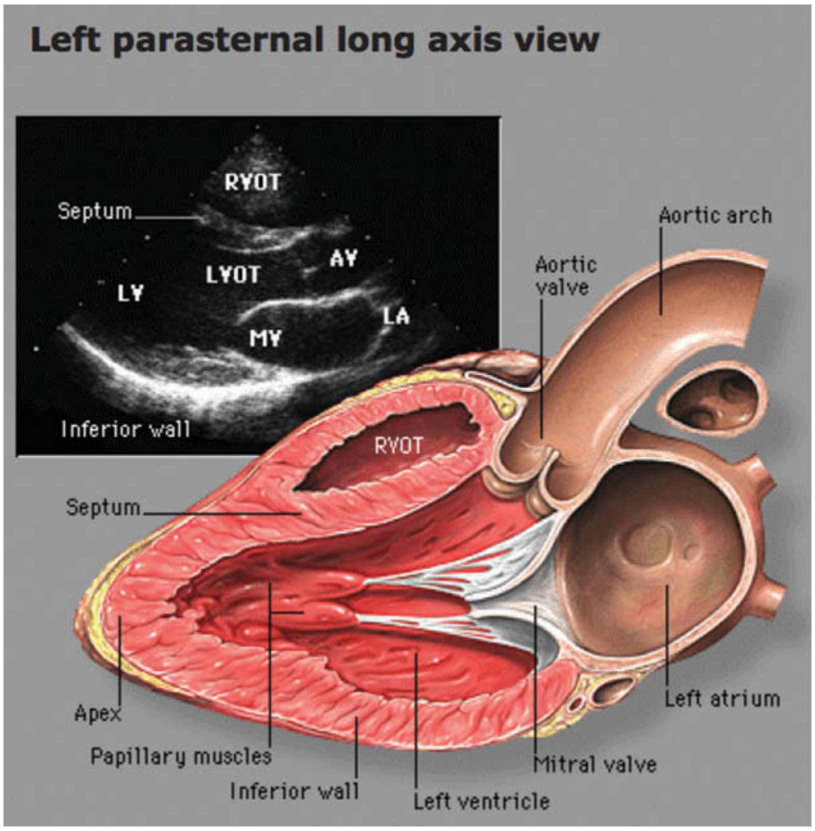

Left parasternal long axis view
RVOT
Septum
AV
LVOT
LV
LA
MV
Inferior wall
Aortic arch
Aortic valve
RVOT
Septum
Apex
Papillary muscles
Inferior wall
Left ventricle
Mitral valve
Left atrium

Figure E4.2
Parasternal long-axis view with US image and correlating anatomy. Courtesy: Patrick J. Lynch and C. Carl Jaffe, Yale University, 2006.

Anatomy and ultrasound technique

The most commonly utilized windows used in ED echo (Figure E4.1):

- Subcostal (subxiphoid) cardiac view (reviewed in the FAST section).
- Parasternal long-axis view, which provides excellent views of the heart valves, pericardial sac, wall motion and descending aorta.
- Parasternal short-axis view, which provides additional information about concentric wall motion and valvular function.
- Apical four-chamber view, which allows estimation of RV size.

Parasternal long axis (PLAX) view

Background

- The most useful view of the heart, allowing for visualization and evaluation of the aortic outflow tract, mitral valve, and descending thoracic aorta (Figure E4.2). Also provides excellent information about pericardial effusions and wall motion.
- Compared with the subcostal view, this view is better tolerated by patients with abdominal pain but may be technically more difficult to obtain (due to interceding lung/ribs/chest wall musculature).

PLAX imaging technique

- The transducer is usually placed in the 2nd–4th intercostal space just to the left of the sternum, with the probe indicator toward the patient's left hip. Do not aim cephalad because the heart is directly under the probe (Figure E4.3).
- The most superficial chamber (near field) is the RV. The LV is oblong and empties into the aortic outflow

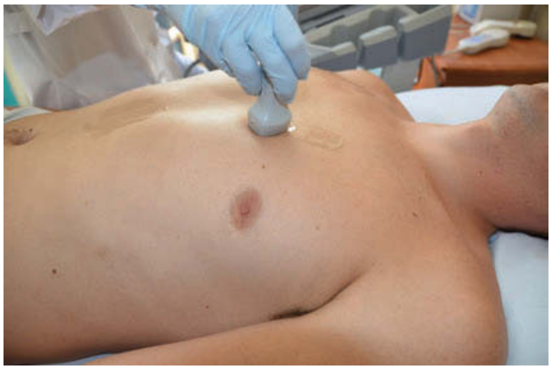


Figure E4.3
Probe placement for parasternal long-axis cardiac view. Note probe marker oriented to left hip (FAST convention).

tract. The mitral valve is visualized between the LA and the LV. The descending thoracic aorta is the circular anechoic structure deep to the heart, behind the pericardium (Figure E4.4).

- If a pericardial effusion is identified in the subcostal view, confirm its presence in the parasternal view. Pleural effusions may be misidentified as pericardial effusions (false positive) in the subxiphoid area (Figure E4.5).

Parasternal short axis (PSA) view

Background

- Provides a circumferential view of the left ventricle and, as the probe is angled superiorly, shows the mitral and aortic valves in their entirety. Provides a sense of circumferential "squeeze" of the LV muscle and relative sizes of the RV and LV (Figure E4.6).

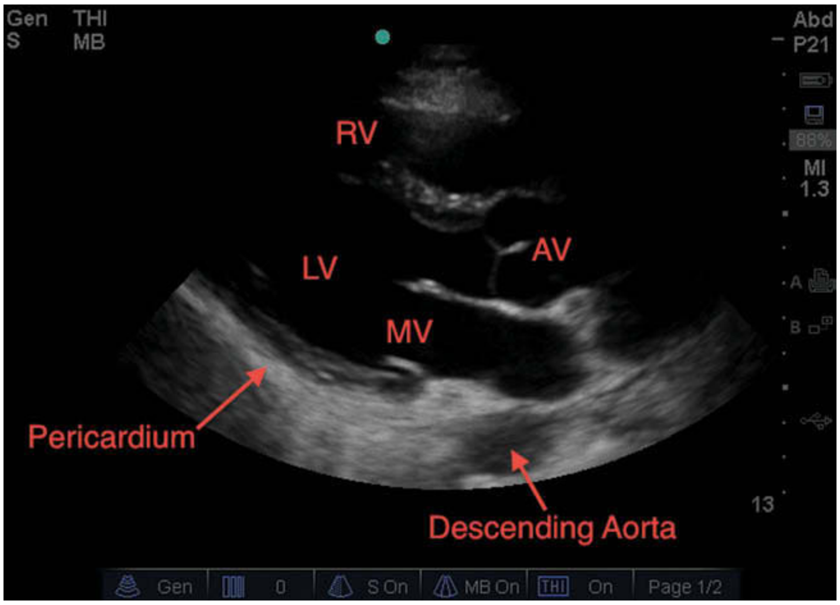

Gen
S
THI
MB
Abd
P21
88%
MI
1.3
RV
AV
LV
MV
Pericardium
Descending Aorta
13
Gen
0
S On
MB On
THI
On
Page 1/2

Figure E4.4
Ultrasound image of normal parasternal long-axis view, also showing descending aorta and pericardium.

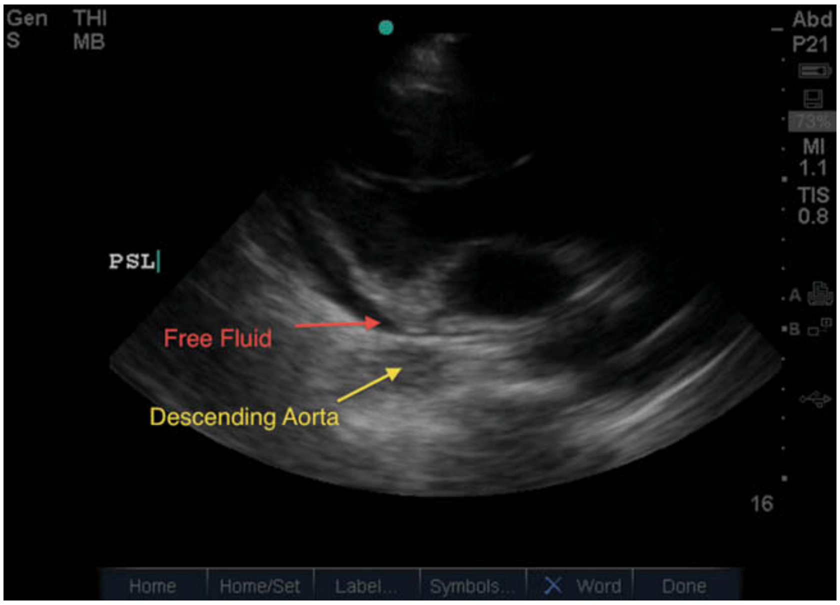

Gen
S
THI
MB
Abd
P21
73%
MI
1.1
TIS
0.8
PSL
Free Fluid
Descending Aorta
16
Home
Home/Set
Label...
Symbols...
Word
Done

Figure E4.5
Parasternal long-axis image with pericardial free fluid. Note that the reflection of free fluid is anterior to the descending aorta, confirming pericardial and not pleural fluid. Compare to E2.21.

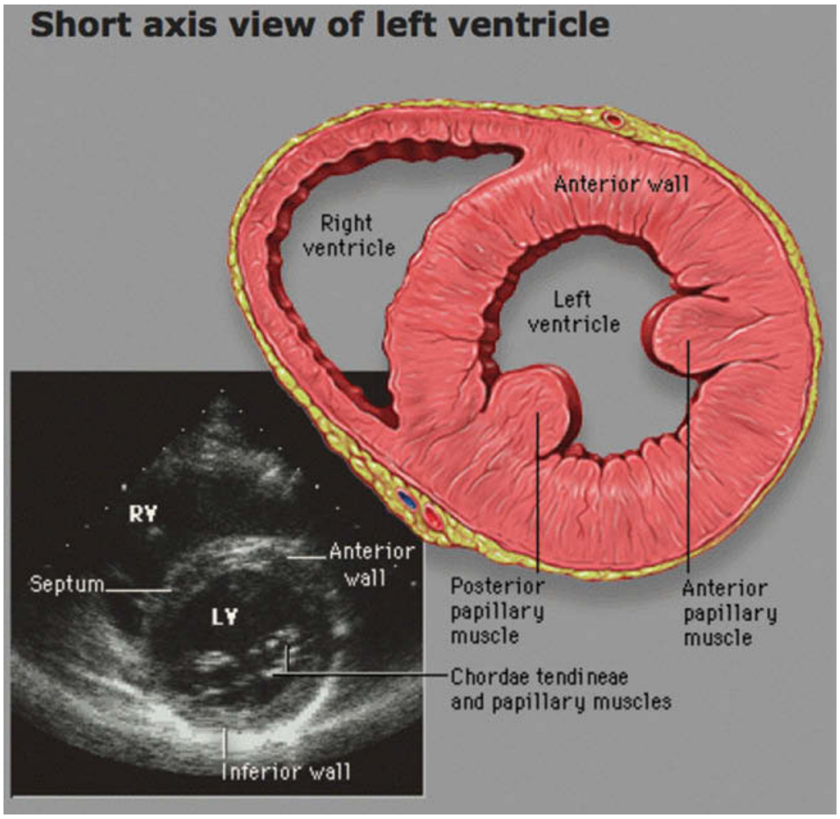

Short axis view of left ventricle
Anterior wall
Right ventricle
Left ventricle
RV
Anterior wall
Septum
LV
Posterior papillary muscle
Anterior papillary muscle
Chordae tendineae and papillary muscles
Inferior wall

Figure E4.6
Parasternal short-axis view with US image and corresponding anatomy. Courtesy: Patrick J. Lynch and C. Carl Jaffe, Yale University, 2006.

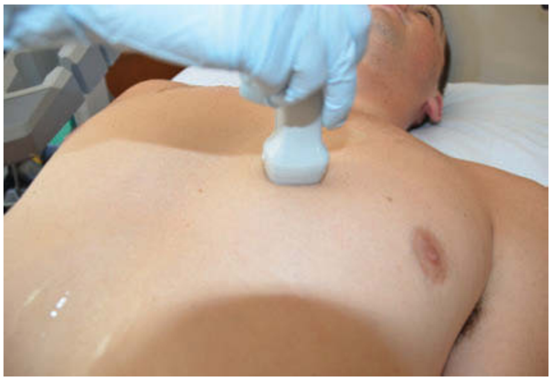


Figure E4.7
Probe placement for parasternal short-axis cardiac view.

PSA imaging technique

- First obtain a good parasternal long-axis view.
- Then rotate the probe 90 degrees clockwise. (Figure E4.7), which will show the heart in the short-axis view (probe marker towards the right hip using FAST conventions) (Figure E4.8).

Apical four-chamber (AP4) view

Background

- A great window to evaluate and compare the relative size of the RV and LV (Figure E4.9), which are also visible on the PLAX and PSA views. The LV should be the larger of the two chambers, and the RV septal wall should not bow into the LV.
- If the RV is larger than the LV in a hemodynamically unstable patient, consider a large pulmonary embolism as the underlying etiology of shock. Smaller pulmonary emboli may not produce this finding; therefore, its absence does not rule out pulmonary embolism.
- The AP4 view also allows excellent visualization of pacer wires into the RV.

AP4 imaging technique

- Ideally, the patient should roll onto his/her left side to swing the heart closer to the chest wall. However, this may not be possible in unstable ED patients, so a supine evaluation is often performed.
- The transducer is placed at the point of maximum impulse (PMI), often just underneath the left nipple (Figure E4.10).
- Using FAST convention, point the probe indicator towards the patient's right side.
- This view evaluates all four chambers simultaneously. The descending aorta can be seen running just to the left of the LA (Figure E4.11). The moderator band may be seen in the RV (usually the smaller of the two ventricles).

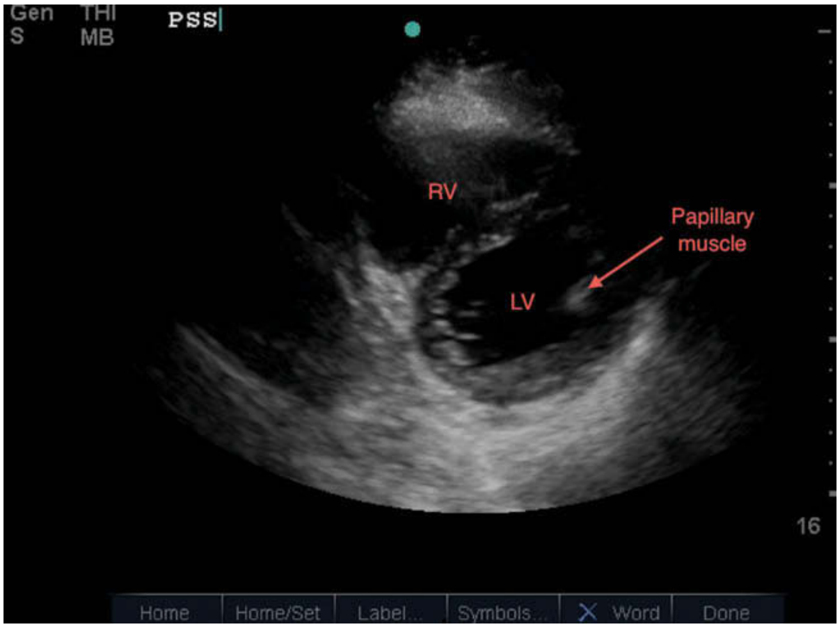

Gen
S
THI
MB
PSS
RV
Papillary muscle
LV
16
Home
Home/Set
Label...
Symbols...
Word
Done

Figure E4.8
Normal parasternal short-axis view, showing the muscular circular left ventricular wall and small RV.

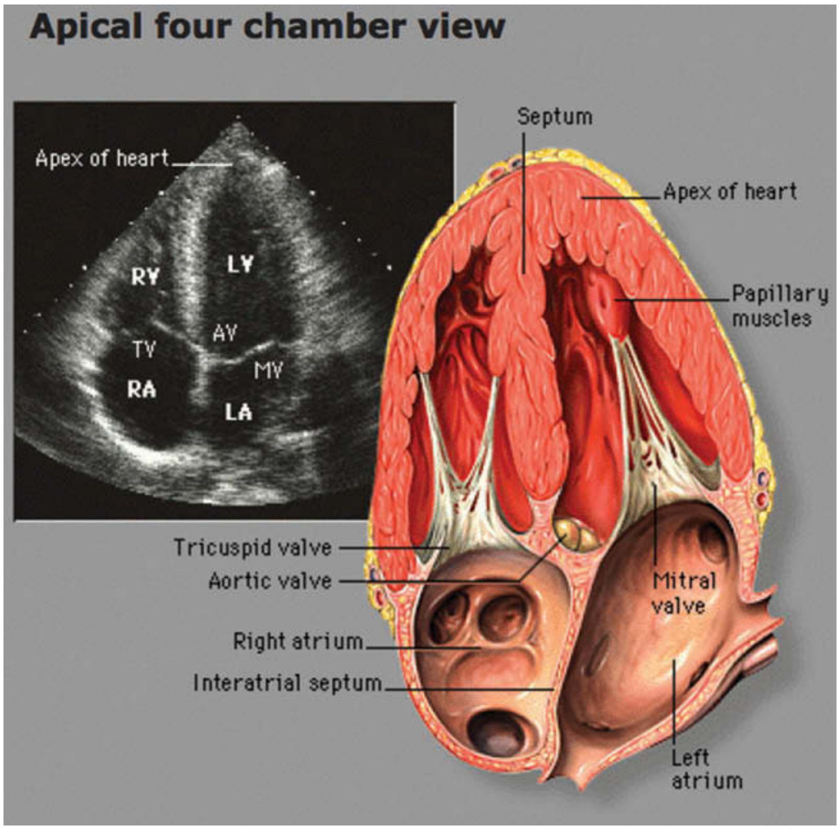

Apical four chamber view
Apex of heart
RV
LV
AV
TV
MV
RA
LA
Septum
Apex of heart
Papillary muscles
Tricuspid valve
Aortic valve
Right atrium
Interatrial septum
Mitral valve
Left atrium

Figure E4.9
Apical four-chamber view with US image and corresponding anatomy. Courtesy: Patrick J. Lynch and C. Carl Jaffe, Yale University, 2006.

Subcostal (subxiphoid) echo technique

Please refer to the FAST section on Cardiac: anatomy and ultrasound technique.

Inferior vena cava (IVC) evaluation

- In shock states, ultrasound of the IVC can quickly evaluate volume status and central venous pressure (CVP).
- The IVC size normally fluctuates with respiration, and its overall size correlates with overall volume status.
- Estimated CVP correlations are listed in Table E4.1.

IVC imaging technique

- Transducer choice: for most adults, a low frequency (2.5–5 MHz) probe is best.

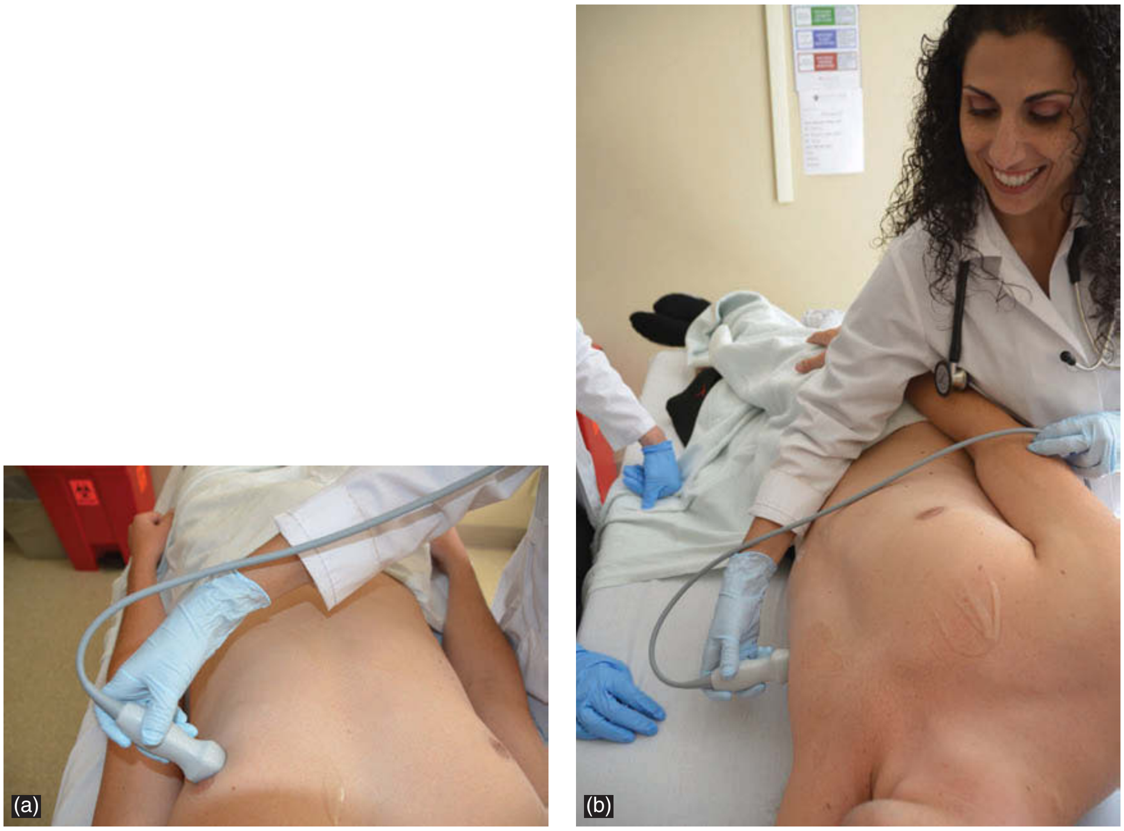


Figure E4.10
(a) Probe placement for apical four-chamber view. The probe is placed at the point of maximal impulse, often below the left nipple. (b) The apical four-chamber view is often enhanced by having the patient roll towards his or her left side.

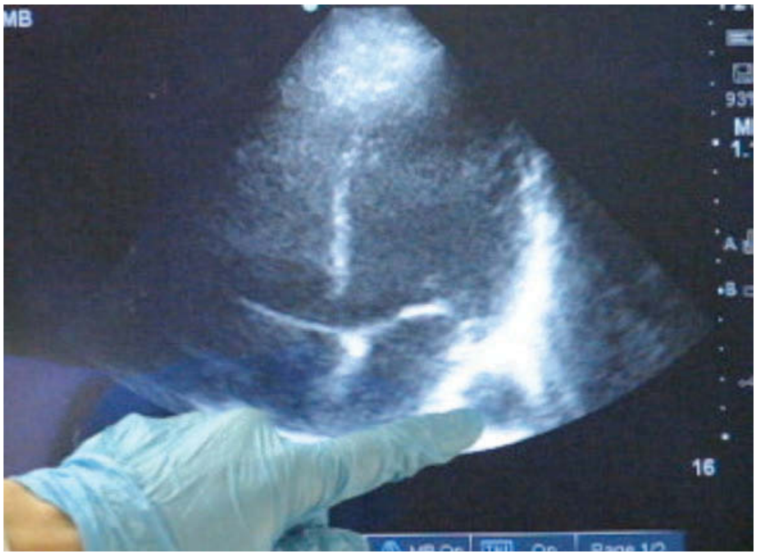


Figure E4.11
Normal real-time apical four-chamber image. The physician is indicating the descending aorta. If in doubt about the left/right orientation, it is helpful to remember that the descending aorta runs next to the true left side of the heart.

- Subcostal transducer placement: hold the probe in the same location as you do for the subcostal cardiac view. Orient the probe longitudinally instead of transverse with the indicator towards the head.
- The aorta is often visualized initially. Aim slightly towards the patient's right of midline to see the IVC. Follow it as it courses through the liver and into the RA (Figure E4.12). Measure it 2 cm distal to where it passes through the diaphragm into the RA.
- Ask the patient to "sniff" and measure the amount of IVC collapse.

Table E4.1 Estimation of CVP using IVC measurements

Estimated CVP	IVC diameter	Collapse with sniff
CVP <5 (dehydrated)	IVC <1.5 cm	>50% collapse
CVP = 5–10 (normal)	IVC = 1.5–2.5 cm	<50% collapse
CVP = 10–15 (slightly elevated pressure)	IVC = 1.5–2.5 cm	<50% collapse
CVP >15 (volume overloaded or other cause of increased pressure)	IVC >2.5 cm	<50% collapse

CVP: central venous pressure; IVC: inferior vena cava.

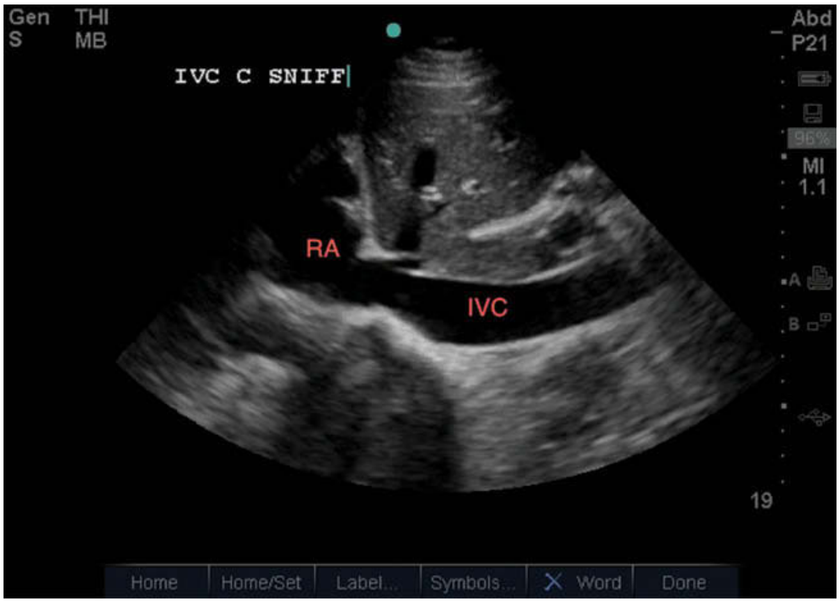


Figure E4.12
IVC view as it courses through the liver and into the right atrium. This IVC is upper limits of normal size. During sniff it collapsed less than 50%, suggesting normal to mildly elevated CVP.

Emergency applications of bedside echo

Pericardial effusion and tamponade

- Appearance: black (anechoic) fluid between the myocardium and pericardium. May appear grey if the blood has clotted.
- In a supine patient, fluid will collect posteriorly first, then anteriorly.
- An anechoic area seen only anteriorly is often just the epicardial fat pad, not a pericardial effusion. Additionally, very small effusions may be normal.
- Acute pericardial effusions may be lethal even if they are not large. Chronic effusions may be sizeable yet tolerated by the patient, as the pericardial fibers have time to stretch.
- Effusions are described based on location (posterior only vs. circumferential), and size (less than or greater than 1 cm)
  - Small: < 1 cm and posterior only
  - Moderate: ≤ 1 cm and circumferential
  - Large: > 1cm and circumferential
- Pericardial tamponade must be excluded if a pericardial effusion is present. Look for signs of tamponade such as the collapse of the RV wall during diastole (Figure E4.13).

Cardiac arrest and shock

- Cardiac echo plays an important role in rapid diagnosis and intervention in cardiac arrest and shock states (Table E4.2).
- During CPR, the best time to perform cardiac echo is during pulse checks. It should not impede other critical interventions.
- Cardiac echo assists with the decision to continue or cease further resuscitation efforts.
  - If a patient has no palpable pulse but has visible organized cardiac wall motion on echo: aggressive resuscitation is indicated.
  - Patients with no pulse and no wall motion on echo: consider cessation of resuscitation.

Emergency pacemaker placement

- Emergency transvenous pacer placement can be facilitated by direct observation of the pacing wire entering the right ventricle (Figure E4.14).
- Confirmation of pacer capture can also be confirmed by seeing ventricular contraction after the pacer spike.

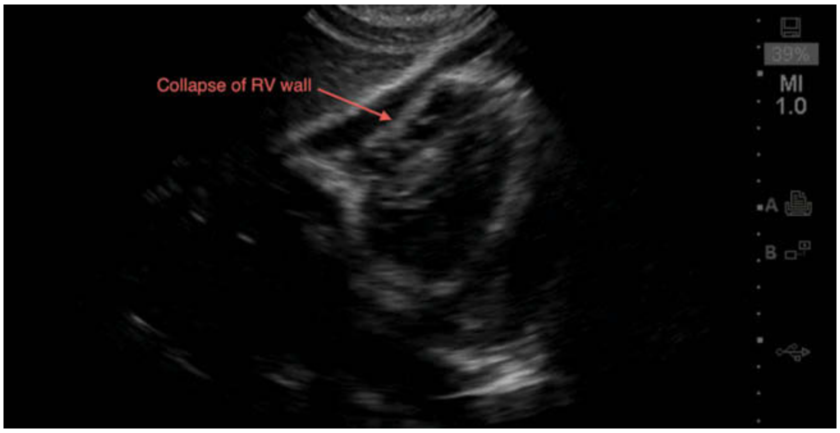


Figure E4.13
Collapse of RV wall indicating tamponade.

Table E4.2 Echocardiography findings in shock

Clinical suspicion	Echo finding	Treatment
Profound hypovolemia	Tachycardia, low LV volume, flat IVC	Aggressive volume resuscitation
Cardiac tamponade	Pericardial effusion, RV collapse, dilated IVC	Pericardiocentesis
Massive PE	Dilated RV, dilated IVC	Anticoagulation, consider thrombolytics
Sepsis	Hyperdynamic, tachycardic	Early goal-directed therapy
Massive MI	Severe hypokinesis	Emergent MI protocol
Severe hyperkalemia	Severe hypokinesis	Electrolyte correction
Cardiac arrest	Cardiac standstill	Consider cessation of efforts if resuscitative measures have failed

IVC: inferior vena cava; LV: left ventricle; MI: myocardial infarction; PE: pulmonary embolism; RV: right ventricle.

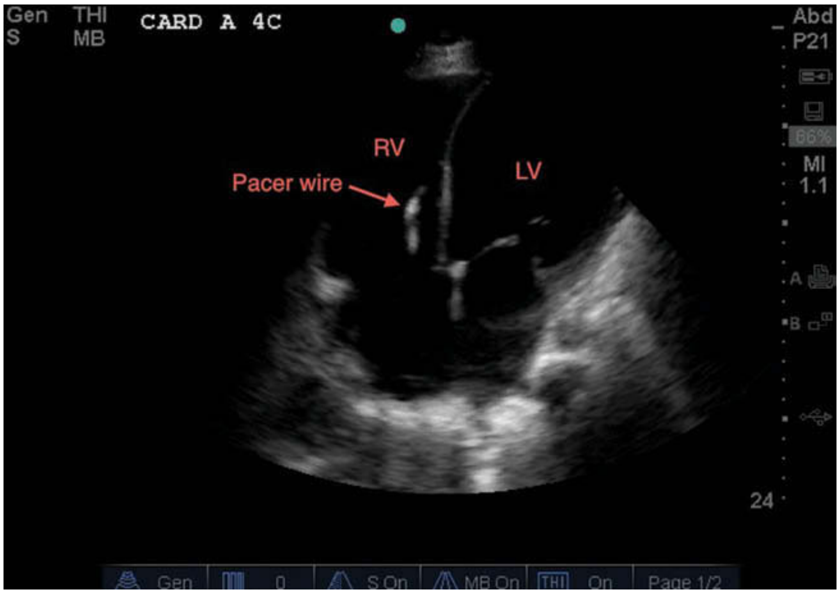


Figure E4.14
Abnormal AP4 view, showing a dilated LV and a pacer wire in the RV.

Thoracic aortic dissection

- Bedside echo may identify the aortic dissection flap on the PLAX view (visualizing the aortic outflow tract and descending thoracic aorta), PSA view (descending aorta), or suprasternal views (aortic arch).
- Bedside echo may identify a pericardial effusion or tamponade associated with a Type A dissection.

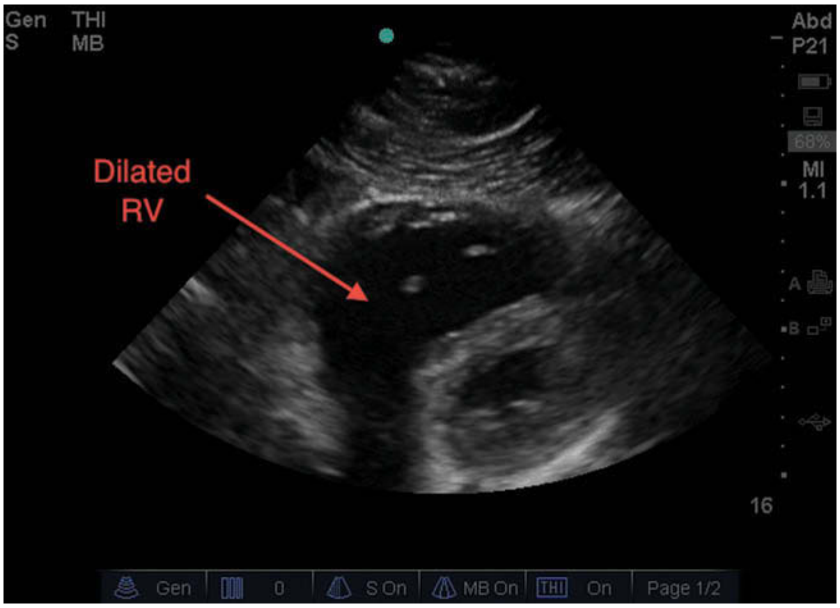

Gen
S
THI
MB
Abd
P21
68%
MI
1.1
Dilated
RV
A
B
16
Gen
0
S On
MB On
THI
On
Page 1/2

Figure E4.15
Abnormal parasternal short-axis view, showing a dilated RV in the setting of a massive pulmonary embolism.

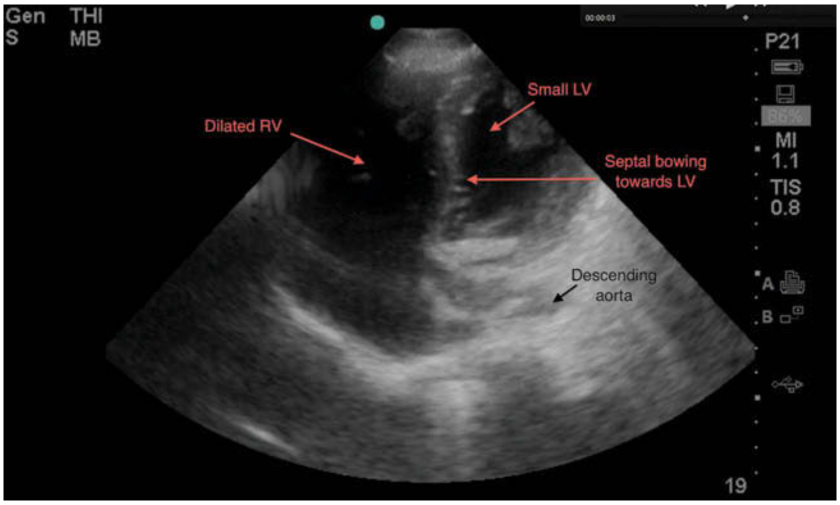

Gen
S
THI
MB
P21
MI
1.1
TIS
0.8
Dilated RV
Small LV
Septal bowing
towards LV
Descending
aorta
A
B
19

Figure E4.16
AP4 view obtained during a code with RV dilation.

- Other imaging modalities (specifically, transesophageal echo and CT angiography) are more sensitive for the detection of aortic dissection than bedside echo.

Hemodynamically significant pulmonary embolism (PE)

- On the AP4 view, a normal RV is usually smaller than the LV, and the wall of the LV usually bows into the RV (due to the higher pressure on the left side).
- In a hemodynamically unstable patient with a large PE, obstruction of RV outflow leads to dilatation and stiffness of the RV(Figure E4.15). On the AP4 view, the RV appears larger than the LV (Figure E4.16).
- Decreased venous return from a large PE may reduce LV filling pressure. This, in association with increased RV pressure, may cause the septum to deviate or bow towards the left (“septal bowing”), a phenomenon most evident during diastole (Figure E4.16).
- Other possible causes of a dilated, hypokinetic RV are primary pulmonary hypertension (PPH), right ventricular infarct and emphysema.
- To help determine the acuity of the RV strain, the normal RV free wall thickness is 2–4 mm. If it is > 5 mm, consider a chronic cause of RV strain, such as PPH.

Section 5 Ultrasound evaluation for abdominal aortic aneurysm

Sarah R. Williams, MD and Laleh Gharahbaghian, MD

Background

- Patients with abdominal aortic aneurysms (AAA) are at increased risk for sudden rupture, a typically fatal event. Early identification of an AAA can be life-saving.
- Bedside ultrasound quickly screens for AAA by evaluating aortic size.
- US typically identifies the aneurysm, not necessarily the bleeding from the aneurysm. US can detect intraperitoneal bleeding from a ruptured AAA but is insensitive for detecting retroperitoneal hemorrhage.
- Emergency bedside US for the presence of AAA is accurate (up to 100% concordance with radiology).

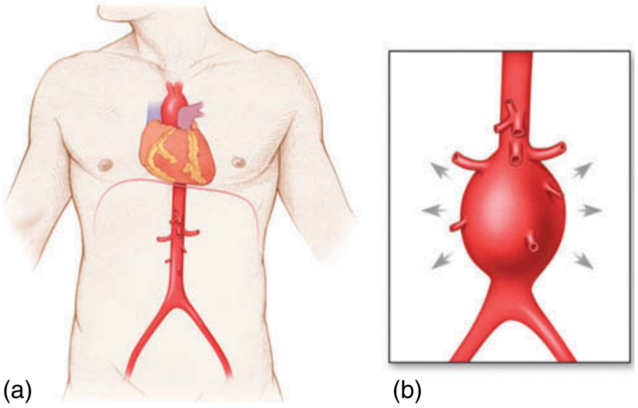


Figure E5.1
(a) Normal aorta. (b) Aortic aneurysm between the renal arteries (projecting laterally) and the iliac arteries (inferior). © Chris Gralapp

- Ideal for the unstable patient, in whom CT cannot be done.
- A hemodynamically unstable patient may be taken directly to the operating room based on the presence of an AAA on US.

Indications

- Chief complaint of abdominal, back or flank pain, especially in the elderly with a history of hypertension.
- Unexplained hypotension.

Probe choice/system presets

- **System choice:** Grayscale imaging is sufficient to evaluate for AAA. However, color flow can help if you are having difficulty identifying the vessel.
- **Probe choice:** In most patients, the 2.5–5 MHz curved or small-footprint phased-array transducer (FAST probe) should be used for the focused AAA evaluation. The larger footprint curved probe may be more effective than the small FAST probe to push bowel gas aside and improve visualization.
- **Machine presets:** Use the ultrasound system abdominal imaging presets. The convention is to place the screen marker on the left side of the image (corresponding to the patient's right side (in transverse orientation) or head (in longitudinal orientation). If these conventions are not followed, the images will be reversed from standard.

Anatomy and ultrasound technique

Aortic anatomy

- The normal aortic diameter is <3 cm (Figure E5.1).
- Longitudinally, the aorta appears as a tubular pulsating structure (Figure E5.2).
- On transverse view, the aorta is a circular, pulsating structure (Figure E5.3a).
- The aorta enters the abdomen at the level of the xiphoid and lies directly anterior to the vertebral bodies. In the epigastrium, the thick walled superior mesenteric artery (SMA) can be seen anterior to the aorta (Figure E5.3a).
- The aorta travels to the right of and parallel to the IVC and bifurcates into the iliac arteries at the level of L4, usually 1–2 cm above the umbilicus (Figure E5.3b).

Aortic imaging technique

- Start with the patient supine, and place the probe at the epigastrium, with the probe indicator pointed towards the patient's right (Figure E5.4a and E5.4b). Locate the circular, pulsatile aorta lying just

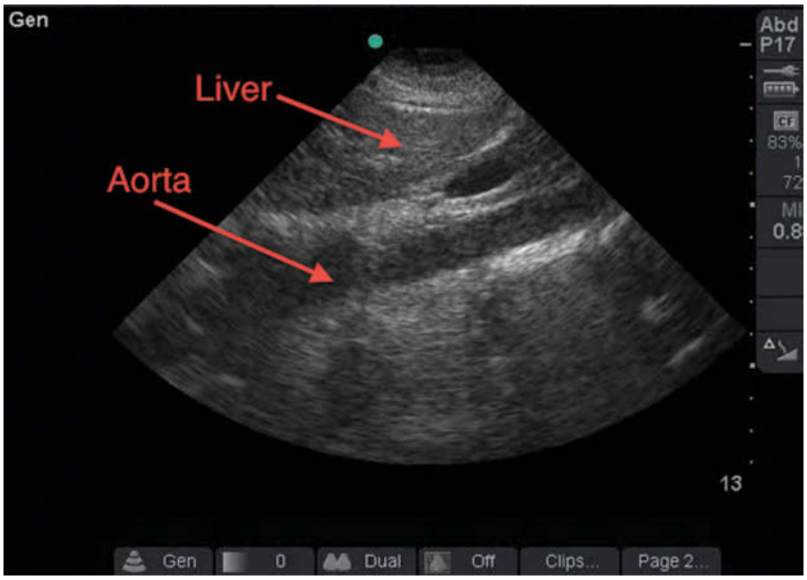

Gen
Abd
P17
CF
83%
1
72
MI
0.8
Liver
Aorta
13
Gen
0
Dual
Off
Clips...
Page 2...

Figure E5.2
Normal aorta in longitudinal orientation. The aorta appears as a dark tube running from left to right.

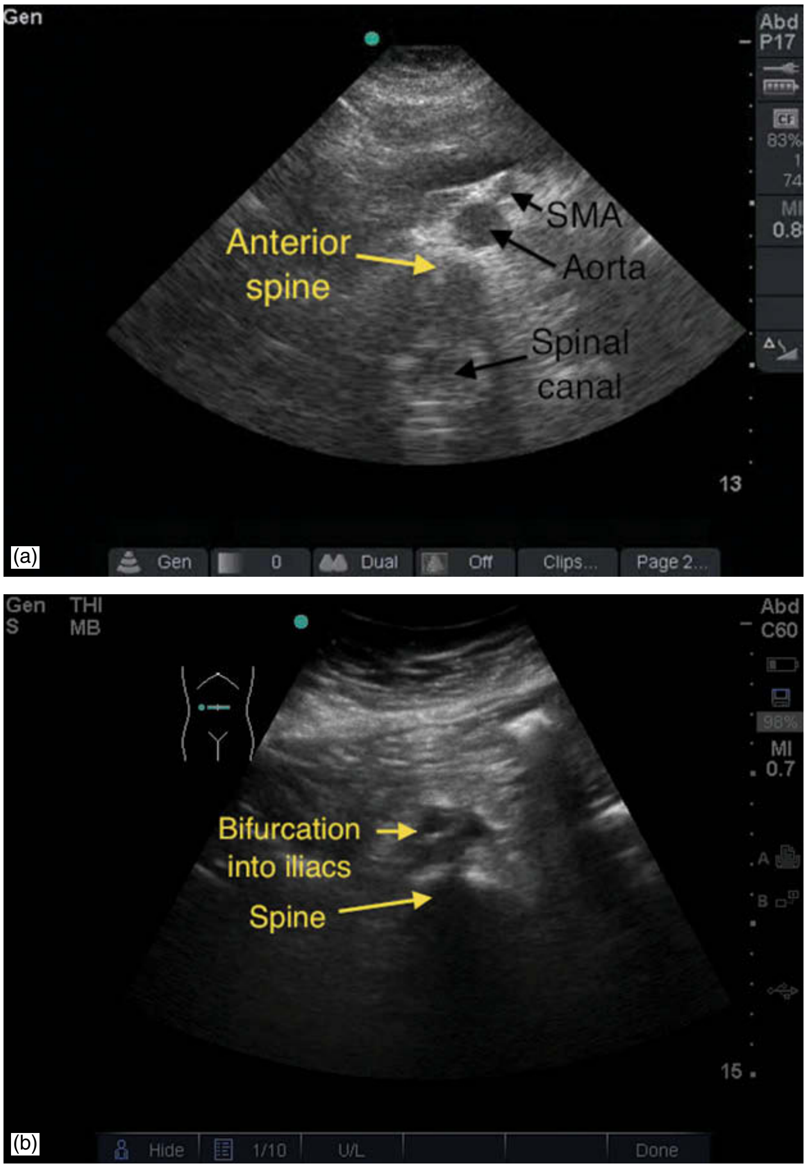

Gen
Abd
P17
CF
83%
1
74
MI
0.8
SMA
Anterior spine
Aorta
Spinal canal
13
(a)
Gen
0
Dual
Off
Clips...
Page 2...
Gen
S
THI
MB
Abd
C60
98%
MI
0.7
Bifurcation into iliacs
Spine
A
B
15
(b)
Hide
1/10
U/L
Done

Figure E5.3
(a) Normal aorta in transverse orientation at the level of the epigastrium. The aorta is a dark circle that runs immediately anterior to the spine. The brightly walled superior mesenteric artery runs anterior to the aorta. (b) The aorta should be followed until it bifurcates into the two iliac arteries (shown), around the level of the umbilicus.

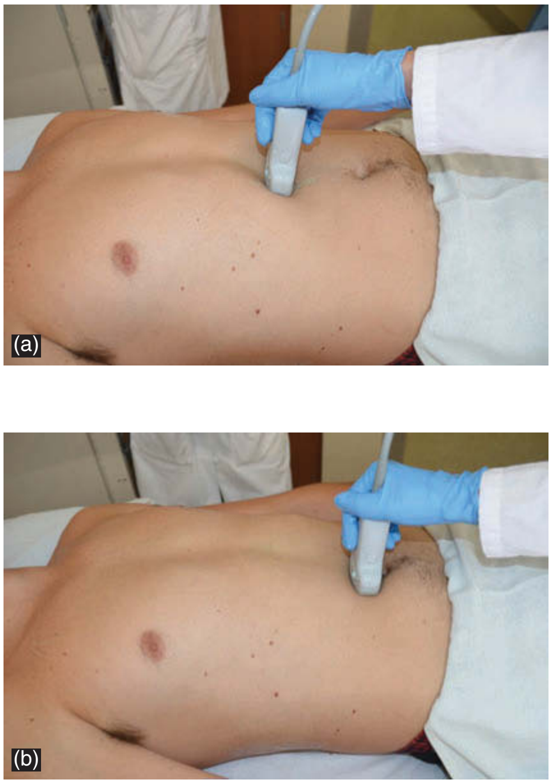


Figure E5.4
Evaluation of aorta-transverse probe placement. Evaluate from the epigastrium (a) to the umbilicus, (b) where the aorta usually bifurcates into the iliacs.

anterior to the vertebral bodies and to the right of the IVC.

- Measure in transverse view from outer wall to outer wall. Turn the probe 90 degrees (indicator pointed towards patient's head) to obtain the longitudinal view (Figure E5.5a and E5.5b). Scan the entire length of the abdominal aorta in both views. Remember that the aorta is not completely evaluated until it is seen in its entirety through to the bifurcation.
- When bowel gas and/or body habitus impedes the view, try these techniques:
  - Change the pressure/angle, push the pannus aside, and/or have the patient bend his/her knees to decrease the abdominal wall tension.
  - Consider applying pressure with a wider footprint probe.

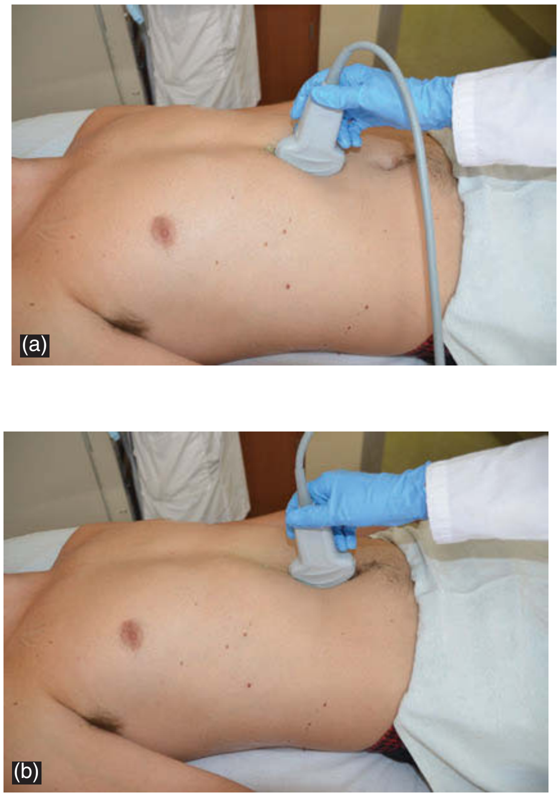


Figure E5.5
Evaluation of aorta-longitudinal probe placement from epigastrium (a) to umbilicus (b).

Abdominal aortic aneurysms

- AAAs are > 3 cm in diameter and are usually located below the level of the renal arteries (95%), and often extend to the iliac arteries (40%).
- AAAs are usually fusiform in shape, but can be saccular. Saccular aneurysms are easy to miss if the entire aorta is not visualized.
- Echogenic thrombus is commonly adherent to the aortic walls. It is important to include the thrombus when measuring the diameter of the aneurysm to avoid underestimating or missing an AAA (Figures E5.6a and E5.6b).

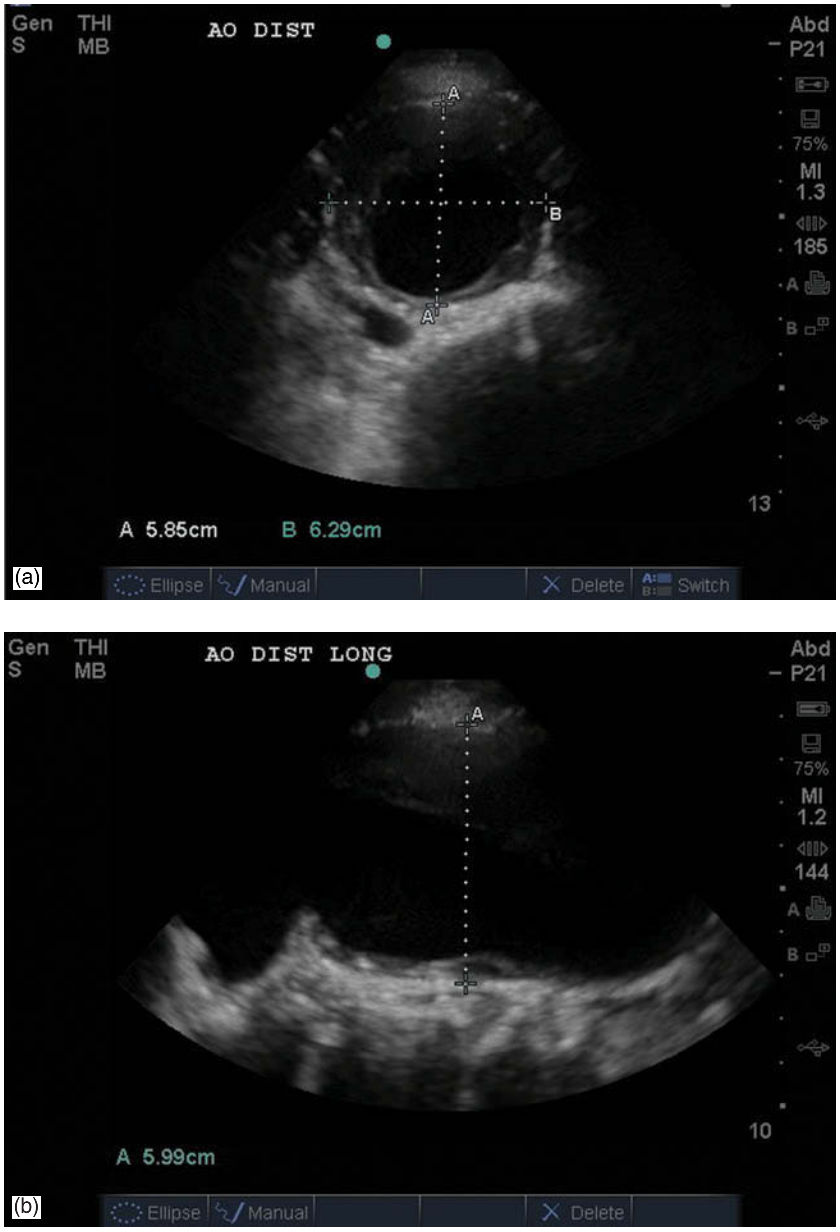

Gen S
THI MB
AO DIST
Abd P21
75%
MI 1.3
185
13
A
B
A 5.85cm
B 6.29cm
(a)
Ellipse
Manual
Delete
Switch
Gen S
THI MB
AO DIST LONG
Abd P21
75%
MI 1.2
144
10
A
A 5.99cm
(b)
Ellipse
Manual
Delete

Figure E5.6
Abdominal aortic aneurysm. Note the echogenic thrombus included in the measurement. Measuring the lumen only would give a false underestimation of the size. (a) Transverse view. (b) Longitudinal view.

Pearls, pitfalls and myths

- Ultrasound of the aorta may be limited by body habitus and bowel gas. Consider CT for these patients.
- Previous aortic repair makes ultrasound challenging. These patients often require CT.
- Do not mistake the IVC for the abdominal aorta. The IVC will have respiratory variation to its pulsations, is more easily compressible and does not have a thick wall. The IVC also runs through the liver, and the aorta is clearly separate. Attempt to find helpful landmarks such as the superior mesenteric artery (which has a bright target of connective tissue around it) and the spine.
- As patients age, the abdominal aorta often becomes tortuous and takes a winding path. If you lose it, go back to the last sighting and start again.

Section 6 RUSH (rapid ultrasound in shock)

Phillips Perera, MD**; Thomas Mailhot,** MD **and Diku Mandavia,** MD

Background

- Rapid ultrasound in shock (RUSH) is an algorithmic ultrasound protocol that rapidly searches for the cause of shock in a critically ill patient.
- The RUSH exam consists of evaluation of the (1) pump, (2) tank, and (3) pipes.

Step 1: Evaluation of the pump

Focused bedside echocardiography (Figure E6.1).

1. Look for a pericardial effusion.
    - When a significant pericardial effusion is present, look for signs of cardiac tamponade (i.e., the presence of diastolic collapse of the right ventricle [RV]).

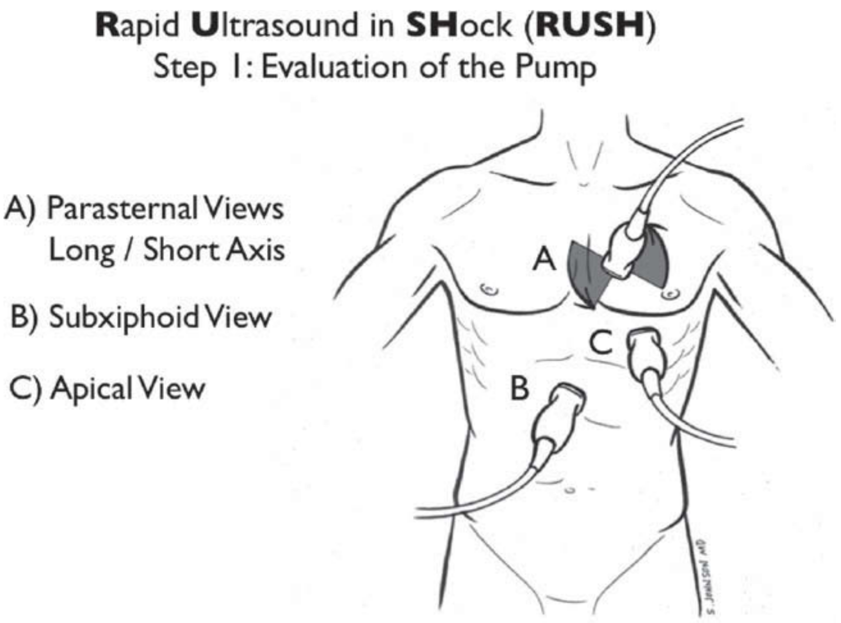

Rapid Ultrasound in SHock (RUSH)
Step 1: Evaluation of the Pump
A) Parasternal Views
Long / Short Axis
B) Subxiphoid View
C) Apical View
A
B
C

Figure E6.1
Rapid ultrasound in shock (RUSH) step 1: Evaluation of the pump. Printed with permission from Phillips Perera, MD.

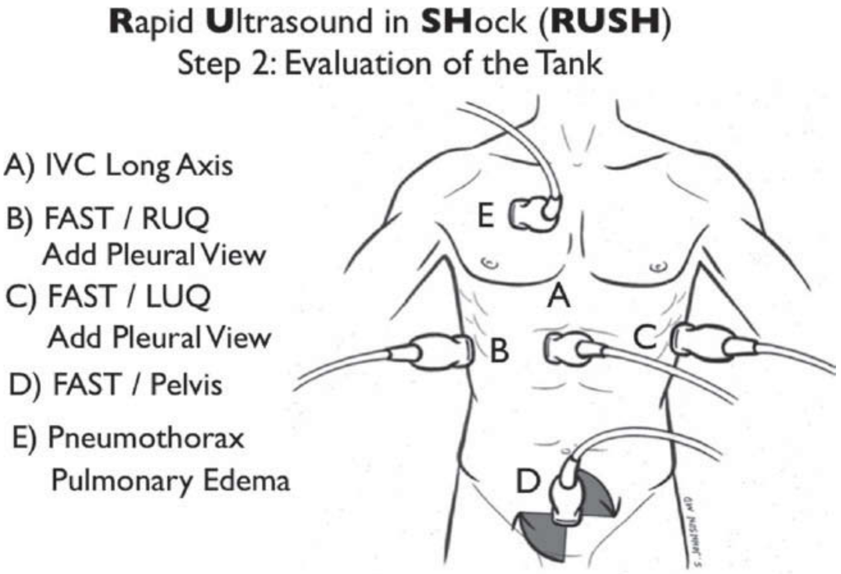

Rapid Ultrasound in SHock (RUSH)
Step 2: Evaluation of the Tank
A) IVC Long Axis
B) FAST / RUQ
Add Pleural View
C) FAST / LUQ
Add Pleural View
D) FAST / Pelvis
E) Pneumothorax
Pulmonary Edema
E
A
B
C
D

Figure E6.2
Rapid ultrasound in shock (RUSH) step 2: Evaluation of the tank. Printed with permission from Phillips Perera, MD.

2. Evaluate left ventricular (LV) global contractility.
    - Poor LV contractility, as evidenced by small percentage change of the chamber's endocardial walls from diastole to systole, is consistent with cardiogenic shock.
3. Look for RV strain.
    - An enlarged RV (as large or larger than the LV) is consistent with RV strain and may suggest an acute pulmonary embolism.

Step 2: Evaluation of the tank

Focused evaluation of the core vascular circuit (Figure E6.2).

1. Assess if the tank is full.
    - **a.** Evaluate the IVC.
        - The IVC should measure < 2 cm in diameter and collapse greater than 50% with deep inspiration or deep sniff maneuver in states of low central venous pressure (CVP) (i.e., correlating to a measurement < 10 cm water).
        - Conversely, a dilated IVC with a diameter of > 2 cm and < 50% collapse with sniff correlates with high CVP (i.e., > 10 cm water).
    - **b.** Evaluate the jugular veins.
        - Place the head of bed at a 30-degree incline and follow the jugular veins from the low neck to the angle of the jaw.
        - Assess the jugular veins in short axis and then long axis to determine the exact position of the meniscus, where the column of blood in the veins ends and the vein walls touch together.
        - A distended jugular vein with an elevated meniscus that fails to significantly collapse with inspiration correlates with high CVP.
        - Conversely, a small jugular vein that collapses low in the neck correlates with low CVP.
    - **c.** Evaluate the lungs.
        - Examination of the lungs to look for pulmonary edema, as manifested by ultrasonic B-lines, can also support the diagnosis of high CVP.
2. Look for leakiness of the tank.
    - Perform the E-FAST (extended focused assessment with sonography in trauma) to identify free fluid in abdominopelvic or thoracic compartments.
    - A positive E-FAST may point to failure of the heart, kidneys or liver, or bleeding (e.g., hemoperitoneum) as the cause of shock.
3. Examine for tank compromise.
    - Tension pneumothorax can cause hemodynamic instability.
    - E-FAST can quickly diagnose pneumothorax at the bedside.

Step 3: Evaluation of the pipes

Focused examination of the major arteries and veins (Figure E6.3).

- The thoracic aorta can be examined for dissection or aneurysm using the parasternal and suprasternal views. though other tests such as transesophageal ECHO and CT angiography are more sensitive
- The abdominal aorta can be evaluated for AAA.
- The femoral and popliteal veins can be screened for deep vein thrombosis (DVT).

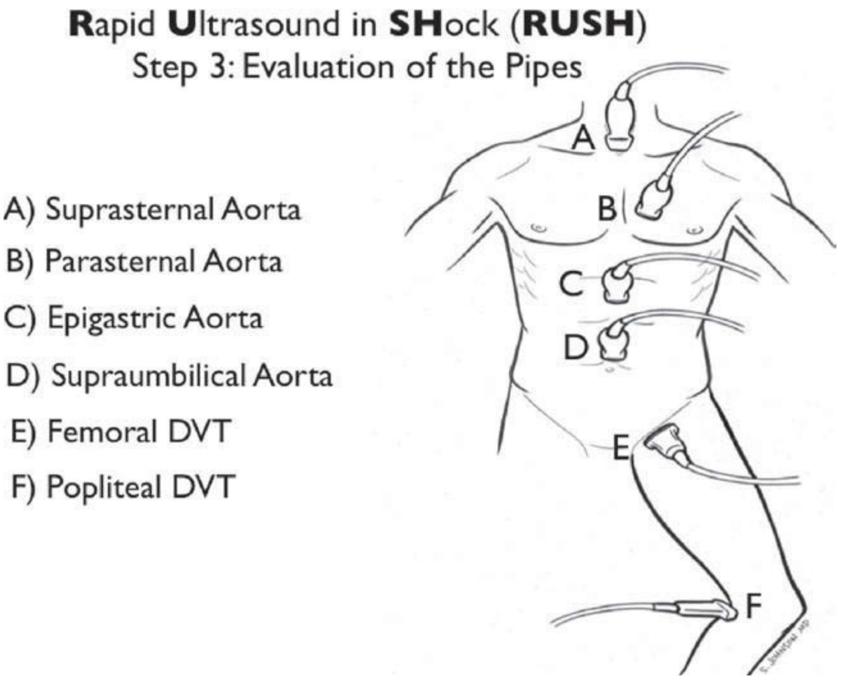


Figure E6.3
Rapid ultrasound in shock (RUSH) step 3: Evaluation of the pipes. Printed with permission from Phillips Perera, MD.

Section 7 Pelvic ultrasound

First trimester pregnancy evaluation

Cathy McLaren Oliver, MD **and Sarah R. Williams,** MD

Background

- Ultrasound (US) is the preferred imaging modality for evaluation of women in their first trimester with symptoms concerning for ectopic pregnancy or miscarriage.
- The goal of emergency pelvic ultrasound is primarily to confirm the presence of an intrauterine pregnancy (IUP).
- Emergency pelvic ultrasound has been shown to decrease time to diagnosis and treatment of ectopic pregnancy.
- In a hemodynamically stable patient without pathologic free fluid, the presence of an IUP on ultrasound reasonably rules out an ectopic pregnancy.
- Any hemodynamically unstable pregnant woman must be considered to have a ruptured ectopic until proven otherwise.
- Pelvic ultrasound can occasionally visualize the ectopic pregnancy; however, indirect signs of ruptured ectopic pregnancy (i.e., positive pregnancy test, no IUP, and free fluid in the pelvis or abdomen) are equally concerning.
- The estimated gestational age (EGA), quantitative beta human chorionic gonadotropin (β-hCG) level and imaging approach (transabdominal vs. endovaginal) should be taken into consideration when interpreting ultrasound images.

Indications

- To evaluate women in the first trimester with symptoms such as abdominal pain and/or vaginal bleeding, which are concerning for ectopic pregnancy, fetal demise, or threatened miscarriage.
- To rapidly screen women of reproductive age with syncope or undifferentiated shock for signs of hemoperitoneum from a ruptured ectopic.

Probe choice/system presets

- **System choice:** Grayscale imaging is sufficient to evaluate for IUP. The capacity to use M-mode is helpful to document fetal heart rate.
- **Probe choice:**
  - **Transabdominal pelvic evaluation:** In most patients, the 2.5–5 MHz large-curved or small-footprint phased-array transducer (FAST probe) should be used.
  - **Endovaginal pelvic evaluation:** Use the mid-frequency 5–7.5 MHz endovaginal (endocavitary) probe.
- **Machine presets:** Use the obstetric imaging presets for optimum results and to use the fetal heart rate software. Abdominal presets are used for FAST components of the evaluation.
- For transabdominal (TA) scanning, place the screen marker on the left side of the image (corresponding to the patient's right side or head). The probe marker should be oriented towards the patient's right side or head, depending on the imaging plane.
- For endovaginal (EV) scanning, the probe marker should rotate between 9 o'clock and 12 o'clock for a full evaluation of the uterus and adnexa.

Anatomy and ultrasound technique

- The uterus is typically midline and positioned posterior to the bladder and anterior to the rectosigmoid colon.
- The pouch of Douglas (cul-de-sac), the space between the colon and uterus, is the most common site for free fluid to collect in the pelvis. It is common to see a small amount of physiologic free fluid there.
- The uterine body and fundus are intraperitoneal; therefore, bleeding from these structures can result in a positive FAST.

Transabdominal imaging technique

- Patient preparation: A full bladder provides an excellent sonographic window for viewing pelvic structures. An IV fluid bolus or retrograde bladder filling with a Foley catheter can expedite the process, if necessary.
- Patient position: supine.
- Probe selection: low frequency.
- Probe placement for transabdominal pelvic scanning is the same as for FAST. Place the probe longitudinally in the midline just superior to the pubic symphysis with the probe marker pointing towards the patient's head (Figure E7.1). Point the probe into the pelvis using the bladder as the sonographic window. This provides a longitudinal view of the uterus and the endometrial stripe should be visible (Figure E7.2).

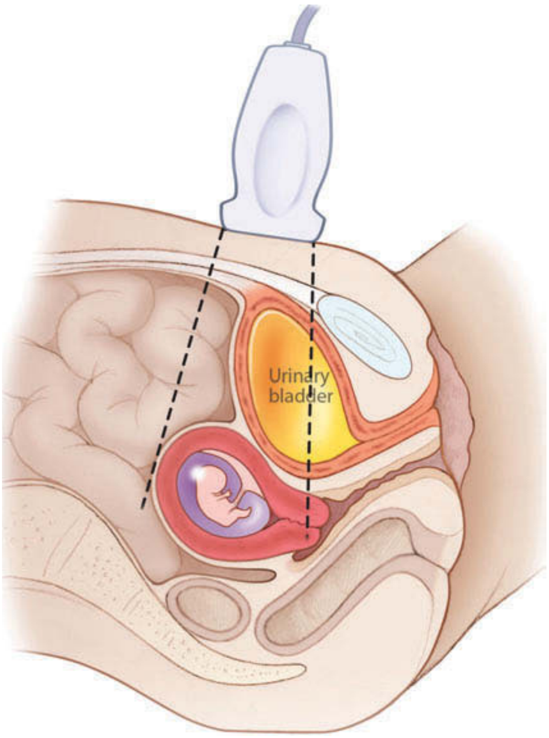


Figure E7.1
Pelvic ultrasound-transabdominal probe placement and anatomy. © Chris Gralapp.

Sweeping the probe from left to right will help if the uterus is not midline and may locate ovaries laterally.

- Next, view the uterus in transverse fashion by turning the probe 90 degrees counterclockwise. The marker will point to the patient's right. Fan the probe superiorly and inferiorly to inspect uterus from fundus to cervix. In both planes look for signs of an IUP and/or an ectopic pregnancy.
- FAST should always be performed as well (see FAST section), as fluid from a ruptured ectopic pregnancy may be more visible in the RUQ than the pelvis.

Endovaginal imaging technique

- Patient preparation: Empty the bladder. Fluid in the bladder increases the distance between the transducer and pelvic structures. Explain the procedure to the patient and offer her the option of inserting the probe herself. Drape the patient appropriately. Endovaginal scanning is ideally performed as part of the pelvic exam.
- Probe selection: endovaginal probe (5–7.5 MHz).
- The probe must be sterilized between patients, and gel must be applied to the probe prior to condom placement. Gel is then placed on the surface of the condom. Failure to do this will cause significant degradation of image quality.
- Patient position: supine with pelvis elevated by pillows for both patient comfort and to help visualize the fundus of uterus.
- Insert (or have patient insert) the probe. The marker dot should be initially pointed upwards towards the ceiling ("12 o'clock"). This provides a long-axis view of the uterus (Figure E7.3). After the uterus is visualized, scan left and right to reveal the ovaries.
- Next rotate the probe 90 degrees to the patient's right ("9 o'clock") to obtain a coronal (short-axis) view of the uterus. In this view, one can often visualize the ovaries and uterus at the same time. Scan superiorly and inferiorly in this plane to look for signs of an IUP or ectopic pregnancy.

Normal early pregnancy anatomy

- Endovaginal (EV) US can visualize an IUP at about 5–6 weeks EGA.
- Transabdominal (TA) US shows an IUP at around 6 weeks EGA (usually 1 week later than by endovaginal examination).
- There are several US findings that suggest the presence of an IUP at 4–6 weeks EGA:

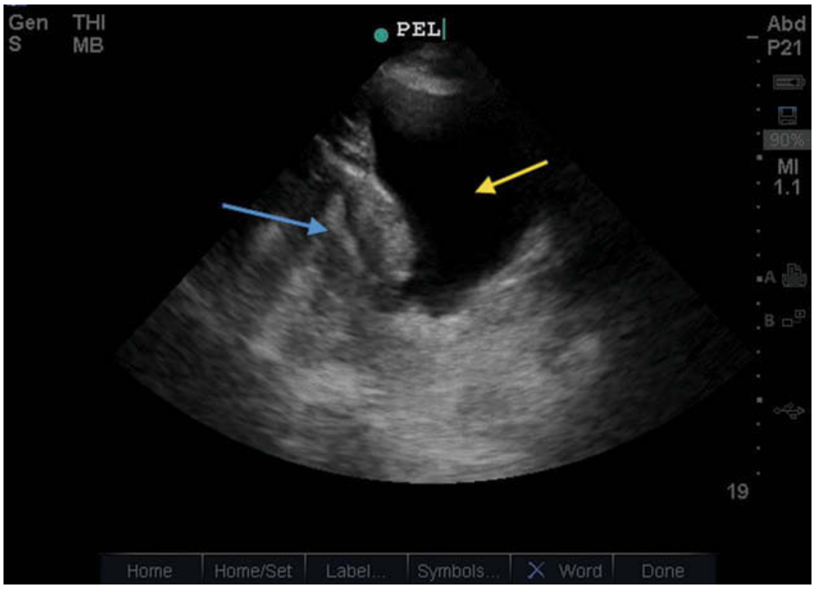


Figure E7.2
Transabdominal pelvic US of non-pregnant uterus (longitudinal orientation). The bladder is seen as a fluid-filled structure (yellow arrow). The uterus is darker and contains the bright endometrial stripe (blue arrow).

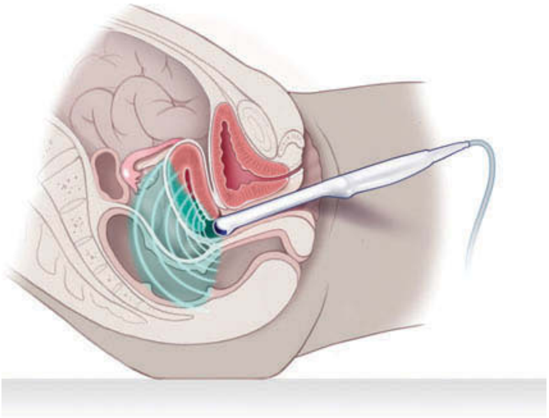


Figure E7.3
Endovaginal probe placement and anatomy. © Chris Gralapp

  - Some experts consider a *gestational sac* (anechoic sac within the uterus) with a *double-decidual sign* (two echogenic uterine rings surrounding gestational sac) to be the first definitive sign of an IUP (Figure E7.4). These findings are seen at 5.5–6 weeks by TA US and 4.5–5 weeks by EV US. These findings are not always present and require experience to interpret. A pseudosac of an ectopic pregnancy can look very similar to a gestational sac but does not have the double decidual sign.
- The next finding is the *yolk sac*, an anechoic thin-walled spherical structure within the gestational sac that appears as a ring (Figure E7.5). Some experts consider this to be the first definitive sign of an IUP. It is visible by 5–5.5 weeks by EV US and 6–6.5 weeks by TA US.
- The next sign of an IUP is the *fetal pole*. It appears as a thickening of or small mass on the yolk sac wall. This is seen initially at 5.5–6 weeks by EV US and 7 weeks by TA US.
- Cardiac activity can be visualized at 6 weeks by EV US and 7 weeks by TA US. A normal fetal heart rate (FHR) confirms fetal viability at time of scanning, and can be measured by M-Mode (Figure E7.6).
- Transabdominal scanning is often but not always sufficient to document an IUP at this stage and beyond, depending on the patient's body habitus and bladder volume (Figure E7.7).

Ectopic pregnancy findings

- Because ruptured ectopic pregnancy is a life-threatening emergency, a FAST should be performed initially on all suspected cases. Free fluid in Morison's pouch correlates highly with need for emergent operative intervention (Figure E7.8).
- An empty uterus with a positive pregnancy test is an ectopic pregnancy until proven otherwise (even in the absence of free fluid).
- Definite signs of ectopic pregnancy (these findings are rare in the ED):
  - Embryo with cardiac activity outside of uterus.
  - Gestational sac with embryo or yolk sac outside of uterus.
- Nonspecific signs of ectopic pregnancy:
  - Free fluid in the abdomen (Figure E7.8) or pelvis (Figure E7.9) is seen in two-thirds of ectopic pregnancies, and is the only US finding in 15% of ectopic pregnancies. To ensure detection of this important finding, all regions of the FAST exam should be visualized, not just the pelvic view

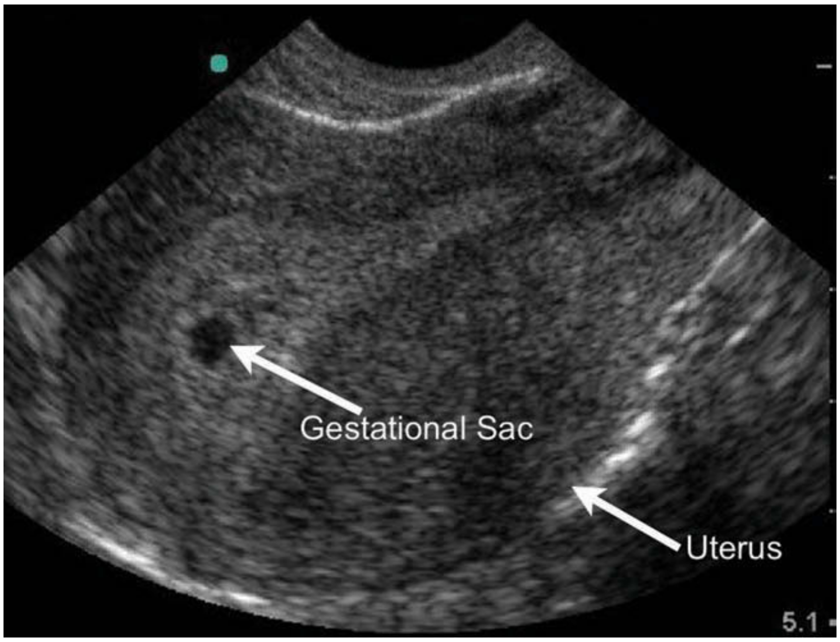


Figure E7.4
Endovaginal US showing the uterus in its long axis. The dark gestational sac can be seen, with a double decidual reaction around it, indicating a very early IUP. Care should be taken at this stage not to mistake the gestational sac with a pseudosac, which can be seen in ectopic pregnancies.

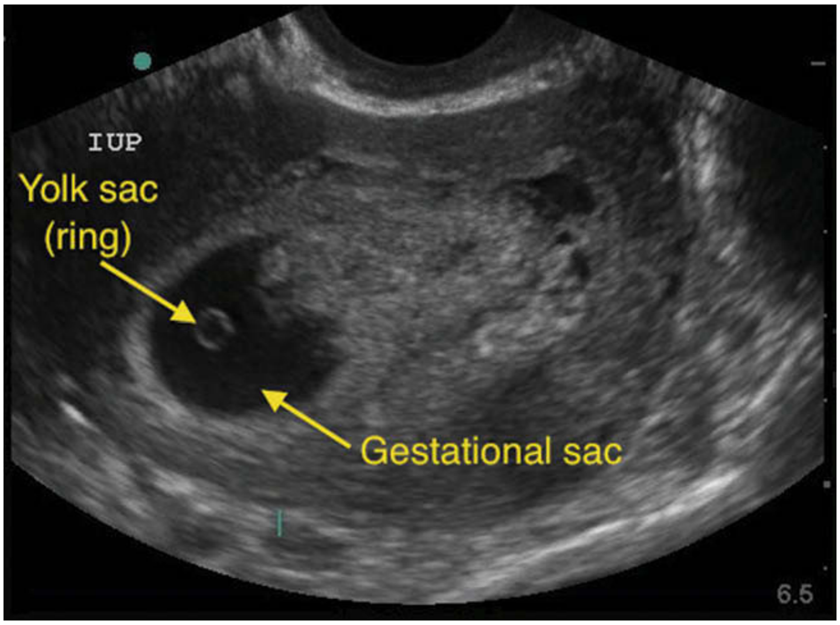

IUP
Yolk sac
(ring)
Gestational sac
6.5

Figure E7.5
Endovaginal US showing the uterus in its long axis. The dark gestational sac can again be seen. The bright yolk sac can clearly be seen within it. This is the earliest reliable indicator of an IUP.

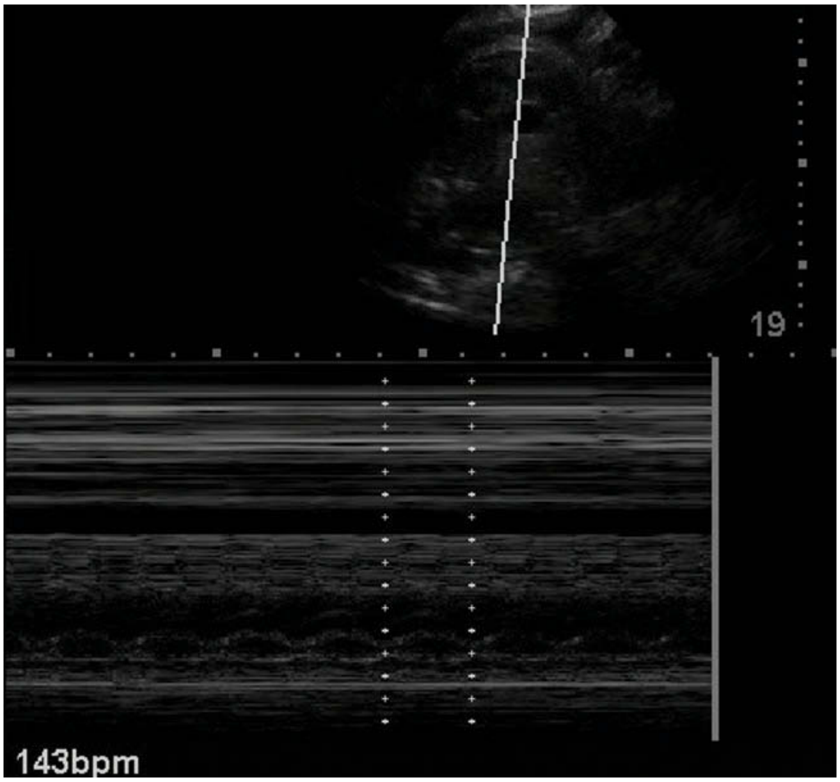


Figure E7.6
Normal fetal heart rate, measured with M-mode.

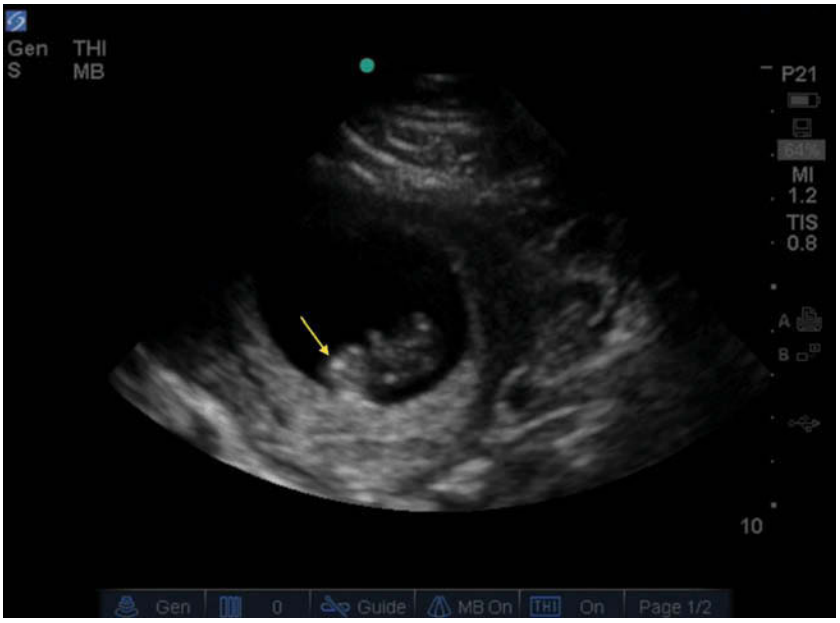
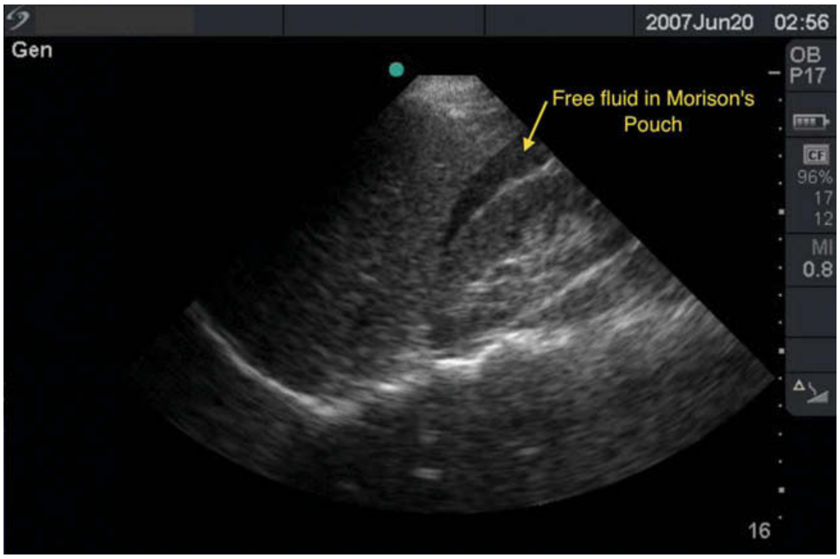


Figure E7.7
Normal early intrauterine pregnancy (IUP). The arrow is pointing to the head of the fetus.



Free fluid in Morison's Pouch

Figure E7.8
Ruptured ectopic pregnancy with positive FAST exam. In this case, emergency obstetric consultation was obtained and patient was taken quickly to the operating room.

(Figure E7.8: this case revealed only trace free fluid in the pelvis).

- A less common finding is a *tubal ring*, a hypoechoic structure surrounded by a relatively thick echogenic ring, located in the adnexa.
- A complex adnexal mass may be seen with ectopic pregnancies, but its absence does not rule out an ectopic pregnancy.

Pearls, pitfalls and myths

- US should be performed on pregnant patients even if the β-hCG is low. Forty percent of ectopic pregnancies have a β-hCG < 1,000 mIU/mL. Definitive or indirect US signs of an ectopic may be present.
- In cases of ruptured ectopic pregnancy, free fluid may collect in the RUQ as opposed to the pelvis. A transabdominal FAST should be performed in addition to the pelvic US. Free fluid in Morison's pouch (a positive FAST) may signify a ruptured ectopic pregnancy requiring emergent operative intervention.
- A pseudogestational sac from an ectopic pregnancy may have a similar appearance to a true gestational sac. In early pregnancy, look for a yolk sac to confirm an IUP.
- Ensure that a normal-appearing pregnancy is actually located in the uterus, not in the adnexa or

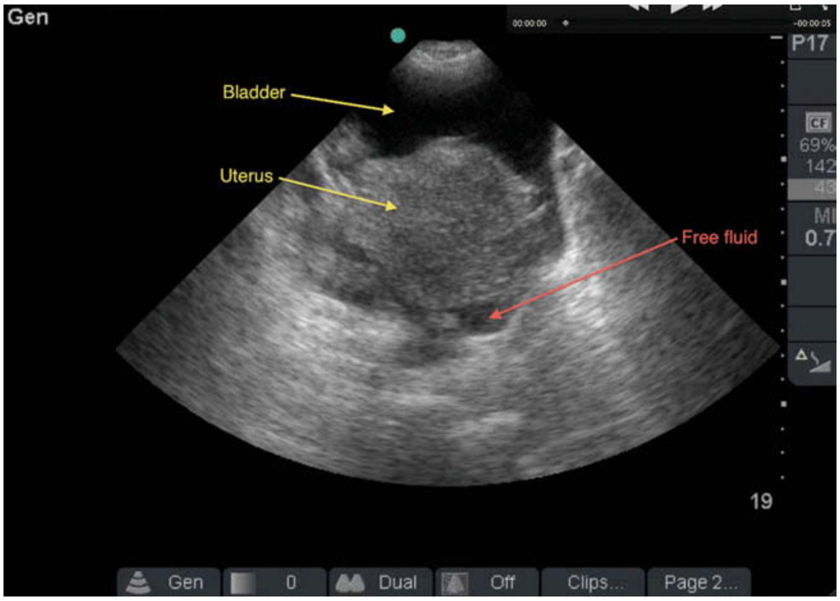


Figure E7.9
Free fluid behind uterus (transverse transabdominal pelvis view). See also Figure E2.14.

cornua. A distinct healthy perimeter of myometrium must be clearly visualized around the entire pregnancy in two planes to confirm a normal pregnancy.

- In a patient with a positive pregnancy test, the key US findings suggestive of ectopic pregnancy are lack of an IUP or a positive FAST.
- The goal of ED ultrasound is to identify emergent pathology. Identification of subtle fetal abnormalities is beyond the scope of this evaluation.
- Current guidelines recommend minimizing fetal US exposure and avoiding power and color flow Doppler imaging (if possible) to decrease the energy to which the fetus is exposed.

Section 8 Biliary evaluation

Sarah R. Williams, MD

Background

- Biliary disease, a common cause of acute abdominal pain, is a spectrum of disorders ranging from biliary colic (extremely painful but not life-threatening) to acute cholecystitis (significant morbidity and mortality with delayed diagnosis).
- Biliary US is a cost-effective and accurate approach to imaging the biliary system compared with CT and hepatobiliary iminodiacetic acid (HIDA) scans. Biliary US is superior to CT in estimating the number and type of gallstones and has the additional benefit of no ionizing radiation.

Indications

- Abdominal pain, usually right upper quadrant (RUQ) or epigastric, but may be generalized.
- New-onset jaundice.
- Screening test for identifying the etiology of sepsis in a non-verbal patient.

Probe choice/system presets

- **System choice:** Grayscale imaging is sufficient to evaluate for emergency biliary pathology. However, color flow Doppler can help to differentiate the bile duct from the portal vein.
- **Probe choice:** In most patients, the 2.5–5 MHz curved or small-footprint phased-array transducer (FAST probe) should be used. The small-footprint probe is helpful when the GB sits high and needs to be viewed between ribs. The larger footprint curved probe provides excellent images when the GB lies below the costal margin.
- **Machine presets:** Use the US system abdominal imaging presets. The convention is to place the screen marker on the left side of the monitor, which corresponds to the patient's right side in the transverse orientation.
- **Probe marker:** Orient the probe so that the probe marker points towards the patient's right side or head, then rotate the probe so that it evaluates the gallbladder in its long and short axis.

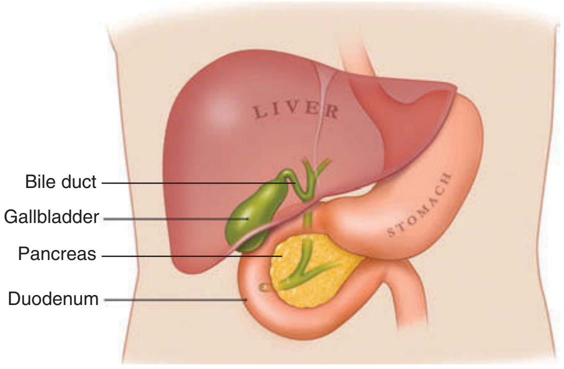


Figure E8.1
External anatomy of gallbladder. © Chris Gralapp

Anatomy and ultrasound technique

Biliary anatomy

- The biliary system includes the organs and ducts (bile ducts, gallbladder, and associated structures) involved in the production and transportation of bile (Figure E8.1).
- Emergency US assessment of the biliary system focuses on three key structures (Figure E8.2):
  1. Gallbladder (most anterior hypoechoic structure)
  2. Portal vein (brightly walled vein)
  3. Bile duct (just anterior to portal vein)

Biliary imaging technique

- Start with the patient supine, and place the probe in the RUQ (Figures E8.3a and E8.3b). Locate the hypoechoic GB, usually the most anterior fluid-filled structure. If in doubt, the portal vein and main lobar fissure of the liver are useful landmarks.
- Image the GB in both longitudinal (Figure E8.2) and transverse orientations(Figure E8.4).
- Moving up between the ribs will sometimes aid in visualization, as will rolling the patient on his/her left side (Figure E8.3b).
- During respiration, the GB often comes into better view as it moves downward with the diaphragm.
- When the gallbladder is difficult to see in the standard RUQ subcostal position, consider swinging the probe around to the side and use the liver as the sonographic window (Figures E8.5 and E8.6).

Biliary disease findings

- Gallstones
  - Evaluate for the presence of gallstones, which are usually hyperechoic (bright) with a dark shadow behind (Figure E8.7).

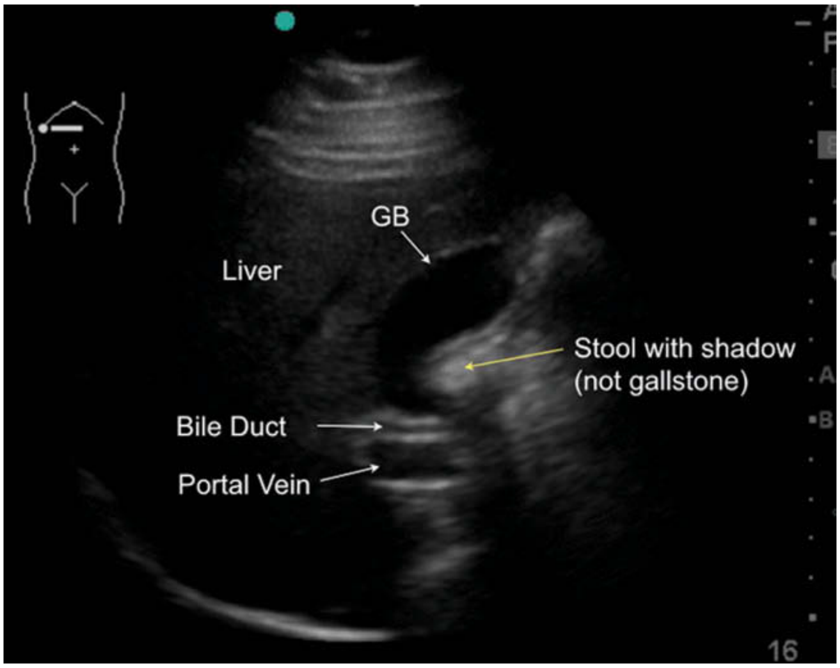
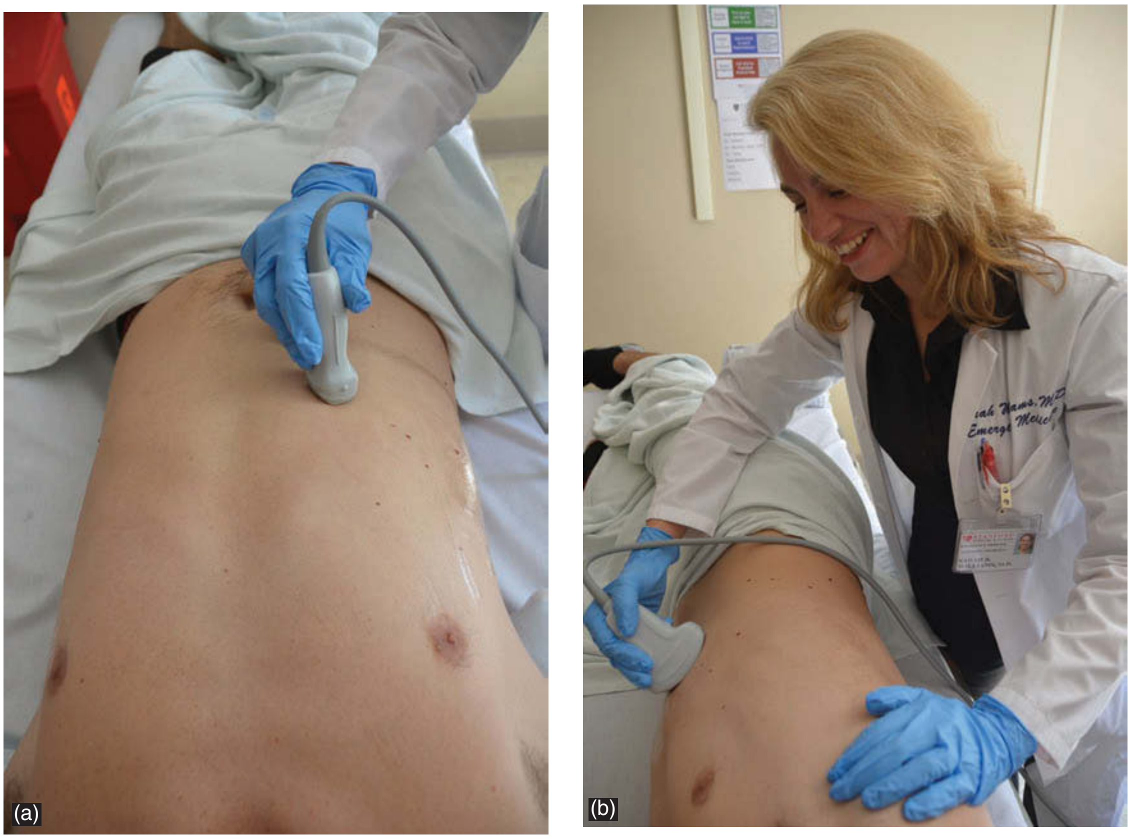


Figure E8.2
Normal gallbladder anatomy, demonstrating the gallbladder in its longitudinal orientation. Note the thin anterior gallbladder wall, fluid-filled gallbladder, and narrow bile duct running just anterior to the bright walls of the portal vein. This image demonstrates stool within the intestines casting a shadow, a potential pitfall. Gallstones also cast shadows, but are within the walls of the gallbladder. If in doubt, the patient or the probe can be turned to view a different orientation.



Figures E8.3
(a) Demonstration of the subcostal approach to the gallbladder. Rotate the probe 90 degrees through both its long and short axis to fully evaluate the gallbladder. (b) Rolling the patient to his or her left side can often help with visualization of the gallbladder.

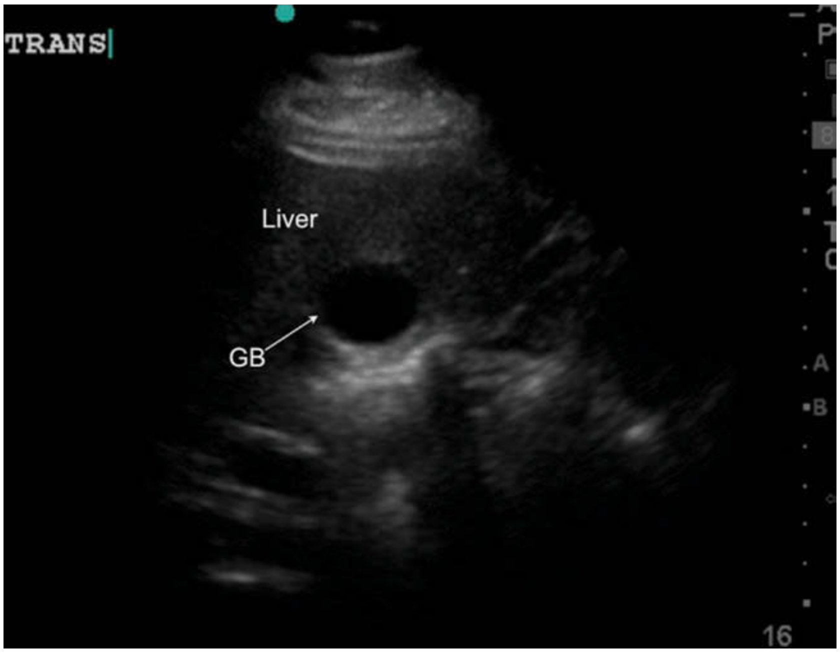


Figure E8.4
Gallbladder in its transverse orientation; appears circular.

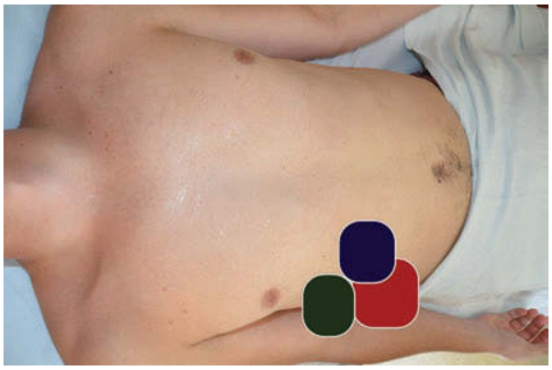


Figure E8.5
Examples of probe placement zones that may aid in visualizing the gallbladder. The small-footprint transducer is useful in evaluating the blue and green zones, between the ribs. The green zone may also be approached from the side (as in Figure E8.6).

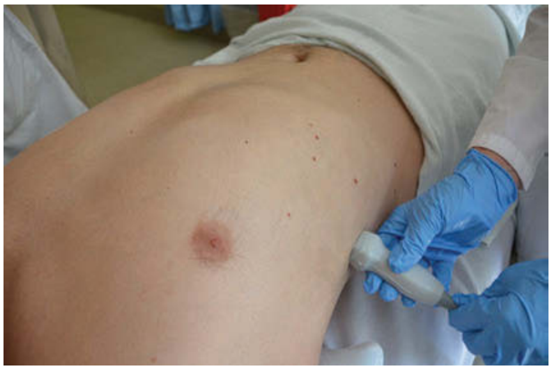


Figure E8.6
Lateral view. This is similar to the Morison's pouch evaluation in the FAST exam; however, instead of pointing towards the flank (as in the FAST), the probe points towards the anterior abdominal wall.

  - Look carefully at the neck of the gallbladder (the junction of the GB and bile duct). Gallstones may be impacted here, causing pain (Figure E8.8).
  - If you are uncertain whether an object is a polyp or gallstone, have the patient roll onto his/her left side. A gallstone will usually move; a polyp will adhere to the wall.
- Anterior GB wall
  - Measure the anterior wall of the gallbladder. Wall thickness > 3 mm may indicate cholecystitis.
- Pericholecystic fluid
  - Evaluate for pericholecystic fluid (fluid around the GB), another marker of cholecystitis.
  - This finding may also be seen with liver disease or congestive heart failure.
- Sonographic Murphy's sign
  - Observe the patient's reaction to compression of the GB with the US probe. If the patient experiences pain, a "sonographic Murphy's sign" is positive and suggestive of cholecystitis.
- Biliary ductal dilatation
  - Locate the common bile duct and measure its width (inner wall to inner wall). Normal bile duct width is < 5 mm in younger patients.
  - The bile duct may dilate with age. A good rule of thumb is that for every decade over 50 years, the bile duct enlarges by 1 mm (i.e., 6 mm at 60 years, 7 mm at 70 years)
  - Bile duct dilatation may indicate biliary obstruction, either from a gallstone or mass.

Biliary pathology

- Biliary colic
  - Acute RUQ or epigastric pain with gallstones evident.

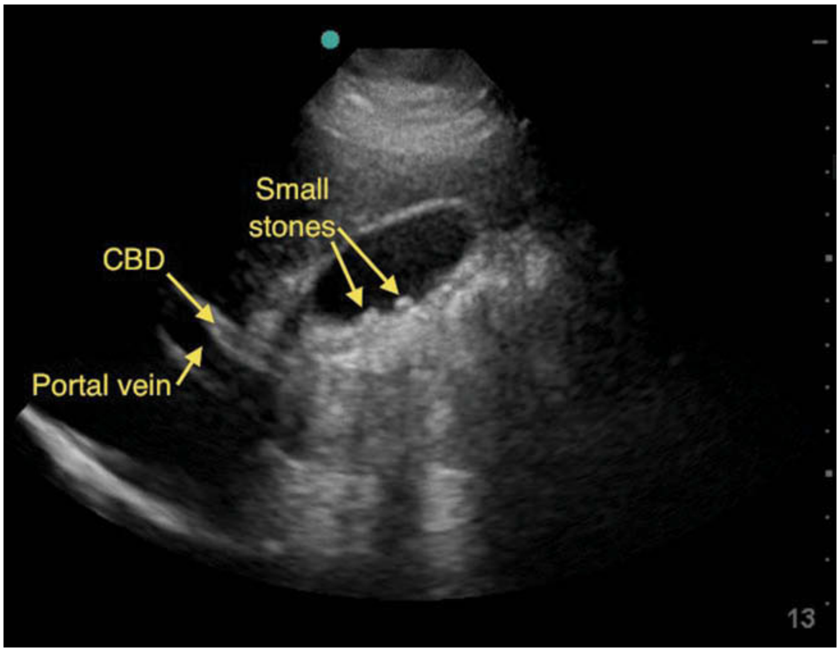

Small
stones
CBD
Portal vein
13

Figure E8.7
Cholelithiases. Multiple small stones in the gallbladder. Note the shadows they cast. In this example, the common bile duct and anterior gallbladder wall are normal.

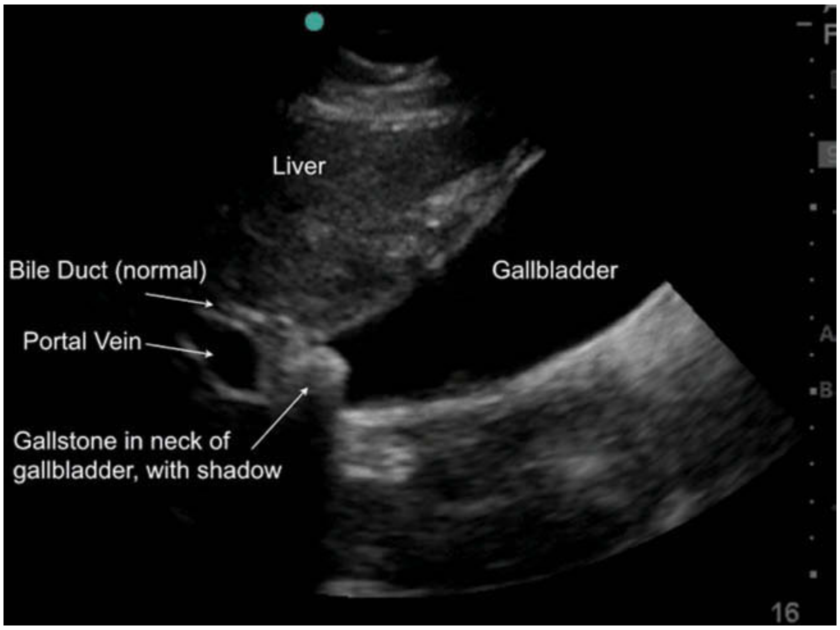

Liver
Bile Duct (normal)
Gallbladder
Portal Vein
Gallstone in neck of
gallbladder, with shadow
16

Figure E8.8
Cholelithiasis. Large gallstone in the neck of the gallbladder, without sonographic evidence of cholecystitis.

- Choledocholithiasis
  - Biliary colic plus biliary ductal dilatation.
- Cholecystitis
  - Biliary colic plus a sonographic Murphy's sign, anterior GB wall thickening, and/or pericholecystic fluid (Figure E8.9).
  - Other gallbladder pathology:
    - Sludge
      - ☐ Hyperechoic collection in the dependent area of the GB
      - ☐ Does not always cast a shadow and may be hard to visualize
    - Polyps
      - ☐ Usually immobile with positional changes (unlike gallstones)
      - ☐ Not typically associated with GB wall thickening
      - ☐ May be obscured by sludge
    - Gallbladder cancer
      - ☐ Suspect if GB is filled with solid material or there are focal masses and a thickened GB wall

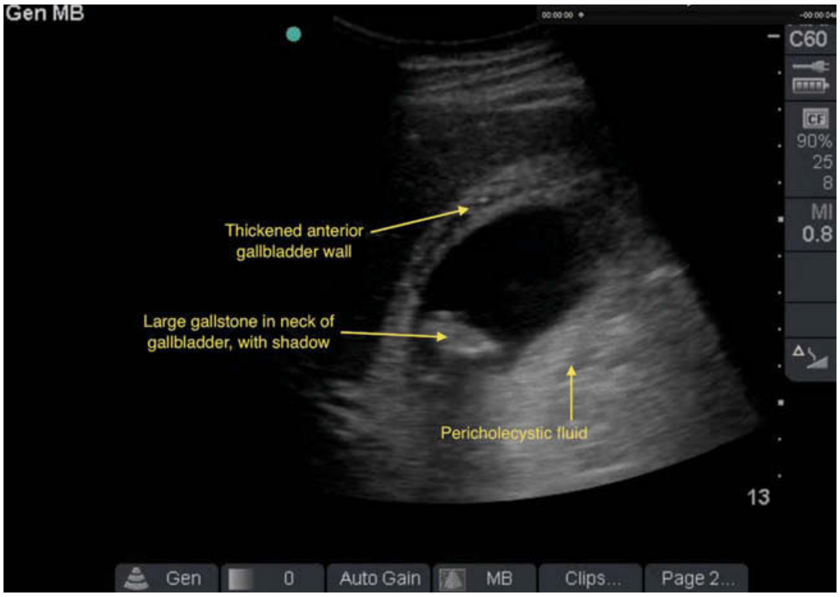

Gen MB
C60
CF
90%
25
8
MI
0.8
Thickened anterior gallbladder wall
Large gallstone in neck of gallbladder, with shadow
Pericholecystic fluid
13
Gen
0
Auto Gain
MB
Clips...
Page 2...

Figure E8.9
Acute cholecystitis. Note the presence of a very thickened gallbladder wall, large gallstone and pericholecystic fluid.

Pearls, pitfalls and myths

- When the GB is contracted, it may be difficult to visualize. Keep the patient NPO (and hydrate with IV fluid if necessary) and reassess in a few hours.
- When the GB is difficult to visualize in the subcostal region, several techniques may be employed:
  - Roll the patient on his/her left side
  - Have the patient to take a deep breath
  - Visualize between the lower ribs
  - Scan from a more lateral position
- A GB full of stones may be mistaken for a loop of bowel. If the GB is difficult to see, search for the wall-echo-shadow sign ("WES" sign), indicating the anterior GB wall with stones in close apposition and shadows behind.
- Cholecystitis may be seen in the absence of gallstones (acalculous cholecystitis).
- Gallstones are frequently asymptomatic; US images should be interpreted in the context of the clinical presentation.

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Appendix F Interpretation of emergency laboratories

Corey R. Heitz, MD

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The ability to diagnose and manage complex clinical scenarios seen in the daily practice of emergency medicine is dependent on correct and efficient laboratory ordering and interpretation. Emergency physicians are often faced with many patient presentations and numerous choices of laboratory tests to aid in diagnosis and treatment. Understanding the strengths and limitations of each individual test is crucial to apply the test in its correct clinical context. The emergency physician must make rational decisions about each laboratory test ordered and base each test ordered on the patient's clinical presentation. Using laboratory data in a "shotgun" mentality, hoping to uncover a diagnosis, often leads to inappropriate testing, unnecessary procedures, delays in care, and potential harm. This appendix focuses on interpretation of laboratory data that are routinely used in the emergency department (ED). It should be noted that normal reference values provided might vary among individual institutions, between genders, and with age. It is recommended to refer to your institution's reference values.

Point-of-care testing

Definition

Point-of-care testing (POCT) is a term used for rapid, bedside testing of various laboratory values. POCT can help narrow a differential diagnosis in a critically ill patient, reduce time to diagnosis of myocardial infarction, and expedite the evaluation and treatment of pulmonary embolism and congestive heart failure. POCT may help meet the ever-present goal of increasing ED efficiency and throughput in the setting of increased ED volumes and time pressures.

Indications

1. *Cardiac evaluation:* POCT for cardiac markers shows variable effects on ED care. Time to diagnosis and treatment is shorter, but it is unclear whether ED length of stay (LOS) is decreased.

2. *B-type natriuretic peptide:* Rapid bedside testing is available for B-type natriuretic peptide (BNP) measurement; interpretation of results is identical to central laboratory testing. Rapid testing may decrease time to diagnosis, treatment and disposition.
3. *Lactate:* POCT for lactate concentration performs similarly to central laboratory testing and decreases the time to identify elevated lactate. This in turn may decrease treatment times in septic patients.
4. *D-dimer:* Bedside assays for D-dimer show similar sensitivities and specificities to the VIDAS laboratory assay. There is a role for D-dimer POCT in the evaluation of venous thromboembolic disease.

Pearls, pitfalls and myths

1. Bedside testing is not available in all facilities at this time.
2. Sensitivities of POCT are similar to that for laboratory analyses, making them good options for use.
3. Cost effectiveness of POCT testing is unclear; although LOS and time to treatment may be improved, there is an associated expense.

Blood and blood components

Complete blood count with differential

Complete blood count (CBC) includes white blood cell (WBC) count, differential, hemoglobin (Hgb), hematocrit (HCT), platelet count and red blood cell (RBC) indices.

Indications

1. Acute blood loss
2. Evaluation of anemia
3. Evaluation of thrombocytopenia
4. Evaluation of serious infection
5. Clinically suspected blood dyscrasia (i.e., leukemia or myelodysplastic syndrome)
6. Determination of neutropenia in an immunocompromised host

Normal values

WBC	4.3–10.8 × 10^3 mm^3
Differential	
Neutrophils (polys)	45–74%
Bands	0–4%
Lymphocytes	16–45%
Monocytes	4–10%
Eosinophils	0–7%
Basophils	0–2%
Hemoglobin	
Males	13–18 g/dL
Females	12–16 g/dL
Hematocrit	
Males	42–52%
Females	37–48%
Platelets	130–450 × $10^3/\mu L$

Absolute neutrophil count (ANC) is calculated using the formula:

$$\text{ANC} = \text{WBC} \times \left(\frac{\text{polys} + \text{bands}}{100}\right)$$

If ANC <1,800 cells/mm^3, then neutropenia is present.

Absolute lymphocyte count (ALC) is calculated using the formula:

$$\text{ALC} = \text{WBC} \times (\text{lymphocyte percentage})$$

The ALC can be used as a surrogate marker for the CD4 count. Those patients with an ALC <1,000 cells/mm^3 are at higher risk for opportunistic infections.

Abnormalities and causes

1. *Elevated WBC*: May be elevated in an acute infection, as well as stress, steroid use and inflammatory states. Other causes include prolonged crying in infants, pain, vomiting, dysrhythmias, pregnancy, neoplasm, exercise, acute myocardial infarction (AMI), surgery and seizures. A "left shift" indicates the presence of immature forms (bands) in the peripheral circulation; this usually represents an infectious state.
2. *Decreased WBC:* May be decreased with infection, septicemia, viral illness and immunocompromised states. Neutropenia, defined as ANC <1,800 cells/mm^3, places the patient at risk of infections from common and opportunistic organisms.
3. *Decreased HCT*: May be secondary to acute blood loss, hemolysis or long-standing anemia. If suspecting acute loss, look for schistocytes on the peripheral blood smear. Pregnancy can also lower the HCT by 10% due to increased plasma volume. Long-standing anemia can be evaluated by the RBC indices. Expect the HCT to drop from administration of fluids in hypovolemic shock or trauma resuscitation.
4. *Increased HCT*: An elevated HCT can be seen in hemoconcentrated states such as dehydration, high altitude, exercise, polycythemia vera or chronic obstructive lung disease.
5. *Increased platelet count*: Thrombocytosis occurs when platelet counts are in excess of 1 million. Platelets are usually large and non-functioning; this condition is seen in myeloproliferative disorders or secondary to iron deficiency anemia, splenectomized states, chronic inflammatory disorders, or hemolytic anemia. Milder platelet elevations may be seen in infectious or systemic inflammatory states.
6. *Decreased platelet count*: Thrombocytopenia occurs when platelet counts are less than the normal range. It may be caused by bone marrow injury from drugs or chemicals, radiation, or infection. It can also be seen in bone marrow failure due to carcinoma, leukemia, lymphoma, or fibrosis. Other causes include menses or poor nutritional states, such as iron, folate and vitamin B12 deficiencies. Diseases such as idiopathic thrombocytopenia (ITP) result in chronically low platelet counts.

Pearls, pitfalls and myths

1. Some facilities have separate orders for CBC with differential and CBC (no differential).
2. Patients with serious infections may have normal or low WBC counts. Overreliance on normal WBC counts in the setting of acute infection may lead to missed diagnosis and delays in patient care.
3. Toxic granulations, Döhle bodies and cytoplasmic vacuolization are remnants of phagocytosis found in neutrophils. These are indicative of more serious bacterial infections.
4. Acute hemorrhage may not be reflected in the initial Hgb or HCT, as the plasma volume must equilibrate for changes to be seen.
5. Geriatric patients will often demonstrate normal to low WBC counts in sepsis.

PT, PTT, INR

Definitions

1. *Prothrombin time (PT):* Measure of the extrinsic (factor VII) and common (factor II, V, X) pathways, as well as warfarin monitoring.
2. *Activated partial thromboplastin time (aPTT)*: Measure of the intrinsic (factors XII, XI, IX, VIII) and common (factors II, V, X) pathways.
3. *International normalized ratio (INR):* A PT ratio derived from the patient's and a control's measured PT, thereby facilitating accurate comparison among different laboratories.

Normal values

PT	9–15 seconds
PTT	22–33 seconds
INR	Warfarin effective between 2 and 3 with low risk of bleeding (normal <1.5)

Indications for obtaining PT

1. Warfarin (Coumadin) therapy
2. Suspected coagulopathy (disseminated intravascular coagulation [DIC], hemophilia)
3. Active bleeding without obvious source
4. Clinical evidence of liver disease
5. History of abnormal, excessive, or spontaneous bleeding
6. History of coagulopathy
7. Before surgery or major vascular procedure if liver disease, malnutrition, or malabsorption exists or clinical history is not available
8. Known or suspected warfarin overdose or excessive ingestion
9. Baseline in acetaminophen overdose

Indications for obtaining PTT

1. IV heparin therapy
2. Suspected coagulopathy (DIC, hemophilia)
3. Active bleeding with or without obvious cause
4. Clinical evidence of liver disease
5. History of abnormal, excessive, or spontaneous bleeding
6. History of coagulopathy
7. Before surgery or major vascular procedure if liver disease, malnutrition, or malabsorption exists or clinical history is not available

Abnormalities and causes

1. *Elevated (prolonged) PT*: Warfarin therapy, liver disease, vitamin K deficiency, antiphospholipid antibodies, congenital factor VII deficiencies, or certain antibiotics.
2. *Elevated (prolonged) PTT*: Heparin therapy, thrombolytic agents, drugs interfering with vitamin K, hemophilia A and B, DIC, moderate to severe von Willebrand's disease, liver failure, antiphospholipid antibodies, or incompletely filled lab tubes (some tubes contain heparin).
3. *Decreased PTT*: Pregnancy, hemolysis, exercise, or anemia.

Pearls, pitfalls and myths

1. Most common causes of prolonged PT and PTT include anticoagulant therapy, vitamin K deficiency (nutritional or secondary to broad-spectrum antibiotics and depletion of bowel flora), liver disease and DIC.
2. Bleeding from excessive warfarin can be managed with 4–5 units of fresh frozen plasma. Vitamin K (5–10 mg) given orally, subcutaneously (SQ) or intravenously (IV) partially reverses the effect of warfarin. Guidelines for the management of an elevated INR are described in Table 15.6.
3. Falsely elevated PTT values are seen if the plasma is excessively turbid or icteric.
4. A PT is *not* indicated before routine hospital admission or routine preoperative testing, in minor trauma, prior to initiation of heparin therapy if other comorbid problems are ruled out, before low-dose subcutaneous heparin therapy, or in patients with a history of alcohol abuse without clinical evidence of liver disease or coagulopathy.
5. A PTT is *not* indicated for patients taking warfarin, in routine hospital admission or preoperative testing, in minor trauma, before initiation of heparin or low-dose subcutaneous heparin therapy, or in patients with a history of alcohol abuse without clinical evidence of liver disease or coagulopathy.

Rh factor

Definition

Rh is an antigen located on cell membranes. Testing for Rh is used to measure the ability of a woman to mount antibodies to antigens that may be present on fetal RBCs (if the father is Rh-positive).

Abnormalities and causes

1. If a pregnant woman is Rh-negative, she can become sensitized to antigens and produce antibodies to fetal RBCs (if the fetus is Rh-positive), causing severe fetal anemia, congestive heart failure (CHF) and *hydrops fetalis*. In order to produce such an antibody response, blood must cross from the fetus to the mother. This usually occurs during delivery, spontaneous miscarriages, abruption, or previous transfusions (affecting subsequent pregnancies).
2. Any pregnant woman with threatened miscarriage or vaginal bleeding should have an Rh factor determination. Rh-negative women should be treated with an injection of anti-D immune globulin (RhoGAM).
3. RhoGAM can be given in both a mini-dose (50 mcg) or full-dose (300 mcg). Indications for the mini-dose include ectopic pregnancy less than 12 weeks gestation, threatened miscarriage less than 12 weeks gestation, or complete miscarriage less than 12 weeks gestation. Full dosing should be given for ectopic, threatened or complete miscarriage greater than 12 weeks gestation, amniocentesis, or fetomaternal transfusion from trauma.

Type and screen

Definition

A type and screen identifies different antigens present on a patient's RBCs.

Indications

1. Anticipation of potential need for transfusion
2. Determination of blood type for RhoGAM administration

Type and crossmatch

Definition

A type and crossmatch is the process by which the laboratory identifies blood for transfusion to a specific patient. In doing so, the lab commits the number of units ordered for that patient.

Indications

1. Hemorrhagic shock
2. HCT less than 21% without active bleeding or less than 30% with active bleeding
3. Obvious upper or lower gastrointestinal bleeding
4. Major vascular surgery, abruptio placenta or placenta previa
5. Coagulation disorders

Fluid chemistry and electrolytes

Serum chemistries

Serum chemistries, also known as serum electrolytes, include sodium, potassium, chloride, bicarbonate, blood urea nitrogen (BUN), creatinine and glucose.

Indications

1. Evaluation of life-threatening hyper- or hypokalemic states
2. Evaluation of mental status changes, coma, or new-onset seizure
3. Evaluation of renal function, chronic hypertension, or diuretic use
4. Evaluation of hyper- or hypoglycemia in the diabetic patient
5. Assessment of acid–base status by anion gap calculation and measurement
6. Evaluation of hydration and volume status, if poor oral intake, repeated vomiting, significant diarrhea, muscle weakness, or alcohol abuse
7. Evaluation of hyperosmolar states
8. Evaluation of upper gastrointestinal bleeding

Normal values

Sodium	135–145 mEq/L
Potassium	3.5–5.0 mEq/L
Chloride	95–110 mEq/L
Bicarbonate	22–28 mEq/L
BUN	8–22 mg/dL
Creatinine	0.7–1.5 mg/dL
Glucose	65–115 mg/dL
Anion gap	10–15

Abnormalities and causes

1. *Elevated sodium*: Hypernatremia is seen in hyperosmolar states, severe dehydration and diabetes insipidus.
2. *Decreased sodium*: Hyponatremia is the most common electrolyte abnormality seen in hospitalized patients. It is further classified as *hypotonic* (etiologies include diuretics, gastrointestinal losses, adrenal insufficiency, CHF and cirrhosis), *isotonic* (etiologies include syndrome of inappropriate antidiuretic hormone [SIADH] secretion, psychogenic polydipsia), or *hypertonic* (hyperglycemia, volume depletion) based on plasma osmolality. Hyponatremia may also be factitious due to hyperglycemia, hyperlipidemia and hyperproteinemia.
3. *Elevated potassium*: Hyperkalemia is often the result of specimen hemolysis. True causes of hyperkalemia include acidosis, tissue damage, acute renal failure,

drug-induced causes (potassium-sparing diuretics, nonsteroidal antiinflammatory drugs [NSAIDs], digoxin, angiotensin-converting enzyme [ACE] inhibitors), and oral and IV administration of potassium. *Pseudohyperkalemia* is a laboratory artifact in which the serum potassium is elevated when the plasma potassium is normal, as occurs with hemolysis, thrombocytosis, leukocytosis and many myeloproliferative disorders.

4. *Decreased potassium*: Hypokalemia results from vomiting, diarrhea, diuretics, acute hyperventilation, insulin administration, or diabetic ketoacidosis (DKA).
5. *Increased chloride*: Results from metabolic acidosis, gastrointestinal bicarbonate loss, respiratory alkalosis, renal acidosis, or hyperparathyroidism.
6. *Decreased chloride*: Vomiting, gastric drainage, or diuretics.
7. *Increased bicarbonate*: Respiratory acidosis or metabolic alkalosis (Cushing's syndrome, vomiting, volume depletion).
8. *Decreased bicarbonate*: Respiratory alkalosis or metabolic acidosis (ketoacidosis, lactic acidosis, diarrhea, renal tubular acidosis, renal failure). Bicarbonate decreases by an average of 15% during pregnancy. May also lower by 5–8 mEq/L due to hyperventilation.
9. *Increased BUN*: Dehydration, high protein intake, exercise, renal failure, upper gastrointestinal bleeding, or CHF.
10. *Decreased BUN*: Low protein intake, high water intake, cirrhosis, or pregnancy.
11. *Increased creatinine*: Renal insufficiency, renal failure, rhabdomyolysis, dehydration, or strenuous exercise.
12. *Decreased creatinine*: Pregnancy, malnutrition, or water intoxication.
13. *Hyperglycemia*: Diabetes mellitus, hyperthyroidism, Cushing's disease, pheochromocytoma, acute illness, glucocorticoid use, lithium, or thiazides.
14. *Hypoglycemia*: Drugs, especially insulin, sulfonylureas, ethanol, insulinoma, sepsis, acute illness, starvation, adrenal insufficiency, growth hormone deficiency, renal failure, hypopituitarism, postprandial, or after gastric surgery.

Pearls, pitfalls and myths

1. Hyperglycemia causes pseudohyponatremia:

$$\text{Corrected sodium} = \text{sodium} + (\text{glucose} - 5)/3.5$$

General rule of thumb: for every 100 mg/dL increase in plasma glucose concentration, the plasma sodium concentration decreases by approximately 1.6 mEq/L.

2. Most often hypo- or hypernatremia can be corrected by treating the underlying condition or by administration of normal saline; 3% saline is rarely needed and must be given very slowly to avoid *central pontine myelinosis*. Be sure to monitor urine output and check electrolytes frequently. Goal should be to correct the sodium gradually (maximum rate of 0.5 mEq/L/hour).
3. Serum potassium <3 mEq/L indicates a total body deficit of 300–400 mEq of total body potassium. Each 10 mEq of potassium replaced will raise the serum potassium by roughly 0.1 mEq/L.
4. Electrocardiogram (ECG) manifestations of hyperkalemia include peaked T waves, followed by loss of the P wave, widening of the QRS complex, and sine wave-appearing tachycardia. Patients may complain of muscle cramps, weakness, paralysis, paresthesias, or tetany. Treatment includes close cardiovascular monitoring, immediate antagonism of potassium at the cardiac membrane with calcium chloride or calcium gluconate, lowering the serum potassium and correcting the underlying cause. Serum potassium can be lowered by administration of sodium bicarbonate, glucose and insulin, and nebulized beta-agonists. Definitive treatment to remove excess potassium from the body includes administration of oral/rectal exchange resins (i.e., polystyrene sulfonate) and hemodialysis.
5. Serum osmolality is calculated by the following equation:

$$\text{Osmolality} = 2 \times \text{Na} + \text{glucose}/18 + \text{BUN}/3.8 + \text{EtOH}/3.7$$

An osmolal gap (the difference between measured serum osmolality and calculated serum osmolarity) indicates the presence of millimolar amounts of an uncharged particle. If the osmolal gap is elevated, the patient's serum ethanol level should be measured. If the gap is greater than 10 once ethanol has been accounted for, then unaccounted (and unmeasured) osmols may represent methanol, ethylene glycol, isopropyl alcohol, acetone, acetylsalicylic acid, or paraldehyde.

6. The anion gap is calculated by the following formula:

$$\text{Anion gap} = \text{Na} - (\text{Cl} + \text{bicarbonate})$$

An elevated anion gap usually indicates the presence of a metabolic acidosis. "MUDPILECATS" is a helpful mnemonic for an elevated anion gap metabolic acidosis (Table 41.6).

7. Patients with severe acidosis tend to have elevated potassium levels, as acidemia shifts potassium from the intracellular to extracellular space. Even when a patient's total body potassium is severely depleted, the serum potassium may be high in acidotic states (e.g., DKA). As the patient is treated, serum potassium levels may fall precipitously if not closely monitored and corrected. Remember to correct the

patient's potassium only after ensuring adequate urine output.

8. Use clinical judgment before ordering serum chemistries. Avoid using chemistry panels as screening tools.
9. Determining blood glucose is critical in the evaluation of any patient with mental status changes, seizure, stroke-like symptoms, coma, drug ingestion, or acute illness. If hypoglycemia is suspected, treat with 1 ampule of D50 (50% dextrose) prior to obtaining formal laboratory results. Bedside glucose determination using glucometers is recommended as long as it does not cause significant delay in administering glucose.
10. Bedside glucometers are often inaccurate in patients with a low pO_2 or HCT, and are affected by an inadequate amount of blood, improper storage reagents and machine calibration.

Calcium

Indications

1. Altered mental status
2. Recent thyroid and parathyroid surgery, neck trauma, or neck surgery
3. Multiple myeloma, bone metastases
4. New-onset renal failure, evaluation of weakness in a hemodialysis patient
5. Acute pancreatitis
6. Evaluation of tetany

Normal values

Calcium, free (ionized)	1.1–1.4 mmol/L
Calcium, total	8.9–10.5 mg/dL

Abnormalities and causes

1. *Hypercalcemia*: Primary hyperparathyroidism, malignancies producing parathyroid-like protein, metastatic bone disease, sarcoidosis, acidosis and excessive ingestion of calcium-containing antacids.
2. *Hypocalcemia*: Acute pancreatitis, rhabdomyolysis, sepsis, malignancy, hepatic or renal insufficiency, tuberculosis, parathyroid adenoma resection, vascular or parathyroid injury during surgery or trauma, and malabsorptive states not allowing uptake of calcium from the small intestine (e.g., pancreatectomy, small bowel resection).

Pearls, pitfalls and myths

1. Hypocalcemia primarily causes neuromuscular (tetany, seizures, muscle cramps, weakness) and cardiovascular effects (prolonged QT interval, dysrhythmias, cardiovascular collapse, refractory hypotension).
2. Calcium levels are most accurate when corrected for abnormal albumin:

$$\text{Corrected calcium (mg/dL)} = \text{serum calcium (mg/dL)} + 0.8 \times [4 - \text{serum albumin (g/dL)}]$$

3. Classic peripheral neurologic findings of hypocalcemia include:
   Chvostek sign: Tap over the facial nerve 2 cm anterior to the tragus of the ear. Twitching of the mouth, nose, eye and facial muscles will occur.
   Trousseau sign: Inflation of a blood pressure cuff above the systolic pressure causes ulnar and median nerve ischemia resulting in carpal spasm. This is usually observed during vital sign measurement at triage.

Magnesium

Indications

1. Evaluation of ventricular dysrhythmias
2. Neuromuscular weakness
3. Poor nutrition, alcoholism, pancreatitis, malabsorptive syndromes

Normal values

Magnesium	1.4–2.5 mg/dL

Abnormalities and causes

1. *Elevated magnesium*: Dehydration, hemoconcentration, hypoglycemia, hemolyzed specimen, cell lysis syndromes, hemolytic anemia, renal failure, or DKA.
2. *Decreased magnesium*: Malabsorption, malnutrition, renal tubular injury, hypoparathyroidism, ethanol use, digoxin use, cyclosporines, or cisplatin use.

Pearls, pitfalls and myths

1. In addition to laboratory testing, magnesium infusion is first-line treatment in torsades de pointes, eclamptic seizures and severe asthma exacerbation (impending respiratory failure). Magnesium has also shown promise as a treatment for migraines. Magnesium should be infused slowly to avoid complications such as a loss of reflexes, muscle weakness, hypotension, vasodilation, or respiratory failure.

Abdominal evaluations

Liver function tests

Liver function tests (LFTs) include aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transpeptidase (GGT), alkaline phosphate (ALK), bilirubin and lactate dehydrogenase (LDH). Of note, AST was formerly known as SGOT, and ALT as SGPT.

Indications

1. Evaluation of the synthetic, excretory and metabolic function of the liver and biliary tract.
2. Evaluation of liver damage from toxic substances, drugs, autoimmune disorders, infectious processes, eclampsia.

Normal values

AST	8–40 IU/L
ALT	Males 8–45 IU/L Females 6–38 IU/L
GGT	Males 11–49 IU/L Females 7–32 IU/L
ALK	30–130 IU/L
Bilirubin	Total 0.3–1.1 mg/dL Direct 0–0.2 mg/dL
LDH	100–212 IU/L

Abnormalities and causes

1. *Elevated AST*: Liver injury (acute and chronic hepatitis), biliary tract obstruction, acute MI (seldom used due to its lack of specificity), skeletal muscle diseases, hemolytic anemia, drug-induced liver or muscle injury.
2. *Decreased AST or ALT*: Pregnancy or chronic renal failure.
3. *Elevated ALT*: Hepatocellular injury, large myocardial damage, skeletal muscle disease, biliary tract obstruction, or drugs inducing liver or muscle injury.
4. *Elevated GGT*: Obstructive jaundice, hepatitis, cirrhosis, metastatic liver disease, pancreatitis, prostate cancer, hyperthyroidism, or diabetes. Used by some as a general screen for liver disease. GGT is elevated in approximately 70% of patients with alcoholic liver disease.
5. *Decreased GGT*: Exercise, pregnancy, or hypothyroidism.
6. *Elevated ALK*: Biliary tract obstruction, metastatic liver lesions, primary biliary cirrhosis, drug-induced hepatitis, metastatic bone cancer, conditions that lead to increased bone turnover (e.g., osteomalacia, Paget's disease, osteosarcoma), or hyperthyroidism. Mainly used to detect and monitor liver or bone disease.
7. *Decreased ALK*: Pregnancy or blood transfusions.
8. *Elevated bilirubin*: Liver or biliary tract diseases, Dubin-Johnson syndrome, drug toxicity, or neonatal hyperbilirubinemia (may be normal).
9. *Decreased bilirubin*: Improper lab storage.
10. *Elevated LDH*: Hemolytic anemia, malignancies, acute hepatitis, MI, shock, strangulated bowel obstruction, amiodarone (chronic use), or hemolyzed specimens. This is a nonspecific marker of cell injury, seen with most types of cell damage.

Pearls, pitfalls and myths

1. With most types of acute hepatitis, ALT is elevated to a higher degree than AST. Very high values occur with ischemic and toxic hepatitis. With muscle injury, AST is usually 3–5 times higher than ALT.
2. With alcoholic hepatitis, the AST:ALT ratio is about 2:1 because alcohol damages the mitochondria, a source of AST. With viral hepatitis, the ALT is usually greater than the AST because toxicity is more liver-specific.
3. During the course of common bile duct obstruction, the AST and ALT will be the first to rise, followed by the ALK and bilirubin.
4. The indirect bilirubin (unconjugated) can be calculated by subtracting the direct bilirubin (conjugated) from the measured total bilirubin.

Lipase

Indications

Used to diagnose pancreatitis. Lipase is the most useful laboratory test in the evaluation of suspected pancreatitis.

Normal values

Lipase	4–50 IU/dL

Abnormalities and causes

1. *Elevated lipase*: Pancreatitis, pancreatic pseudocyst, renal failure, heparin, or drugs causing pancreatic injury.
2. *Decreased lipase*: Hepatitis, falsely lowered in obstructive jaundice, pancreatic insufficiency.

Pearls, pitfalls and myths

1. Serum lipase is the most sensitive test for evaluating pancreatic injury; it typically remains elevated up to 7 days following injury. Elevations >3 times normal markedly increase the likelihood of pancreatitis. Though lipase is frequently used as a diagnostic marker for pancreatitis (without amylase), some reports question its specificity, as elevations may be seen in other disorders.

Amylase

Indications

Often used in the diagnosis of pancreatitis, although has less utility than serum lipase.

Normal values

Amylase	60–110 IU/L

Abnormalities and causes

1. *Elevated amylase*: Acute pancreatitis, pancreatic pseudocyst, salivary gland inflammation, intestinal ischemia, DKA, choledocholithiasis, renal failure, cirrhosis, or ovarian cysts. It may also be elevated

in ectopic pregnancy; however, it is not used as a screening test due to its lack of specificity.

2. *Decreased amylase*: Chronic pancreatitis, hypertriglyceridemia, pancreatic insufficiency, or malnutrition.

Pearls, pitfalls and myths

1. May remain elevated in acute pancreatitis 36–48 hours after onset. Less specific than lipase in the diagnosis of pancreatitis, so utility is limited as it does not rule in or rule out pancreatitis.
2. May be normal in patients with chronic pancreatitis ("burnt-out" pancreas).

Cardiovascular

Cardiac markers

Cardiac markers include creatine kinase (CK), CK isoenzymes MB (CK-MB), troponins T and I, and myoglobin.

Indications

1. Evaluation of patients with suspected acute coronary syndrome (ACS). Myoglobin, present in all muscle tissue, is generally elevated within 2 hours of symptom onset. It is limited by its lack of specificity. Current recommendations suggest confirming a positive myoglobin with a more specific test. Work-up including ECG, CK-MB and troponin shows promise in improving early diagnosis of AMI, as do strategies using serial biomarkers in short succession, evaluating for small changes over time. Troponins have the advantage in that they remain elevated longer than other cardiac markers, allowing for diagnosis after delayed presentation.

Normal values

CK	
Females	45–135 IU/L
Males	55–170 IU/L
CK-MB	<10 ng/mL (mass)
Troponin-I	<1.6 ng/mL
Troponin-T	<0.1 ng/mL
Myoglobin	<90 ng/mL

Abnormalities and causes

1. *Elevated CK*: CK is a muscle enzyme found in skeletal muscle, cardiac muscle and brain tissue. Elevations may be seen with strenuous exercise, muscle damage, acute MI, hypothyroidism, malignant hyperthermia, neuroleptic malignant syndrome, or intramuscular injection.
2. *Decreased CK*: Pregnancy, aging, malnutrition, or hyperthyroidism.
3. *Elevated CK-MB*: Myocardial infarction, myocarditis, cardiac contusion, or skeletal muscle injury (total CK-MB but not relative index).
4. *Troponin*: Troponins are small enzymes found in muscle tissue. Two of these enzymes (troponin-I and troponin-T) are found only in cardiac tissue. Troponin-I and Troponin-T may be elevated in myocardial infarction, unstable angina and renal failure. Highly sensitive assays for troponin-I markedly improve diagnostic performance, and are used exclusively as markers of myocardial infarction at some facilities. The highly sensitive assays have lower reference values than other troponins, and can be positive earlier after onset of injury.
5. *Elevated myoglobin*: Skeletal muscle injury, myocardial infarction, renal failure, seizures, or exercise.

Pearls, pitfalls and myths

1. Myoglobin rises earliest with muscle injury (within 2–4 hours), peaks in 8–12 hours, and returns to normal within 24–40 hours. It lacks cardiospecificity since it is abundant in all muscle tissue.
2. CK-MB is first detected by 3–4 hours, peaks by 12 hours, and returns to normal within 24–36 hours. With myocardial damage, CK-MB is greater than 10% of the total CK released. Values between 3% and 10% are indeterminate. A relative index is obtained by dividing CK-MB by total CK and multiplying by 100. A ratio <3 is indicative of skeletal muscle; a ratio >5 indicates cardiac muscle injury. CK-MB is less specific, and therefore less useful, than troponin.
3. Troponin rises 3–4 hours after injury and remains elevated for up to 10 days. A positive troponin in the setting of unstable angina or non–ST-segment elevation myocardial infarction is associated with a four times greater risk of death than patients without a positive troponin. An increase in troponin of ≥0.04 over 4 hours, even if the value is not above the threshold for positivity, is suggestive of an ischemic event. Troponin is the most specific of the cardiac markers. Different troponin lab assays may have slightly different values for normal and abnormal results.
4. Normal cardiac markers within the first few hours after symptom onset do not rule out ACS and should not be used as a basis for discharging a patient from the ED. Some protocols suggest that rapid serial measurements in the pain-free patient can rule out AMI; these patients are still at risk for ACS and need further evaluation in the appropriate clinical setting.

D-dimer

Definition

Plasma D-dimer is a breakdown product of fibrin clots, and is elevated in numerous disorders where fibrin deposition and breakdown occurs, such as venous thromboembolism (VTE), systemic inflammatory states, and some chronic disease states. D-dimer can be detected by an enzyme-linked immunosorbent assay (ELISA) or latex agglutination. Multiple commercial assays are available, so it is necessary to know the assay used at your institution and the cutoff values.

Indications

1. Evaluation of suspected venous thromboembolic disease
2. Detecting fragments of cross-linked fibrin, mainly in DIC
3. Controversy exists between various methods, but the ELISA appears to have the best mix of sensitivity and specificity

Normal values

D-dimer	<500 FEU; <1.6 mg/mL

Abnormalities and causes

1. *Elevated D-dimer:* Venous thromboembolic disease, deep vein thrombosis (DVT), pulmonary embolism (PE), malignancy, DIC, pregnancy, or chronic inflammatory conditions.

Pearls, pitfalls and myths

1. A patient with a negative D-dimer and a low pretest probability for PE is unlikely to have a significant PE.
2. A patient with high pretest probability is not sufficiently evaluated with a D-dimer, and needs imaging studies to rule out pulmonary embolism.
3. The D-dimer is a nonspecific test, and therefore may be elevated in many disease states. It cannot be used to rule in thromboembolic disease, but may dictate further evaluation.
4. Clinical decision rules may be used to rule out PE without the use of D-dimer testing.

B-type natriuretic peptide

Indications

1. B-type natriuretic peptide (BNP) is both a diagnostic and prognostic marker in undifferentiated dyspnea and CHF. It is predominantly secreted from the ventricular myocardium during myocardial pressure and stretching. NT-proBNP is the inactive amino terminal cleaved from the precursor molecule, and has shown promise as another marker for the evaluation of acute dyspnea.

Normal values

Normal BNP	<100 pg/mL
Intermediate	100–400 pg/mL
Elevated	400 pg/mL

Abnormalities and causes

1. A BNP level <100 pg/mL is approximately 85% accurate in excluding CHF as the cause of dyspnea, and values >400 pg/mL are highly suggestive of heart failure. Increased levels need to be interpreted in the context of a patient's clinical status, as not every patient with an increased BNP level has decompensated CHF.

Pearls, pitfalls and myths

1. The diagnosis of CHF can often be made on clinical and radiographic grounds, and a BNP level does not necessarily assist with diagnostic accuracy.
2. Intermediate levels may prove helpful for prognosis if a patient's baseline BNP level is known.
3. Patients with intermediate levels may still be diagnosed with CHF based on clinical factors.

Blood gases and acid–base

Arterial and venous gases

Definition

Arterial and venous blood gas (ABG and VBG) results include blood pH, pCO_2, pO_2 and bicarbonate (HCO_3^-).

Indications

1. Evaluation of respiratory and metabolic acid–base disturbances including poisonings, respiratory failure, diabetic and alcoholic ketoacidosis, or severe chronic obstructive pulmonary disease (COPD) exacerbations
2. Undifferentiated shock
3. Unexplained coma or confusion/obtundation

Normal values

Blood pH	7.38–7.42
pCO_2	38–42 mmHg
pO_2	83–108 mmHg
HCO_3^-	22–26 mmol/L

Abnormalities and causes

1. *Elevated pH*: Vomiting, volume contraction, hyperaldosteronism, early CHF, drugs causing alkalosis, or anxiety.
2. *Decreased pH*: Ketoacidosis, lactic acidosis, renal failure, respiratory failure, chronic obstructive lung disease, drugs causing acidosis (isoniazid, iron, salicylates), or ethylene glycol; also delayed sample measurement.
3. *Increased pCO_2*: Respiratory failure, hypoventilation, COPD, CNS depression, metabolic alkalosis, or drugs depressing respiration (alcohol, barbiturates, opiates, benzodiazepines); also delayed measurement of sample.

4. *Decreased* pCO_2: Hyperventilation, anxiety, interstitial lung disease, cirrhosis, metabolic acidosis, hyperthyroidism, PE, or aspirin.
5. *Increased* pO_2: Oxygen therapy, excessive air bubbles in specimen, hyperventilation, or aspirin.
6. *Decreased* pO_2: COPD, pneumonia, interstitial lung disease, PE, CHF, shock, CNS depression, right-to-left cardiac shunts, or drugs that depress respiration.
7. *Elevated* HCO_3^- : Respiratory acidosis, metabolic alkalosis (such as from vomiting, Cushing's syndrome, or volume depletion), diuretics, or glucocorticoids.
8. *Decreased* HCO_3^-: Respiratory alkalosis, metabolic acidosis (such as ketoacidosis, lactic acidosis, renal failure, or diarrhea), carbonic anhydrase inhibitors, ethylene glycol, methanol, aspirin, or pregnancy.

Pearls, pitfalls and myths

1. Prior to obtaining the arterial sample, a patient must be evaluated for both radial and ulnar arterial blood supply. The *Allen test* determines the presence of collateral flow from both the radial and ulnar artery in the hand. If abnormal, radial artery cannulation should be avoided to prevent ischemic complications.
2. ABG specimens must be handled appropriately and run expeditiously. Errors arise from excess air bubbles in the sample, excess heparin in the syringe, and delays in placing the sample on ice.
3. If a patient is hypoxemic, attempt to determine the etiology. Do not just treat with supplemental oxygen.
4. The ABG measurement in a patient with carbon monoxide poisoning will help by identifying the presence of metabolic acidosis with a normal pO_2.
5. ABG samples are not necessary if pulse oximetry is sufficient to guide management.
6. VBG specimens are sufficient to evaluate a patient for acidemia, as the pH correlates highly with the value from an ABG. pO_2 values from a VBG will be lower than from an ABG; pCO_2 values have less correlation with those from an ABG.
7. The SaO2 from an ABG may be a calculated value and not measured. The exception to this is when the ABG is performed on a machine that also performs co-oximetry.

Alveolar-arterial oxygen gradient

Definition

The alveolar-arterial (A–a) oxygen gradient can be used to differentiate between hypoxia caused by hypoventilation alone (i.e., neuromuscular diseases, overdoses) in which the A–a gradient is normal, and hypoxia caused by ventilation–perfusion mismatch, right-to-left shunting and diffusion abnormalities (i.e., PE, CHF, acute respiratory distress syndrome [ARDS], COPD) in which the A–a gradient is abnormal. The A–a gradient is calculated using the formula:

$$\text{A–a gradient} = [\mathrm{FiO_2} \times (\text{barometric pressure} - 47\ \text{mmHg})] - \left(\mathrm{pO_2} + \frac{\mathrm{pCO_2}}{0.8}\right)$$

For an ABG drawn on room air at sea level, the formula becomes:

$$\text{A–a gradient} = 150 - \left(\mathrm{pO_2} + \frac{\mathrm{pCO_2}}{0.8}\right)$$

The A–a gradient is increased in smokers and patients with intrinsic lung disease, and increases as people age. A simple formula to take this variation into account is:

$$\text{Normal A–a gradient} < \frac{\text{age}}{4} + 4$$

Pearls, pitfalls and myths

1. The A–a gradient cannot be determined accurately in a patient who is receiving O_2 by nasal cannula, as the FiO_2 of supplemental oxygen must be estimated.
2. Calculating the A–a gradient has clinical implications, in that hypoxia in the presence of a normal gradient is treated by improving ventilation, whereas an increased A–a gradient should be treated with supplemental oxygen and evaluated for treatable causes.
3. The A–a gradient is neither sensitive nor specific for the diagnosis of PE. A normal A–a gradient in a young, otherwise healthy patient does not exclude the diagnosis.

Co-oximetry

Definition

Co-oximetry measures levels of various hemoglobin forms in the blood, and can also be used to measure the SvO_2, which is the venous oxygen saturation. Depending on the laboratory, it can be a separate test or performed along with an ABG.

Indications

1. To evaluate for the presence of abnormal hemoglobin forms (e.g., carboxyhemoglobin, methemoglobin).
2. To evaluate the SvO_2, an indicator of tissue oxygen consumption.

Abnormalities

1. *Carboxyhemoglobin*: Carboxyhemoglobin is formed in the presence of carbon monoxide. Clinical intoxication results in symptoms ranging from dyspnea, mild headache, and malaise to obtundation and coma. Normal levels in a nonsmoker are generally 1–3%, and as high as 15% in smokers. Treatment is generally initiated at levels >25%. Treatment consists of 100% oxygen. Hyperbaric oxygen therapy is indicated for increased levels and worsening clinical status, including pregnancy.

2. *Methemoglobin*: Methemoglobin is formed when hemoglobin is oxidized, causing a reduction of oxygen binding. This can be a result of medication exposure, commonly mucosal absorption of benzocaine derivatives. Normal methemoglobin values are approximately 1.5% or less.
3. *SvO_2:* Venous oxygen saturation indicates tissue metabolic activity. The greater the level of oxygen saturation, the less oxygen utilized during perfusion. During high metabolic states such as sepsis, SvO_2 can be low. Ideal SvO_2 values are greater than 70%.

Lactate

Definition

Lactic acid is produced by cells under times of oxidative stress, often as a result of poor tissue perfusion. Blood lactate levels can be measured in the ED and followed during hospitalization.

Normal Values

Lactate	<2–2.4 mmol/L

Indications

1. Evaluation of tissue perfusion during sepsis and hemorrhagic shock
2. Evaluation of mesenteric perfusion
3. Monitoring of resuscitation status during sepsis and hemorrhagic shock

Pearls, pitfalls and myths

1. Lactate levels >4 mmol/L represent severely decreased tissue perfusion.
2. Lactate levels are independent predictors of mortality, even in the absence of hemodynamic instability.
3. Initial treatment for elevated lactate is volume resuscitation.
4. Lactate levels can be followed as often as every 2 hours in critically ill patients.
5. A lactate can be obtained from whole blood (venous sample) or an ABG at some facilities. If a venous lactate is obtained, a sample from a central line is more accurate than a peripheral sample (due to vasoconstriction or tourniquet use).

Urine studies, pregnancy and sexually transmitted infections

Urinalysis

Definition

A urinalysis includes specific gravity, pH, protein, glucose, ketones, blood, bilirubin, urobilinogen, nitrite and leukocyte esterase. Microscopy includes crystals, epithelial cells, RBCs, WBCs, hyaline casts, granular casts, cellular casts and organisms.

Indications

1. Evaluation of suspected asymptomatic bacteriuria, cystitis, urethritis, prostatitis and pyelonephritis
2. Evaluation of hydration status
3. Evaluation of suspected ureterolithiasis
4. Evaluation for rhabdomyolysis
5. Evaluation of acute renal failure and acute tubular necrosis
6. Evaluation of DKA and ketonuria

Normal values

Urine: Normally urine is sterile and transparent, with a specific gravity of 1.010 and normal pH. Urine is yellow due to urochrome pigment, with the degree of coloration related to urine concentration. Cloudiness indicates the presence of particulate matter such as crystals, RBCs, WBCs, mucus, or bacteria.

Causes of abnormal urine color

Red	Blood, hemoglobin, porphyrins, beets
Brown	Hemoglobin, myoglobin, bilirubin, nitrofurantoin
Orange	Urates, phenazopyridine (Pyridium), sulfasalazine
Dark	Hemoglobin, myoglobin, alkaptonuria

Abnormalities and causes

1. *Specific gravity*: May be variable due to disorders of urine-concentrating ability or changes in fluid status. Alkaline pH may lead to false results.
2. *pH*: Acid–base disorders affect the urine pH. Some urinary tract infections (UTI) may produce alkaline pH.
3. *Protein*: Increased protein is seen in glomerular diseases, infections, tubular disorders and exercise. Urine dipstick analysis is sensitive for albumin. Globulins are less easily detected. Other proteins (such as Bence–Jones proteins) may be missed entirely.
4. *Glucose*: May be elevated in patients with diabetes mellitus. Elevated glucose in the urine suggests a serum glucose >170 mg/dL. Glucosuria can be present in normal individuals after consuming a high-glucose beverage, as well as patients receiving dextrose infusions.
5. *Ketones*: Ketoacidosis will produce ketonuria. Dipstick urinalysis measures acetoacetic acid or acetone but not beta-hydroxybutyrate. Urine ketones have a high negative predictive value for DKA but a lower positive predictive value.
6. *Blood*: Bleeding at any site in the urinary tract will produce hematuria, including glomerulonephritis, tumors, stones, infection, coagulopathy, hemolysis, or myoglobinuria with muscle damage.

7. *Bilirubin*: Most commonly seen in liver and biliary disease. Complete biliary obstruction is suggested when the bilirubin is positive with a negative urobilinogen.
8. *Urobilinogen*: Seen in hemolysis or cirrhosis.
9. *Nitrites*: Positive in most Gram-negative infections; based on an organism's ability to convert urinary nitrates to nitrite.
10. *Leukocyte esterase*: Positive in the setting of most urinary tract infections; may also be positive in abdominal inflammatory disorders, such as appendicitis.
11. *Crystals*: Uric acid and calcium oxalate crystals may be part of a normal urinalysis. Calcium oxalate crystals are occasionally seen with nephrolithiasis.
12. *Epithelial cells*: An adequate urine specimen, either by mid-stream collection or urethral catheterization, should have less than five epithelial cells per high-power field. Squamous or transitional cells are normal. *Clue cells* are vaginal epithelial cells to which bacteria have attached.
13. *RBCs*: Indicative of glomerular injury, nephrolithiasis, inflammation, or neoplasm.
14. *WBCs*: Counts higher than five per high-power field indicate infection of the bladder, kidney, prostate, cervix, or vagina.
15. *Hyaline casts*: Seen in cases of proteinuria (especially diabetes), dehydration, or exercise.
16. *Granular casts*: RBC casts seen in glomerulonephritis; WBC casts seen in pyelonephritis.
17. Organisms: Contaminated specimens, cystitis, pyelonephritis, urethritis, or prostatitis.

Pearls, pitfalls and myths

1. Accuracy of urine testing depends on the care with which specimens are collected and transported. Avoid specimen contamination with periurethral flora during collection. Use of sterile containers and prompt transport to the laboratory may prevent microbial growth.
2. Urine specimens with high numbers of leukocytes or RBCs, the presence of leukocyte esterase or nitrites, and culture colony counts of a single organism >100,000 colony-forming units/mL (CFU/mL) are indicators of infection.
3. The presence of one organism per oil-immersion field of stained uncentrifuged urine is consistent with >100,000 CFU/mL.
4. Urine dipsticks are often used in the ED setting. A positive leukocyte esterase test combined with positive nitrites is predictive of a UTI.
5. *Pyuria* (defined as >5 WBCs per high-power field) does not always indicate infection. *Sterile pyuria* can be found in inflammatory conditions such as appendicitis, pelvic inflammatory disease, or renal tuberculosis.
6. Urine cultures should be reserved for the following settings: children, pregnant women, immunocompromised patients, recently treated patients with clinical relapse, suspected neutropenia, known abnormalities of the urinary tract such as neurogenic bladder, pyelonephritis, diabetic patients, and renal dialysis patients.
7. Myoglobin from muscle breakdown may mimic blood on a dipstick UA; it can be differentiated from true hematuria with a urine myoglobin test or the absence of RBCs on urine microscopy.
8. Microscopic hematuria in the setting of abdominal trauma is not indicative of renal or ureteral injury; gross hematuria needs further evaluation.

Pregnancy tests

Tests of pregnancy include urine pregnancy tests, serum qualitative tests, and serum quantitative human chorionic gonadotropin (HCG) tests.

Indications

1. Identification or exclusion of pregnancy in reproductive-aged females with vaginal bleeding, trauma, abdominal or pelvic pain, or sexual assault
2. Determination of serum HCG for correlation with ultrasound diagnosis
3. Diagnosing ectopic pregnancy, monitoring of trophoblastic tumors and screening for fetal abnormalities
4. Monitoring the quantitative HCG in threatened miscarriages

Normal values

Urine tests vary in the amount of HCG needed to produce a positive test. Assays using antibodies to the beta subunit (β-HCG assays) will detect 25 mU/mL, recognizing pregnancy with 95% sensitivity 1 week after the first missed menstrual cycle.

During pregnancy, plasma HCG levels increase predictably, except after 20 weeks.

Duration of pregnancy (weeks)	Plasma human chorionic gonadotropin (mU/mL)
1	5–50
2	50–500
3	100–10,000
4	1,000–30,000
5	3,500–115,000
6–8	12,000–270,000
8–12	15,000–220,000
20–40	3,000–5,000

Abnormalities and causes

1. *Elevated HCG*: In normal pregnancy, HCG becomes elevated 1–2 weeks after fertilization and doubles approximately every 2 days, reaching its peak at 8–10 weeks. In ectopic pregnancy, HCG levels rise more slowly. With trophoblastic tumors (molar pregnancy),

HCG levels rise slowly at first, but reach levels higher than expected in normal pregnancy. Spontaneous miscarriage and fetal non-viability are usually preceded by placental and fetal tissue death, causing failure of the expected normal rise in serum HCG.

Pearls, pitfalls and myths

1. β-HCG of 2,000 (around 35 days gestation) should have a visible uterine gestational sac on transvaginal ultrasound. β-HCG of 6,000 (around 42 days gestation) should have a visible uterine gestational sac on transabdominal ultrasound.
2. Ectopic pregnancy often occurs at a low HCG level and should be aggressively sought if clinical signs or symptoms are suggestive. HCG levels tend to be higher in normal than abnormal pregnancy.
3. A single quantitative HCG cannot be relied on to make decisions about a particular pregnancy; therefore, serial HCGs and close follow-up should be assured.

Urine testing for STIs

Definition

With the development of nucleic acid amplification techniques (NAATs), it has become possible to diagnose sexually transmitted infections (STIs), such as *Chlamydia trachomatis*, *Neisseria gonorrhea* and *Trichomonas*, from urine samples. NAATs have better sensitivity for STIs than traditional cultures, and urine samples have sensitivities at least as high as genital swabs.

Indications

1. Suspected STI in males and females
2. Screening for STI in pregnant females

Pearls, pitfalls and myths

1. NAATs essentially eliminate the need for urogenital swabbing when performed appropriately.
2. More rapid results than traditional culture.
3. Must be a *first-void* sample. The patient must be instructed NOT to clean the urethral meatus and to void directly into the sampling device.
4. The development of rapid first-void tests for chlamydia may allow definitive diagnosis within 1 hour with adequate sensitivity.

Genital swabs for STIs

Definition

During the evaluation of a patient for an STI, genital swabs may be tested for gonococcus, *C. trachomatis*, herpes simplex, fungi and protozoa.

Pearls, pitfalls and myths

1. *Gonorrhea and chlamydia*: Previously done by culture, these swabs are now typically DNA-amplification probes, offering much higher sensitivity and specificity. To perform adequately, the swab must be taken from the cervical canal, not the vaginal vault.
2. *Herpes simplex:* Viral culture of ulcerations suspected to be herpetic lesions can be performed. Samples should be obtained by unroofing a vesicle and collecting fluid onto the swab, followed by scraping the base of the ulcer to ensure tissue collection. Diagnosis is made by Tzanck smear.
3. *Fungal and protozoal infections*: During vaginal examination, samples of vaginal fluid can be obtained and placed on microscopy slides. One slide should have a drop of sterile saline placed, the other a drop of potassium hydroxide (KOH). Both slides are examined for the presence of white blood cells, epithelial cells, fungi (usually *Candida*), protozoans (*Trichomonas*), and bacteria. The KOH prep will lyse the epithelial cells, improving detection of fungi.

Syphilis

Definition

Syphilis is caused by *Treponema pallidum* and can be presumptively diagnosed in the ED by blood testing.

Pearls, pitfalls and myths

1. Testing for syphilis includes rapid plasma reagin (RPR) and venereal disease research laboratory (VDRL). These are non-treponemal antibody tests and are not specific to syphilis. Positive tests must be confirmed with a specific treponemal test, the fluorescent treponemal antibody absorption test (FTA-ABS). Treatment decisions can be made based on non-treponemal tests.
2. The FTA-ABS will be the first test to become positive, and it will remain positive for the entire life of the patient despite therapy; non-treponemal tests become positive later and may become negative after treatment.
3. All syphilis tests have their lowest sensitivities during primary and late-phase syphilis. During secondary syphilis, all tests approach 100% sensitivity.
4. Definitive diagnosis of a syphilis infection can be made by observing treponemal organisms under dark-field microscopy.

Inflammatory markers

Erythrocyte sedimentation rate

Definition

The erythrocyte sedimentation rate (ESR) is a nonspecific measure of systemic inflammation and the ability of RBCs to clump. RBC clumping increases with higher levels of fibrinogen.

Normal values

Male	0–15 mm/hr
Female	0–20 mm/hr

Abnormalities and causes

1. *Increased ESR:* Generally increased in any inflammatory, infectious, autoimmune, or malignant process. Striking elevations are seen in polymyalgia rheumatica and temporal arteritis.

Pearls, pitfalls and myths

1. The ESR in many conditions is too nonspecific to effectively rule in a particular disease process. It may be elevated in lymphoma and in conditions where cold agglutinins are present, such as mycoplasma or Epstein-Barr virus infections. It is often elevated during the second trimester of pregnancy.
2. A normal or low ESR is not sufficient to rule out inflammatory states (such as septic arthritis).
3. ESR increases with age; the formula (age + 10)/2 approximates the upper limit of normal.

C-reactive protein

Definition

C-reactive protein (CRP) is an acute-phase reactant produced by the liver in response to circulating levels of cytokines (such as interleukin-6). CRP may be elevated in a number of infectious or inflammatory states, and has been used as a marker of systemic inflammation.

Normal values

C-reactive protein	1–10 mg/mL

Abnormalities

1. Elevated in the presence of inflammation or bacterial infection; may also be slightly elevated during pregnancy and viral infection.
2. Higher numbers generally indicate greater systemic inflammation.

Indications

1. Evaluate a patient with suspected septic arthritis
2. Evaluate a patient with suspected appendicitis
3. As part of a cardiac risk profile

Pearls, pitfalls and myths

1. Like ESR and other inflammatory markers, CRP is a nonspecific test.
2. CRP has a more rapid rise and shorter half-life than ESR, giving it more utility in the evaluation of acute infections.
3. CRP levels do not have sufficient negative predictive value to rule out bacterial illness in the acute setting.
4. Data are conflicting regarding whether normal CRP levels can effectively rule out appendicitis.
5. Serial levels show promise to evaluate disease progression and prognosis.
6. Highly-sensitive CRP (hs-CRP) may identify cardiac disease risk and prognosis. However, it is unclear whether elevated levels are a cause or an effect of cardiac disease; thus, the role in the acute setting is limited.

Procalcitonin

Definition

Procalcitonin (PCT) is a molecule made by cells in response to microbial infection, as well as in the presence of interleukins and other cytokines. It is down-regulated in viral infection.

Normal Values

Procalcitonin	<0.25 mcg/L (ng/mL)

Indications

1. Determine appropriateness of antibiotic therapy in the setting of acute infection (often in the lower respiratory tract)
2. Evaluate disease severity and prognosis in septic patients

Pearls, pitfalls and myths

1. Not routinely used use in most EDs at this time.
2. Clinical severity is still a factor in the decision to treat with antibiotics.
3. Antibiotic therapy should be discouraged with PCT levels of <0.25 mcg/L, and encouraged with levels >0.25 mcg/L.
4. When PCT is used to guide therapy in lower respiratory tract infections, outcomes are similar, with decreased rates of antibiotic exposure and adverse events.
5. Commonly available assays lack sufficient sensitivity for widespread use.
6. Numerous variables affect prognosis in septic patients, so PCT cannot be used in exclusivity.

Toxicology screens

Toxicology screens may identify the presence or absence of illegal or abused substances, such as amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine, methadone, opiates, and phencyclidine. Toxicology screens vary from institution to institution.

Indications

1. General screen for the presence of illegal or abused substances from the urine. Indicated in patients who are symptomatic and the diagnosis is unclear or questionable.

Normal values

Time after ingestion when drugs of abuse test positive:

Amphetamines	2–3 days
Barbiturates	Varies; up to several weeks
Benzodiazepines	Varies depending on half-life; most up to 2–3 days
Cannabinoids	Up to 1 week in single use
Cocaine	8–12 hours after single use; 2–3 days in chronic users
Methadone	2–3 days
Opiates	1–2 days after single use; 2–4 weeks in chronic users
Phencyclidine	5–7 days after single use; 1–2 weeks in chronic users

Pearls, pitfalls and myths

1. A negative test does not necessarily exclude the presence of a drug. Many drugs are not detected by a toxicology screen, such as isoniazid, lithium, antihypertensives, anticoagulants, muscle relaxants, hallucinogens, newer antidepressants (selective serotonin reuptake inhibitors [SSRIs]), cardiac medications, mushrooms, plants, beta-blockers, insect repellants, cyanide, solvents, antibiotics, hypoglycemics, pesticides, phenol, household products, herbicides, ethylene glycol, nitrates, nitrites, thyroid hormones, and H2-antagonists.
2. Many false-positive drug tests occur in compounds having similar chemical structure.
3. It is more appropriate to understand toxidromes and narrow the scope of toxicologic screening by direct serum quantitative levels whenever possible.
4. Important specific serum drug levels can be ordered when clinically necessary, including acetaminophen, salicylic acid, ethanol, ethylene glycol, methanol, isopropyl alcohol, carbon monoxide, iron, tricyclic antidepressants (TCAs), organophosphates, lithium, digoxin, phenytoin, carbamazepine, and valproic acid.
5. A full serum toxicology screen may be useful in critically ill patients in whom an unsuspected toxin may dictate a change in management.

Additional specimen analysis

Blood cultures

Definition

Culturing blood for bacterial contamination is a common practice in many EDs. Typically, peripheral blood cultures are collected in both an aerobic and anaerobic culture medium. Blood cultures are often drawn when there is suspicion of systemic infection from cellulitis, pneumonia, pyelonephritis, meningitis, or lower urinary tract infections. In recent years, a desire to improve efficiency of antibiotic ordering and speed of drug delivery has increased the numbers of blood cultures drawn in patients with pneumonia; some feel this increase is inappropriate.

Indications

1. Evaluation of suspected bacteremia
2. Guide definitive antibiotic therapy in severe infection

Pearls, pitfalls and myths

1. Blood cultures have not been shown to significantly influence therapy in most infections.
2. There is a high rate of false-positive blood cultures, so results must be interpreted in light of the most common pathogens.
3. Blood cultures are no longer routinely recommended for well-appearing febrile children 3–36 months of age.

Viral swabs

Definition

Viral testing is available for several common viral agents, including respiratory syncytial virus (RSV), influenza A and B, and H1N1 influenza. Samples are typically collected by nasal or saline instillation. Culture, polymerase chain reaction (PCR), or immune assays are then performed, with high sensitivities and specificities for the presence of viral particles.

Indications

1. Suspected viral infection
2. Evaluation for a source of infection in a child or adult without an alternative clear source
3. Evaluation of patients presenting to the hospital for purposes of isolation or epidemiology

Pearls, pitfalls and myths

1. *RSV*: Testing for RSV does not typically change management. Clinical factors should be used to guide therapy as well as disposition. Some hospitals require RSV testing on admitted children to avoid infecting other children.
2. *Influenza*: Some studies suggest that rapid influenza testing is equivalent to clinical judgment, and should not be used routinely. However, other data indicate that clinical findings are not sufficiently predictive, and practitioners should use epidemiologic data or rapid viral testing. Confirmation of influenza infection may be useful in the elderly or very young.

3. *H1N1:* Influenza H1N1 is a novel influenza virus that became pandemic during the spring of 2009. Although H1N1 is a strain of influenza A, rapid antigen tests do not hold enough sensitivity to rule it out. PCR tests specific for H1N1 are available.

Throat swabs

Definition

Throat swabs are performed by gently stroking cotton-tipped applicators across the posterior oropharynx. Most often, the microorganism being isolated is group A beta-hemolytic streptococcus (GABHS). Throat swabs can be sent for rapid antigen testing, culture, or both.

Indications

1. Suspected pharyngeal infection by group A streptococcus

Pearls, pitfalls and myths

1. Rapid streptococcal testing confers speed at the expense of sensitivity. Reports place the sensitivity at approximately 80–85%.
2. Clinical criteria can be used to limit testing and treatment.
3. If rapid testing is positive, a confirmatory culture should be sent. This will also help in the rare case of treatment failure.

Sputum testing

Definition

Sputum samples can be sent from the ED and tested for multiple pathogens. Samples should be collected from bronchial aspirates when possible, as incorrect sampling can result in polymicrobial culture results from common oral and pharyngeal flora.

Indications

1. Microbial identification in pneumonia patients
2. Suspicion of tuberculosis (acid-fast bacilli [AFB])

Pearls, pitfalls and myths

1. Induced sputum samples often contain multiple bacteria that are commonly found in the respiratory tract.
2. Deep bronchial aspirates are possible in intubated patients, and may be more useful than induced samples.
3. Testing for tuberculosis requires the use of a specific collection device for AFB and multiple negative results, so is not recommended in the ED.

Wound cultures

Definition

Fluid taken from abscesses and infected wounds can be sent for aerobic and anaerobic cultures.

Pearls, pitfalls and myths

1. Culturing wounds or abscess fluid does not typically help guide therapy, and should not be routinely performed.
2. With the rise of methicillin-resistant *Staphlococcus aureus* (MRSA), abscesses are being cultured more frequently. If MRSA is suspected, knowledge of your local resistance patterns may be more useful than a wound culture.
3. Cultures may be more useful to evaluate treatment failures.

Stool examinations

Definition

Stool specimens can be obtained to evaluate for infectious processes. Tests include Gram stain, culture, Wright stain for fecal leukocytes, and inspection for ova and parasites.

Indications

1. Evaluate for infections and treatable causes of diarrhea

Pearls, pitfalls and myths

1. The majority of infectious diarrhea is treated supportively, so stool culture may not change management.
2. Clinical factors may be more useful in the evaluation of diarrhea, such as the presence or absence of fever, bloody stools, recent travel, and ingestion of potentially contaminated food or water.
3. Stool culture is indicated in prolonged diarrheal illness, as well as in cases of suspected *Clostridium difficile* colitis.
4. Fecal leukocytes are present with large bowel inflammation, and have been used to screen for infectious diarrhea. However, this test does not have sufficient negative predictive value to rule out infectious (bacterial) disease.

Cerebrospinal fluid

Definition

Cerebrospinal fluid (CSF) bathes the central nervous system, providing nutrients, protection, and disposal of waste. CSF can be obtained from lumbar puncture (LP), from ventriculo-peritoneal (VP) shunts, or during surgical procedures.

Indications

1. Investigation for CSF infections (meningitis, encephalitis)
2. Suspected subarachnoid hemorrhage
3. Evaluation for inflammatory disorders, such as multiple sclerosis (MS) and Guillain-Barré syndrome (GBS)

Normal values

CSF component	Adults	Neonates
Color	Clear	Clear
Red blood cells	0 cells/μL	0 cells/μL
White blood cells	0–5 cells/μL	0–30 cell/μL
Glucose	50–80 mg/dL	approaches plasma glucose
Protein	15–45 mg/dL	<150 mg/dL

Abnormalities

1. *Color*: Pink CSF implies a traumatic tap or blood in the subarachnoid space. Xanthochromia indicates the presence of RBC pigments in the centrifuged sample and is highly predictive of subarachnoid hemorrhage (SAH). Cloudy CSF is concerning for infection.
2. *RBCs*: The presence of any RBCs is abnormal. Smaller amounts are likely due to trauma during the LP, whereas larger numbers (in the thousands) are likely pathologic and may indicate subarachnoid hemorrhage. Clearing of the RBCs in successive tubes suggests traumatic LP. RBCs may suggest herpes infection in the appropriate clinical setting.
3. *WBCs*: Elevated WBCs suggest infection. The WBC differential can be helpful in distinguishing between bacterial (high polymorphonuclear leukocytes [PMNs]) and viral (high lymphocytes) causes of infection. In a traumatic tap, approximately 1 WBC is seen for every 700 RBCs.
4. *Glucose*: Decreased in bacterial infection as well as hypoglycemia; in adults, CSF glucose should be approximately 50–70% of plasma.
5. *Protein*: Elevated in viral infections or inflammatory states, such as MS and GBS.

Pearls, pitfalls and myths

1. Visual xanthochromia is not as useful as spectrophotometric determination. The sample should be protected from light and run immediately (not batched).
2. Historically, a WBC:RBC ratio of 1:700 has been used to estimate the number of WBCs in the setting of a traumatic tap.
3. Elevated cell counts will increase the protein concentration in the CSF (8 mg/dL for every 10,000 RBCs).
4. Additional specific tests are needed to evaluate for MS, GBS, and other diseases.

Synovial fluid

Definitions

Synovial fluid can be obtained from arthrocentesis of the affected joint. Common joints aspirated are the knee, hip, wrist, elbow and ankle.

Indications

1. Evaluation of suspected septic arthritis
2. Diagnosis of inflammatory arthritis (i.e., gout, pseudogout)

Normal values

Clarity	Clear
Color	Clear to pale yellow
WBCs	0–150 cells/mL
PMNs	<25%
RBCs	0 cells/mL
Glucose	0–10 mg/dL
Crystals	None
Gram stain/culture	No organisms/negative

Abnormalities

1. *Clarity*: Normal joint fluid is transparent. Cloudy synovial fluid may prevent newsprint from being easily read through the fluid, and usually results from increased WBCs or protein.
2. *Color*: Normal joint fluid is colorless to light yellow. Red or bloody synovial fluid is often from traumatic hemarthrosis.
3. *WBCs*: Increased WBCs are seen in inflammatory and infectious states.
4. *PMNs*: The presence of PMNs is highly suggestive of infection, as opposed to the absence of PMNs, which indicates an inflammatory, likely non-infectious state.
5. *RBCs*: Elevated RBCs can be seen from traumatic arthrocentesis or hemarthrosis. RBCs can also be elevated in infectious states.
6. *Glucose*: Decreased in infection; normal to slightly decreased with inflammation.
7. *Crystals*: Can be seen in gout or pseudogout. Crystals in gout are negatively birefringent, and are commonly monosodium urate monohydrate (urate). Crystals in pseudogout are positively birefringent, and are generally calcium pyrophosphate dihydrate. Other crystals identified less commonly include apatite and other basic calcium phosphates (apatite gout), calcium oxalate (oxalate gout), and lipids (lipid gout).
8. *Gram stain/culture*: Organisms on Gram stain or culture are generally pathologic, although poor sterile technique can result in contaminants.

Gonococcal arthritis may result in aspirates lacking characteristic Gram-negative intracellular organisms, and culture (using pre-warmed chocolate agar to increase yield) is positive only 50% of the time.

Pearls, pitfalls and myths

1. An infected joint will have synovial fluid to be aspirated; therefore, a properly performed "dry tap" suggests no infection.
2. WBC, CRP and ESR cannot be used to rule out septic arthritis. Only joint fluid aspirate has high enough sensitivity.
3. Septic arthritis will typically have a WBC count >50,000. If the WBCs are significantly <50,000, it is unlikely to be an infectious process.
4. Inflammatory arthritides (gout, pseudogout, rheumatoid, lupus) may have total WBC counts >50,000. In such cases, the PMN percentage differentiates septic arthritis from non-infectious causes (Table 32.5).

Hormones

Cortisol

Definition

Cortisol is released by the adrenal cortex after stimulation by adrenocorticotropic hormone (ACTH) in response to stress or hypoglycemia. Approximately 10% is free (90% is protein-bound); it is thought that this 10% is active. Cortisol levels exhibit a diurnal variation, with the highest levels in the morning. Cortisol production can be induced by the administration of cosyntropin (a commercially available ACTH analogue) in what is referred to as a *Cortrosyn (Cosyntropin) stim test*.

Indications

1. Evaluate for adrenal insufficiency in the setting of persistent hypotension from sepsis, as well undifferentiated hypotension

Normal values

8 AM cortisol	5–10 mcg/dL
4 PM cortisol	3–12 mcg/dL
Cosyntropin stim test	18–20 mcg/dL

Abnormalities

1. Low serum cortisol at any point suggests adrenal insufficiency.
2. A random cortisol of >25 mcg/dL at times of metabolic stress makes adrenal insufficiency unlikely.
3. Lack of response to cosyntropin may indicate inability of the adrenals to respond to stress and relative adrenal insufficiency (in the absence of absolute insufficiency).

Pearls, pitfalls and myths

1. The cosyntropin stim test is performed by administering 250 mcg cosyntropin and checking cortisol levels at 30 and 60 minutes post-administration.
2. Previous administration of glucocorticoids (such as prednisone or methylprednisolone) may interfere with the stim test. If steroids are to be given, dexamethasone is a better choice as it does not interfere with the test.
3. More useful than a *stim test* is a random cortisol; if <25 mcg/dL in a critically ill patient, relative adrenal insufficiency is likely present.

Thyroid studies

Definition

Thyroid hormone is produced by the thyroid gland in response to thyroid-stimulating hormone (TSH). T3 and T4 are the metabolically active forms of the hormone, and are mostly bound to plasma proteins.

Indications

1. TSH, free T3 and free T4 can be measured. TSH, FT3 and FT4 measurements help diagnose hypo- and hyperthyroid states when clinically suspected.

Normal values

TSH	0.4–4.5 mIU/L
Free T3 (FT3)	0.5 ng/dL
Free T4 (FT4)	2 ng/dL

Abnormalities

1. Low TSH levels with low T3 and T4 levels indicate secondary hypothyroidism, whereas low TSH levels with high T3 and T4 levels indicate primary hyperthyroidism.
2. High TSH levels with low T3 and T4 levels indicate primary hypothyroidism (lack of hormone synthesis), whereas low TSH levels with low T3 and T4 levels are seen in pituitary disorders (secondary hypothyroidism).

Pearls, pitfalls and myths

1. Circulating hormone levels can be affected by medications that alter protein binding.
2. Levels must always be interpreted together to diagnose the correct disorder.
3. Hypo- and hyperthyroidism are part of the differential diagnosis for numerous disease processes, so consideration of these diagnoses is important. Common presentations include

depression, psychoses, dysrhythmias and weight changes.

4. Some facilities cannot get same-day or stat levels, but ordering the test may be useful to consulting or primary care providers.

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~StormRG~